A 15-year-old boy develops severe cardiomyopathy following myocarditis from Coxsackie virus and is placed on the cardiac transplant list. Two weeks following his cardiac transplantation from a matched donor, he develops dyspnea on exertion. Extensive evaluation is undertaken including cardiac catheterization and endomyocardial biopsy to assess for acute allograft rejection. Which of the following histologic findings is most consistent with this diagnosis?

- A. Concentric coronary artery intimal thickening
- B. Dense interstitial lymphocytic infiltrate
- C. Patchy necrosis with granulation tissue
- D. Perivascular infiltrate with abundant eosinophils
- E. Scant inflammatory cells and interstitial fibrosis

Submit

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A 15-year-old boy develops severe cardiomyopathy following myocarditis from Coxsackie virus and is placed on the cardiac transplant list. Two weeks following his cardiac transplantation from a matched donor, he develops dyspnea on exertion. Extensive evaluation is undertaken including cardiac catheterization and endomyocardial biopsy to assess for acute allograft rejection. Which of the following histologic findings is most consistent with this diagnosis?

- ✗ A. Concentric coronary artery intimal thickening (4%)
- ✓ B. Dense interstitial lymphocytic infiltrate (76%)
- C. Patchy necrosis with granulation tissue (7%)
- D. Perivascular infiltrate with abundant eosinophils (8%)
- E. Scant inflammatory cells and interstitial fibrosis (3%)

Incorrect
Correct answer
B

76%
Answered correctly

05 mins, 44 secs
Time Spent

2023
Version

Explanation

Transplant rejection reactions			
Type of rejection	**Onset time**	**Etiology**	**Morphology**
Hyperacute	Minutes to hours	• Preformed recipient antibodies against graft antigens	• Gross mottling & cyanosis • Arterial fibrinoid necrosis & capillary thrombotic occlusion
		• Exposure to donor antigens induces	• **Cellular:** lymphocytic interstitial infiltrate & endotheliitis

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Explanation

Transplant rejection reactions			
Type of rejection	**Onset time**	**Etiology**	**Morphology**
Hyperacute	Minutes to hours	• Preformed recipient antibodies against graft antigens	• Gross mottling & cyanosis • Arterial fibrinoid necrosis & capillary thrombotic occlusion
Acute	Usually <6 months	• Exposure to donor antigens induces activation of naive immune cells • Predominantly **cell-mediated**	• **Cellular:** lymphocytic interstitial infiltrate & endotheliitis • Humoral: C4d deposition, neutrophilic infiltrate, necrotizing vasculitis
Chronic	Months to years	• Chronic low-grade immune response refractory to immunosuppression • Mixed cell-mediated and humoral	• Vascular wall thickening & luminal narrowing • Interstitial fibrosis & parenchymal atrophy

Acute cardiac transplant rejection typically occurs **within 6 months** of transplantation and can be diagnosed via endomyocardial biopsy. Acute rejection can be **cell-mediated** (ie, acute cellular rejection [ACR]) or, less commonly, antibody-mediated (ie, acute antibody-mediated rejection).

In ACR, host **T-lymphocytes** are sensitized against foreign human leukocyte antigens (HLA) in the cardiac allograft, which leads to inflammation and injury to the transplanted organ. Microscopic features of ACR include an **interstitial lymphocytic infiltrate** (primarily T-lymphocytes) and **damaged myocytes**.

Symptoms consistent with progressive rejection include those found in systolic dysfunction (eg, dyspnea on exertion). However, cardiac transplant recipients undergo close surveillance with regular endomyocardial biopsies,

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Acute cardiac transplant rejection typically occurs **within 6 months** of transplantation and can be diagnosed via endomyocardial biopsy. Acute rejection can be **cell-mediated** (ie, acute cellular rejection [ACR]) or, less commonly, antibody-mediated (ie, acute antibody-mediated rejection).

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Symptoms consistent with progressive rejection include those found in systolic dysfunction (eg, dyspnea on exertion). However, cardiac transplant recipients undergo close surveillance with regular endomyocardial biopsies, and acute rejection is often diagnosed before patients become symptomatic.

(Choice A) Concentric intimal thickening of the coronary arteries can be seen in cardiac allograft vasculopathy, an important cause of ischemic damage and long-term graft dysfunction. The pathogenesis is complex and includes the release of cytokines that cause smooth muscle cell proliferation in the vessel wall.

(Choice C) Patchy necrosis with granulation tissue is indicative of ischemic damage to the donor heart. Ischemia in a donor heart can occur in a variety of situations including during attempted resuscitation of the donor patient, during transport of the organ from the donor to the recipient, and acutely during initial perfusion of the organ after transplantation.

(Choice D) Perivascular infiltrate with abundant eosinophils is a histopathologic feature of hypersensitivity myocarditis. Hypersensitivity myocarditis often represents an immunologic response to a newly initiated drug.

(Choice E) Interstitial fibrosis with scant inflammation can be seen in chronic rejection of solid organ transplants. Chronic rejection is mediated by host T-lymphocytes as well as antibodies and classically occurs months to years following transplantation.

Educational objective:
Acute cardiac transplant rejection usually occurs weeks to months following transplantation and is primarily a cell-mediated process. In acute cellular rejection, endomyocardial biopsy shows an interstitial lymphocytic infiltrate with myocyte damage.

Immunology Cardiovascular System Heart transplantation

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Acute cardiac transplant rejection typically occurs **within 6 months** of transplantation and can be diagnosed via endomyocardial biopsy. Acute rejection can be **cell-mediated** (ie, acute cellular rejection [ACR]) or less comm

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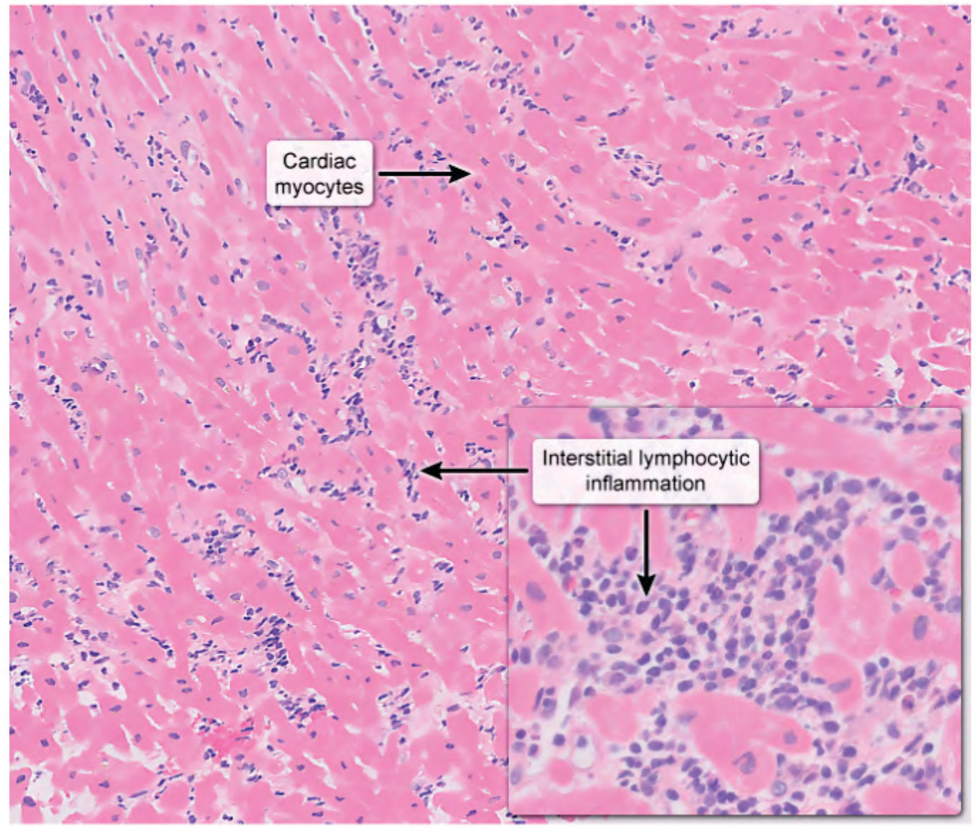
Cardiac acute cellular rejection



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Immunology | Cardiovascular System | Heart transplantation

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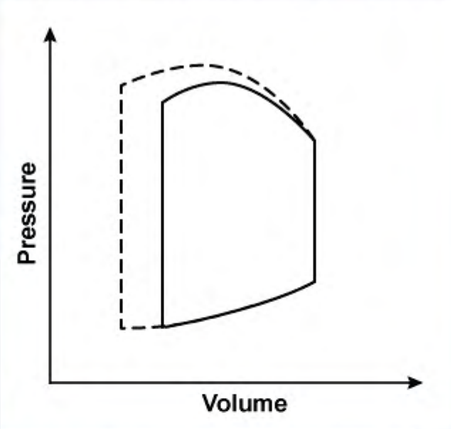
A 54-year-old man is admitted to the hospital due to cough, shortness of breath, and chest discomfort. He has a known history of coronary artery disease and underwent coronary artery bypass graft surgery 3 years ago. As a result of drug therapy, the left ventricular pressure-volume loop changes from a solid to a dashed line, as shown in the graph below (the loop is unchanged in areas where no dashed line is seen):



Which of the following parameters is most likely increased as a result of this change?

- A. End-diastolic pressure
- B. End-systolic volume
- C. Stroke volume
- D. Ventricular compliance
- E. Ventricular preload

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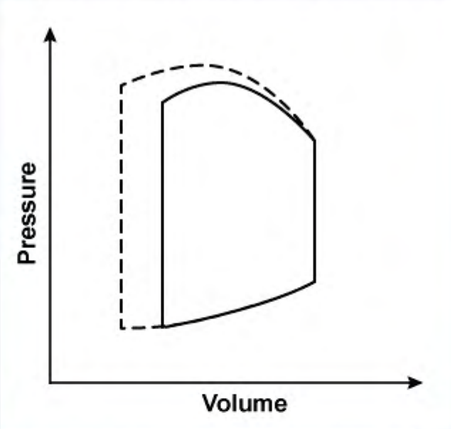

Which of the following parameters is most likely increased as a result of this change?

- ✗ A. End-diastolic pressure (4%)
- B. End-systolic volume (5%)
- ✓ C. Stroke volume (81%)
- D. Ventricular compliance (3%)
- E. Ventricular preload (4%)

Incorrect
Correct answer
C

81%
Answered correctly

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Explanation

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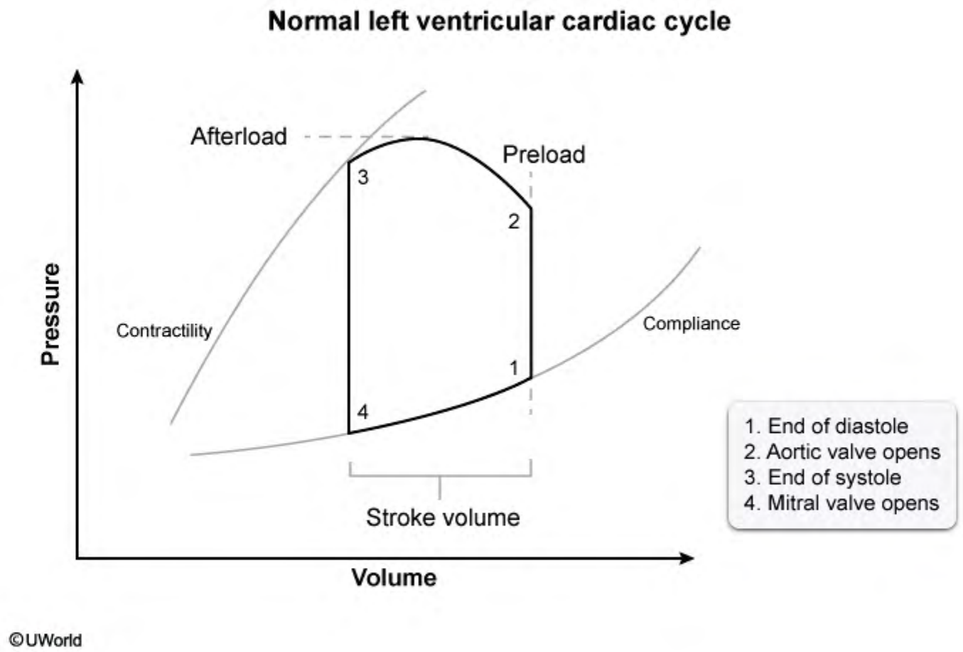

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(Choices A and D) LV end-diastolic pressure (indicated at point 1) and compliance are unchanged in this patient.

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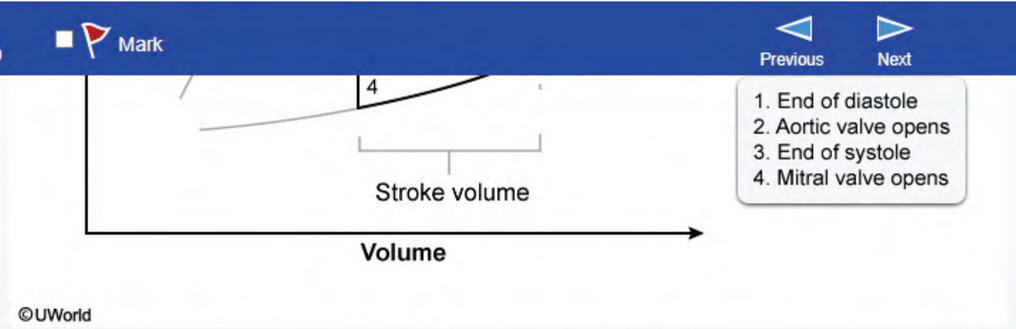

As a result of drug therapy, point 3 on this patient's left ventricular (LV) pressure-volume loop shifts up and to the left; this represents an **increase in cardiac contractility** that would occur with a positive inotropic agent (eg, digoxin, dobutamine). Although LV preload, represented by the volume at point 1, is unchanged **(Choice E)**, the increase in contractility causes an increase in the fraction of LV volume ejected (ie, increased ejection fraction) and **increases stroke volume** (the horizontal distance between points 1 and 3). The volume remaining in the left ventricle at the end of systole (end-systolic volume represented by the volume at point 3) is consequently decreased **(Choice B)**. LV afterload (the maximum pressure reached during blood ejection) is slightly increased due to the increase in contractility.

(Choices A and D) LV end-diastolic pressure (indicated at point 1) and compliance are unchanged in this patient. Compliance is represented by the curve at the bottom of the pressure-volume loop (between point 4 and point 1) and determines the rate of LV pressure increase as LV volume increases during diastolic filling. Compliance is typically unchanged in the short term, but it can change over the long term. Decreased LV compliance (eg, due to concentric LV hypertrophy from prolonged systemic hypertension) would increase the slope of the compliance curve and increase end-diastolic pressure for a given LV end-diastolic volume.

Educational objective:
An increase in cardiac contractility shifts the top left corner (point 3) of the left ventricular pressure-volume loop up and to the left and increases stroke volume due to an increase in the fraction of LV preload that is ejected during systole (ie, increased ejection fraction).

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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https://t.me/USMLEWorldStep1

1. End of diastole
2. Aortic valve opens

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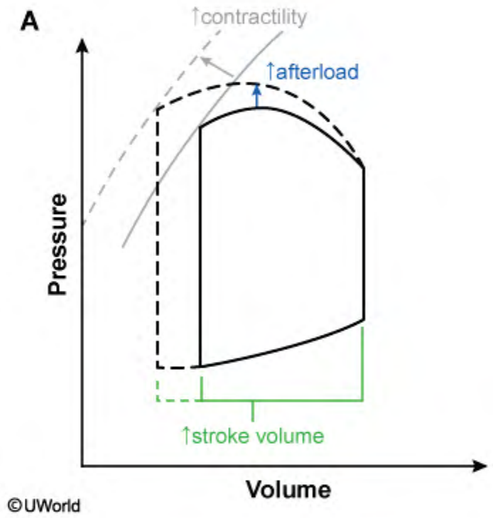

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1. End of diastole
2. Aortic valve opens

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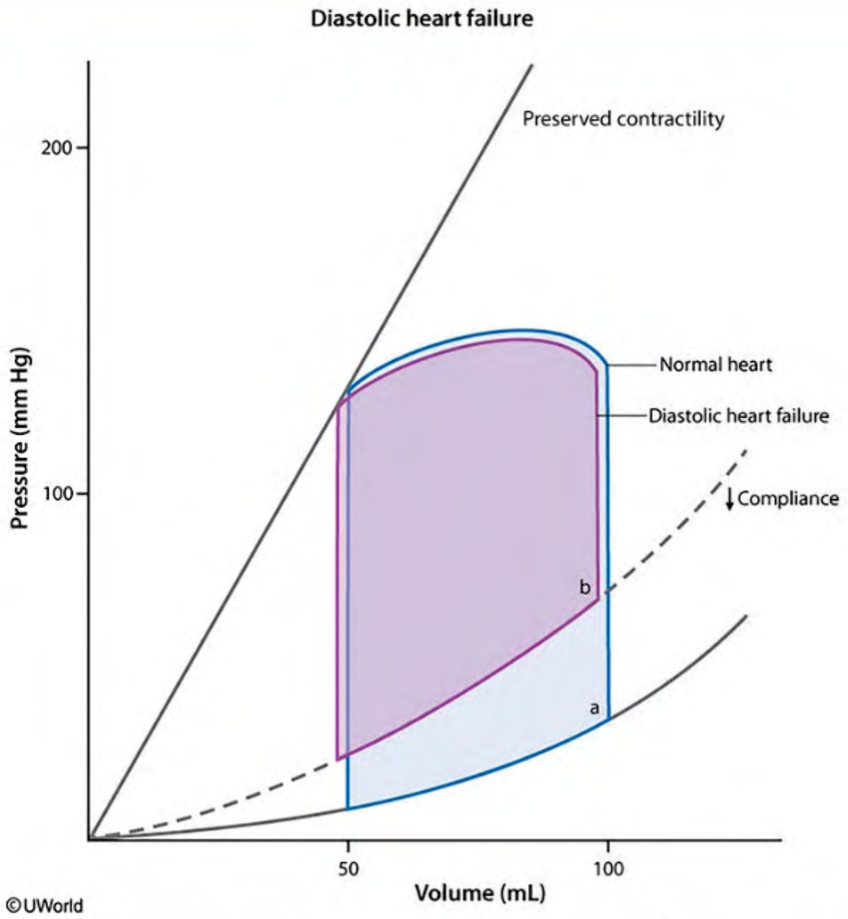

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https://t.me/USMLEWorldStep1

A 64-year-old man presents to the office with chest discomfort and mild shortness of breath that started one hour ago after an argument with his wife. He has a long history of hypertension and diabetes mellitus. His blood pressure is 180/100 mmHg and his heart rate is 100/min. An EKG taken in the emergency department shows no signs of acute ischemia. Cardiac auscultation in the left decubitus position on full expiration reveals a presystolic sound. Which of the following best explains this physical examination finding?

- A. Papillary muscle dysfunction
- B. Left ventricular hypertrophy
- C. Increased left ventricular preload
- D. Aortic regurgitation
- E. Mitral leaflet fusion and fibrosis

Submit

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- A. Papillary muscle dysfunction (7%)
- ✓ B. Left ventricular hypertrophy (60%)
- C. Increased left ventricular preload (14%)
- D. Aortic regurgitation (5%)
- E. Mitral leaflet fusion and fibrosis (10%)

Correct | 60% Answered correctly | 04 secs Time Spent | 2023 Version

Explanation

An **S4** is an isolated presystolic (late diastolic) sound heard just prior to S1. An S4 is caused by **left atrial contraction** that forces atrial blood into a poorly compliant left ventricle (LV) that is nearly filled to capacity. When blood is forced into the ventricle by the atrium under these conditions it generates a large, rapid increase in LV pressure thereby producing a sound possibly resulting from rapid deceleration of the forced blood or from impact of the filling ventricle with the chest wall. LV hypertrophy and other causes of diastolic heart failure therefore favor generation of an S4 sound. This patient's LV hypertrophy is likely secondary to **chronic hypertension**.

(Choice A) Papillary muscle dysfunction may cause incomplete closure of the atrioventricular valves resulting in a murmur audible during systole.

(Choice C) An increase in LV preload is typically accommodated by stretching in a normally compliant ventricle

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Correct

60%
Answered correctly

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Time Spent

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Version

Explanation

An **S4** is an isolated presystolic (late diastolic) sound heard just prior to S1. An S4 is caused by **left atrial contraction** that forces atrial blood into a poorly compliant left ventricle (LV) that is nearly filled to capacity. When blood is forced into the ventricle by the atrium under these conditions it generates a large, rapid increase in LV pressure thereby producing a sound possibly resulting from rapid deceleration of the forced blood or from impact of the filling ventricle with the chest wall. LV hypertrophy and other causes of diastolic heart failure therefore favor generation of an S4 sound. This patient's LV hypertrophy is likely secondary to **chronic hypertension**.

(Choice A) Papillary muscle dysfunction may cause incomplete closure of the atrioventricular valves resulting in a murmur audible during systole.

(Choice C) An increase in LV preload is typically accommodated by stretching in a normally compliant ventricle and does not result in characteristic heart sounds.

(Choice D) Aortic regurgitation causes a decrescendo early and mid-diastolic murmur.

(Choice E) Mitral leaflet fusion and fibrosis may cause mitral stenosis resulting in a mid-diastolic murmur with possible late diastolic (pre-systolic) accentuation due to atrial contraction.

Educational objective:
An S4 sound is best heard near the cardiac apex at the point of maximal impulse with the patient in the left lateral decubitus position. It results from the atrial contraction, which forces blood into an LV that has reached its limit of compliance. Chronic hypertension causes left ventricular hypertrophy, which itself may promote the generation of an S4 sound.

Physiology	Cardiovascular System	Heart sounds
Subject	System	Topic

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A 33-year-old man comes to the emergency department due to fever, chills, cough, and shortness of breath over the last week. He smokes a pack of cigarettes a day, consumes 2 or 3 alcoholic beverages daily, and uses intravenous heroin 2 or 3 times per week. He is febrile and tachycardic. Blood cultures grow *Staphylococcus aureus*. Chest x-ray shows bilateral nodular lesions with areas of cavitation. The valve most likely affected in this patient can be best evaluated by auscultation at which of the following sites?

- A. A
- B. B

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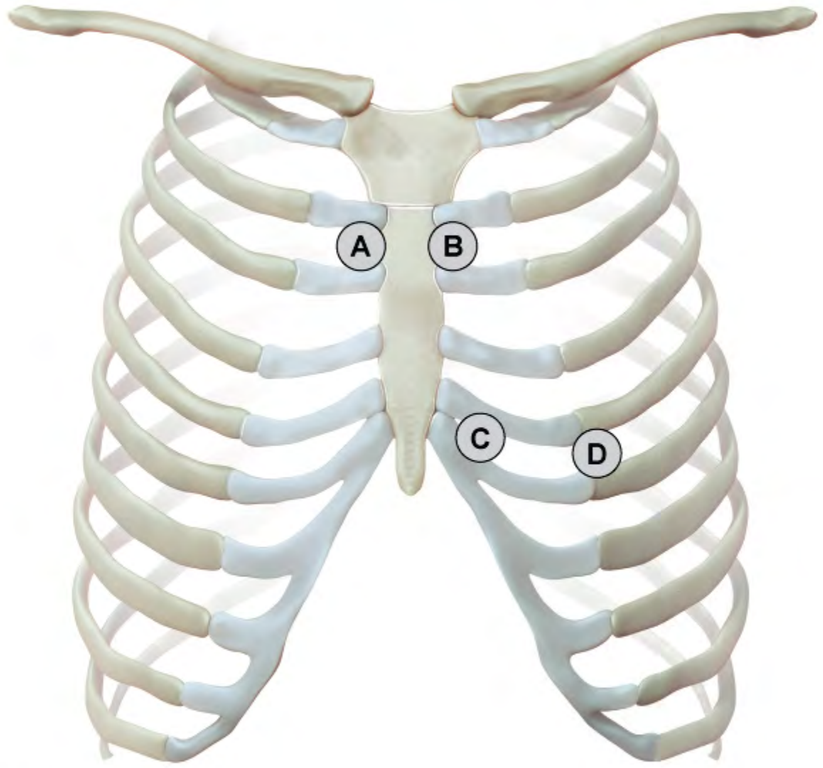

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- A. A
- B. B
- C. C
- D. D

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- A. A (2%)
- B. B (1%)
- ✓ C. C (88%)
- D. D (7%)

Correct

88%
Answered correctly

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Explanation

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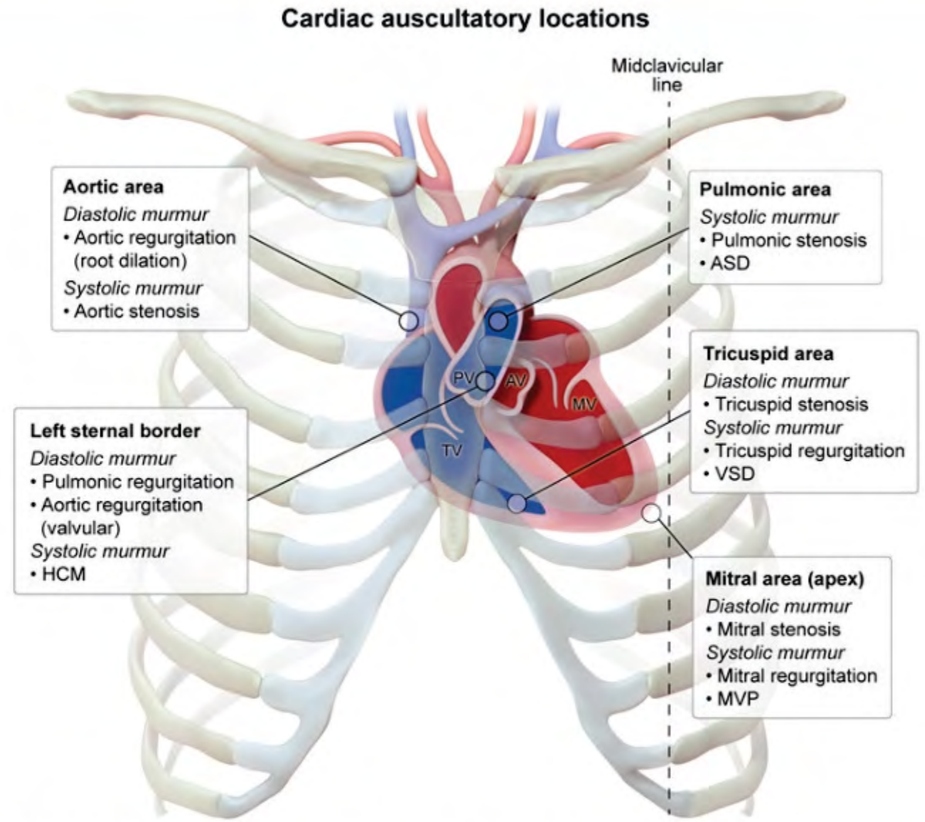

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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intravenous drug users; bacterial adherence may be encouraged by the inadvertent injection of particulate matter

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The cavitary pulmonary nodules on this patient's chest x-ray are consistent with **septic emboli**, which are a common complication of **right-sided infective endocarditis** (IE). The **tricuspid valve** is frequently affected in intravenous drug users; bacterial adherence may be encouraged by the inadvertent injection of particulate matter that damages the tricuspid valve endothelium. *Staphylococcus aureus* is the most common organism responsible for IE in intravenous drug users.

A murmur of IE typically results from regurgitation of blood due to impaired closure of the affected valve. Although tricuspid valve endocarditis does not always cause a murmur, an early- or holo-systolic murmur of **tricuspid regurgitation** is sometimes appreciated. This murmur is best auscultated in the 4th or 5th intercostal space at the **left lower sternal border** (the tricuspid auscultation area), and it increases in intensity with maneuvers that increase right ventricular blood volume (eg, inspiration, supine leg raise).

(Choices A and D) Most aortic valve murmurs are best auscultated in the 2nd intercostal space at the right upper sternal border. Mitral valve murmurs are best auscultated at the cardiac apex, which is usually located near the midclavicular line in the left 5th intercostal space. IE affecting these left-sided heart valves may cause septic embolization to the kidneys, brain, or other systemic organs but not to the pulmonary circulation.

(Choice B) Pulmonic valve murmurs are best auscultated in the 2nd intercostal space at the left upper sternal border. IE affecting the pulmonic valve can occur in intravenous drug users and lead to septic pulmonary emboli, but it is far less common than tricuspid valve endocarditis.

Educational objective:
Infective endocarditis in intravenous drug users commonly affects the tricuspid valve, often leading to septic pulmonary emboli. Patients can have an early- or holo-systolic murmur of tricuspid regurgitation, which is best auscultated in the 4th or 5th intercostal space at the left lower sternal border.

Anatomy	Cardiovascular System	Tricuspid regurgitation
Subject	System	Topic

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A 32-year-old woman comes to the office for preconception medical evaluation. She feels well, reports no health issues, and has had regular menstruations. The patient has been an elite long-distance swimmer since high school and recently retired from professional competition. She still continues to practice swimming regularly. The patient does not use tobacco, alcohol, or illicit drugs. Blood pressure is 114/66 mm Hg and pulse is 60/min. Physical examination shows no abnormalities. Compared to a person of similar age and sex with sedentary lifestyle, which of the following cardiac changes are most likely present in this patient?

	Left ventricular ejection fraction	Left ventricle cavity size	Maximum cardiac output
○ A.	Decreased	Increased	Increased
○ B.	Increased	Decreased	Increased
○ C.	Increased	Decreased	Unchanged
○ D.	Unchanged	Increased	Increased
○ E.	Decreased	Unchanged	Unchanged

Submit

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		Left ventricular ejection fraction	Left ventricle cavity size	Maximum cardiac output	
✗	A.	Decreased	Increased	Increased	(5%)
	B.	Increased	Decreased	Increased	(38%)
	C.	Increased	Decreased	Unchanged	(6%)
✓	D.	Unchanged	Increased	Increased	(47%)
	E.	Decreased	Unchanged	Unchanged	(2%)

Incorrect
Correct answer
D

47%
Answered correctly

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Explanation

Athlete's heart adaptations

Normal

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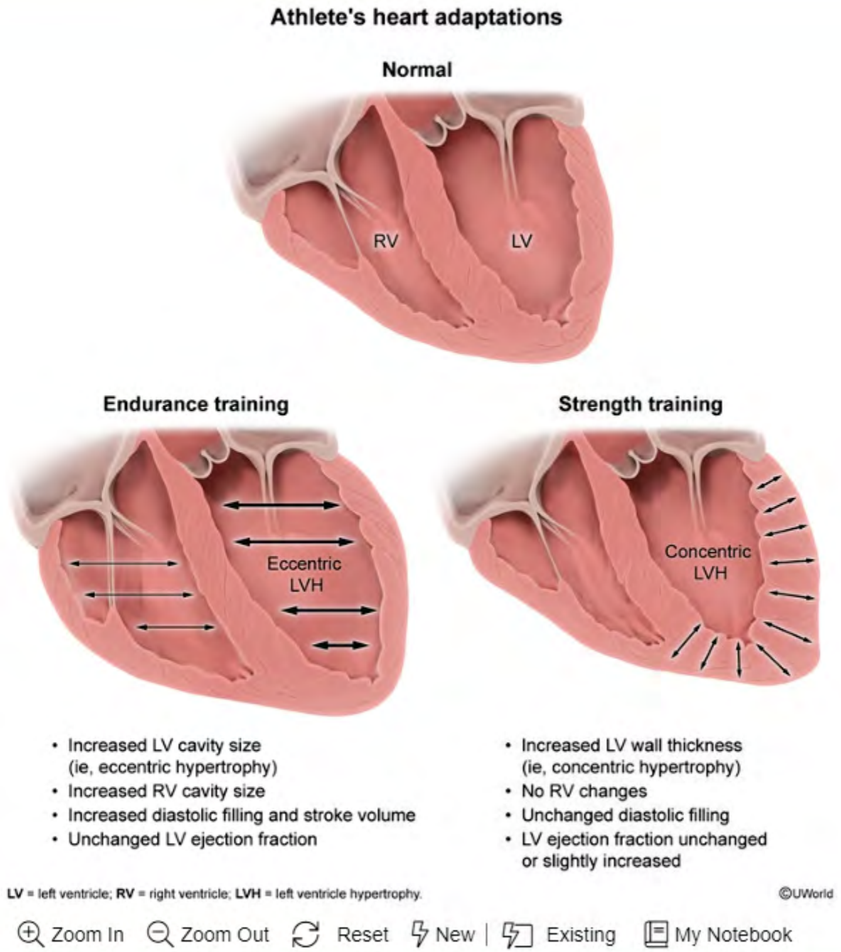

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Normal

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LV = left ventricle; **RV** = right ventricle; **LVH** = left ventricle hypertrophy.

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Resting cardiac output is approximately 5 L/min and commonly increases to 15 or 20 L/min with exercise. Highly trained endurance athletes with the characteristic cardiovascular adaptations of **athlete's heart** can often increase their cardiac output up to 35 or 40 L/min. Maximum heart rate does not significantly increase in highly trained athletes; therefore, the **increased maximum cardiac output** is primarily due to **increased stroke volume** (cardiac output = stroke volume x heart rate).

To meet the metabolic demands of frequent and intense **endurance training** (eg, long-distance swimming) the body gradually increases both red blood cell mass and plasma volume to increase oxygen carrying capacity. In addition, skeletal muscles develop increased arteriolar and capillary density, which improves oxygen uptake and also causes overall **reduced systemic vascular resistance** (SVR).

Both the increased blood volume and reduced SVR contribute to increased cardiac venous return (ie, preload), which places increased volume load on the left ventricle. In response, the left ventricle undergoes eccentric hypertrophy to **increase left ventricular cavity size** and improve diastolic filling capacity. The resulting increase in end-diastolic volume capacity is the primary driver of the marked increase in stroke volume that occurs with endurance training (a training-induced increase in myocardial contractility also somewhat contributes). Because end-diastolic volume and stroke volume are both proportionally increased, **left ventricular ejection fraction** is mostly **unchanged**.

The right ventricle also undergoes similar cavity enlargement to support the increase in maximum cardiac output.

Educational objective:
With endurance training, the physiologic changes of athlete's heart allow for increased maximum cardiac output via an increase in stroke volume. The left ventricle undergoes eccentric hypertrophy to increase left ventricular cavity size and improve diastolic filling capacity. The resulting increase in end-diastolic volume capacity allows for increased stroke volume and cardiac output. Because end-diastolic volume and stroke volume are both proportionally increased, left ventricular ejection fraction is mostly unchanged.

References

- Cardiovascular adaptations to exercise training.

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LV = left ventricle; RV = right ventricle; LVH = left ventricle hypertrophy.

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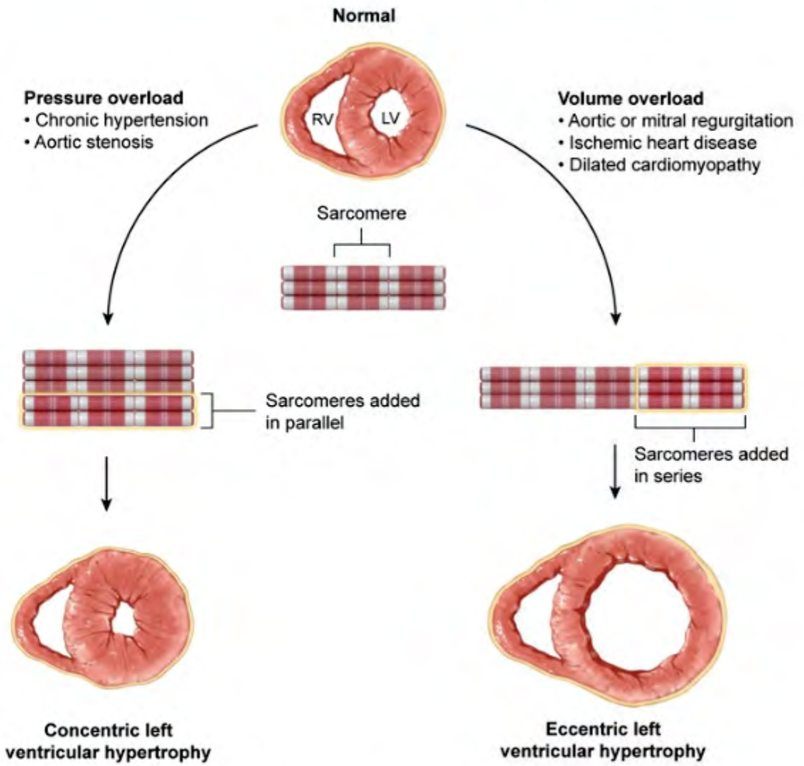
Concentric & eccentric left ventricular hypertrophy*



*The right ventricle undergoes similar adaptation when exposed to chronic pressure or volume overload.
LV = left ventricle; RV = right ventricle.

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LV = left ventricle; RV = right ventricle; LVH = left ventricle hypertrophy.

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Stroke volume & ejection fraction

Stroke volume (SV) = End-diastolic volume (EDV) − End-systolic volume

$$\text{Ejection fraction} = \frac{SV}{EDV}$$

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Stroke volume & ejection fraction

Stroke volume (SV) = End-diastolic volume (EDV) − End-systolic volume

$$\text{Ejection fraction} = \frac{\text{SV}}{\text{EDV}}$$

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A 48-year-old man with diet-controlled type 2 diabetes mellitus is being evaluated for occasional retrosternal chest pain. He has no history of hypertension, and his blood pressure measurements during office visits were always within normal limits. There is no family history of cardiovascular disease. The patient undergoes exercise treadmill stress testing. He walks for 7 minutes on the treadmill and stops due to fatigue but does not experience chest pain. His ECG does not show any abnormal changes. Heart rate ranges from 70/min at rest to 132/min at peak and mean blood pressure ranges from 95 mm Hg at rest to 112 mm Hg at peak. Which of the following parameters was most likely decreased during peak stress compared to the resting state in this patient?

- A. Cardiac output
- B. Left ventricular end-diastolic pressure
- C. Pulmonary artery systolic pressure
- D. Systemic systolic blood pressure
- E. Total systemic vascular resistance

Submit

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- A. Cardiac output (8%)
- ✗ B. Left ventricular end-diastolic pressure (28%)
- C. Pulmonary artery systolic pressure (8%)
- D. Systemic systolic blood pressure (1%)
- ✓ E. Total systemic vascular resistance (51%)

Incorrect
Correct answer
E

51%
Answered correctly

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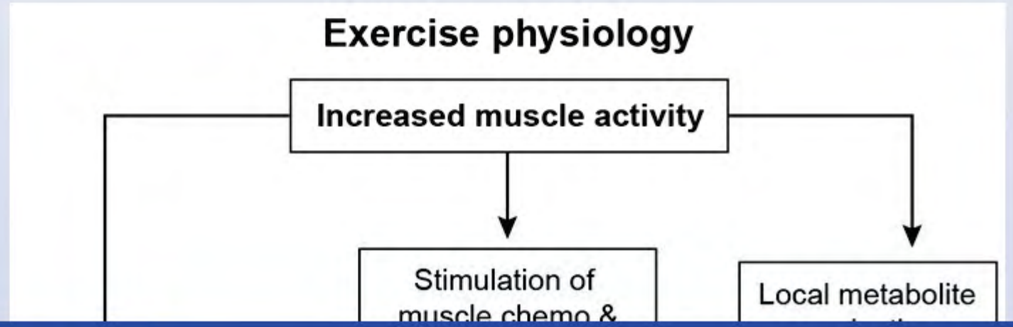
Explanation



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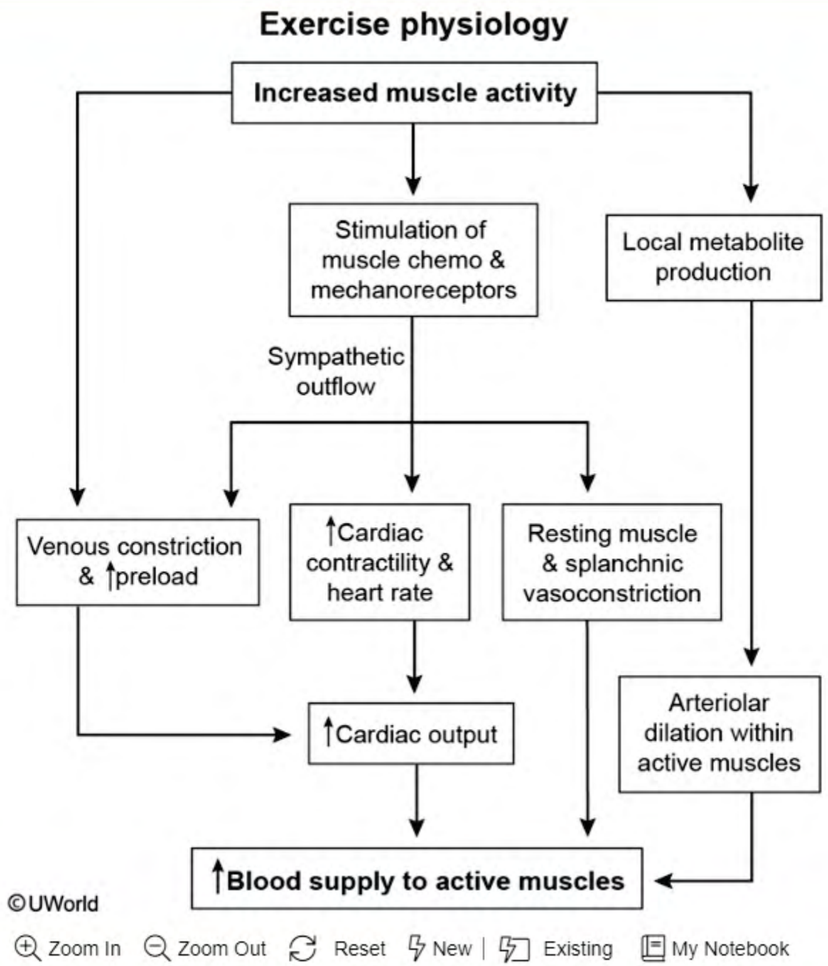

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Stimulation of muscle chemo &

Local metabolite

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Exercise is associated with several hemodynamic and vasoregulatory adjustments to provide maximal blood flow to the active muscles. Stimulation of mechano- and chemoreceptors in the exercising muscle causes sympathetic activation and withdrawal of vagal tone, resulting in increased heart rate (HR), stroke volume, and overall cardiac output **(Choice A)**. At higher levels of exercise, HR is predominantly responsible for most of the increase in CO.

Sympathetic discharge causes contraction of the arterioles in all tissues except the actively working muscles, effectively shunting blood toward the exercising muscle. This also results in an increase in systolic blood pressure, which helps improve perfusion of actively contracting skeletal muscle **(Choice D)**. However, the rise in the mean arterial pressure is much lower due to an **overall decrease in systemic vascular resistance**. This occurs due to the substantial arteriolar vasodilation in active skeletal muscles, which is mediated by local release of adenosine, potassium ions, ATP, CO_2, and lactate.

(Choice B) Left ventricular (LV) end-diastolic volume and pressure both rise during exercise due to an increase in LV filling (increased venous return) by peripheral vasoconstriction and the pumping actions of actively contracting muscles.

(Choice C) Pulmonary artery systolic pressure typically rises during exercise; however, similar to systemic circulation, the rise in pulmonary pressure is much smaller than the increase in blood flow across the pulmonary circulation due to a fall in pulmonary vascular resistance.

Educational objective:
Exercising muscles can receive up to 85% of the total cardiac output during periods of strenuous activity. Although sympathetic discharge during exercise causes increased cardiac output and splanchnic vasoconstriction, there is only a modest increase in mean blood pressure as vasodilation within active skeletal muscles significantly decreases the total systemic vascular resistance.

References

- Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes.
- Hemodynamic and neurohumoral responses to dynamic exercise: normal subjects versus patients with heart disease.

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A group of forensic pathologists are analyzing tissue samples of adolescents age 13-18 to study the aging process. Autopsy of a 14-year-old boy who died in a motor vehicle accident shows several minimally raised yellow spots on the inner surface of the abdominal aorta. The rest of the cardiovascular findings during the autopsy are unremarkable. He had no known medical problems. There was no family history of cardiovascular disease or sudden cardiac death. Which of the following is most likely to be the predominant cell type in these lesions on light microscopy?

- A. Eosinophils
- B. Fibroblasts
- C. Macrophages
- D. Mast cells
- E. Neutrophils

Submit

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- A. Eosinophils (1%)
- ✗ B. Fibroblasts (19%)
- ✓ C. Macrophages (71%)
- D. Mast cells (1%)
- E. Neutrophils (7%)

Incorrect
Correct answer
C

71%
Answered correctly

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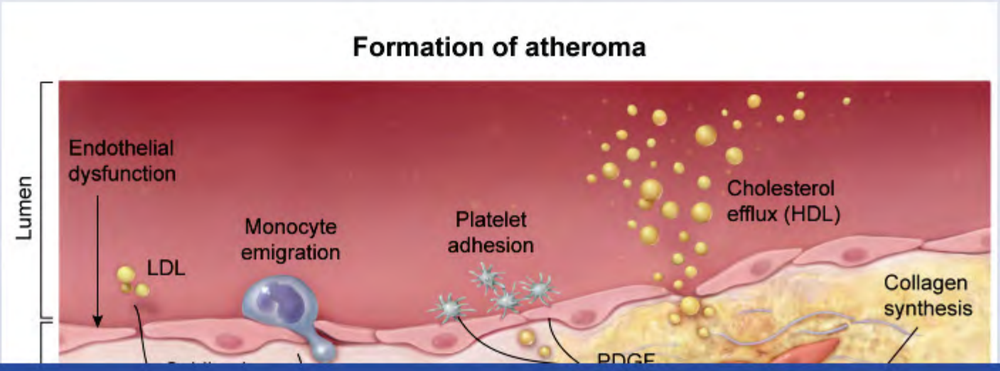

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A group of forensic pathologists are analyzing tissue samples of adolescents age 13-18 to study the aging proces
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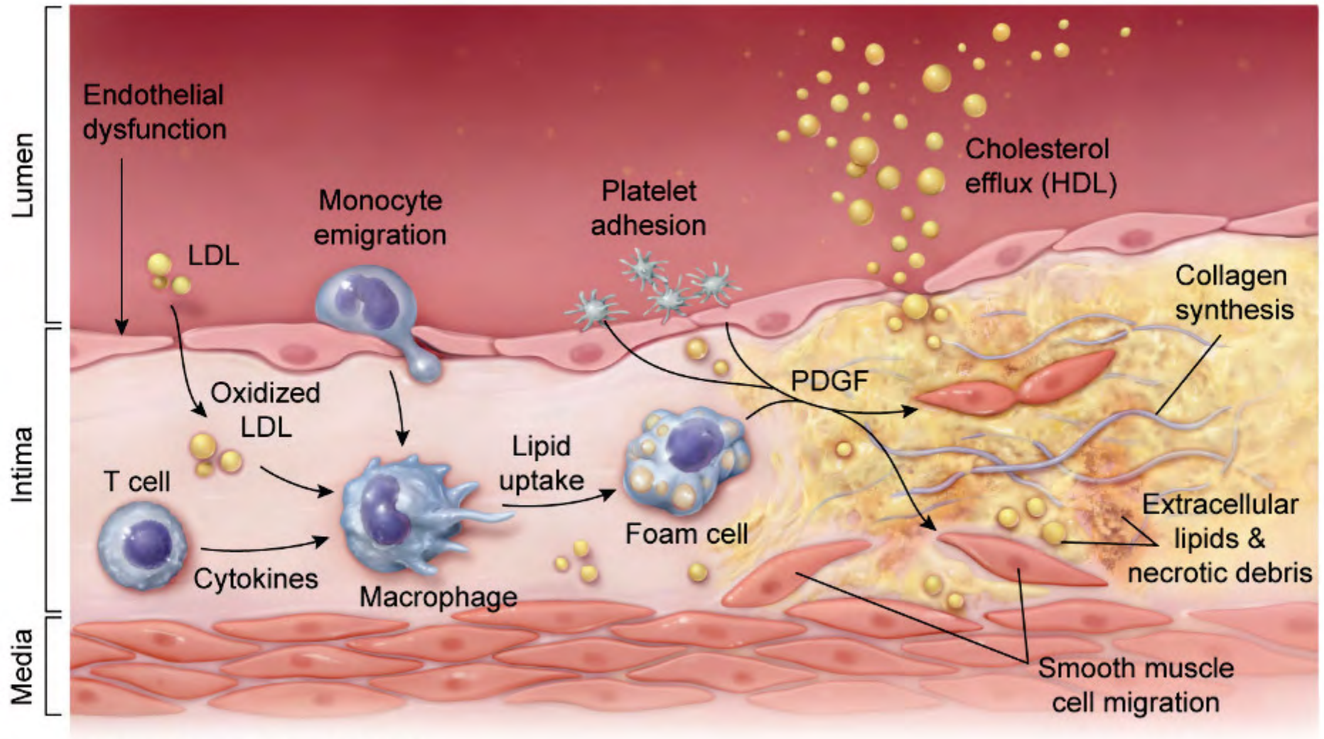
Formation of atheroma



PDGF = platelet-derived growth factor.

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PDGF = platelet-derived growth factor.
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The lesions described during autopsy on the inner surface of the abdominal aorta are consistent with **fatty streaks**. These are the earliest lesions in the progression to atherosclerosis and can be seen as early as the second decade of life. Atherosclerosis is a pathophysiologic process that begins in childhood with the development of focal intimal thickening and lipid accumulation. It progresses in the following sequence:

- **Endothelial injury/dysfunction** causing increased vascular permeability and enhanced leukocyte adhesion
- Accumulation of lipoproteins (LDL) within the vessel wall
- **Monocyte adhesion** to the endothelium, followed by migration and transformation into lipid-laden **macrophages (foam cells)**
- Release of growth factors by adherent platelets, activated macrophages, and endothelial cells
- Recruitment of T cells and **smooth muscle cell proliferation**, which lead to extracellular matrix production

With advancing age, the chronic inflammatory process initiated by endothelial injury transitions from fatty streaks within the intima (composed mainly of lipid-laden foam cells) into atherosclerotic plaques, such as fibrous cap atheromas and fibrous plaques.

(Choices A, B, D, and E) Eosinophils, fibroblasts, mast cells, and neutrophils are not the predominant cell types found in fatty streaks.

Educational objective:
Fatty streaks are the earliest lesions of atherosclerosis and can be seen as early as the second decade of life. They appear as a collection of lipid-laden macrophages (foam cells) in the intima that can eventually progress to atherosclerotic plaques.

References

- Pathogenesis of atherosclerosis.

Pathology	Cardiovascular System	Atherosclerosis
Subject	System	Topic

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A 53-year-old woman comes to the office due to aching pain in her legs. She is a hair stylist and has cut down on her work hours due to the discomfort. The patient has a history of migraine and untreated chronic hepatitis C. She smokes a pack of cigarettes daily but does not use alcohol or recreational drugs. Physical examination findings are shown in the image below.

An abnormality affecting which of the following is the most likely cause of this patient's symptoms?

- A. Arterial intima
- B. Capillary permeability

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An abnormality affecting which of the following is the most likely cause of this patient's symptoms?

- A. Arterial intima (1%)
- B. Capillary permeability (1%)
- C. Lymphoid capillaries (0%)
- D. Plasma oncotic pressure (5%)
- ✗ E. Vasa vasorum (3%)
- ✓ F. Venous valves (87%)

Incorrect
Correct answer
F

87%
Answered correctly

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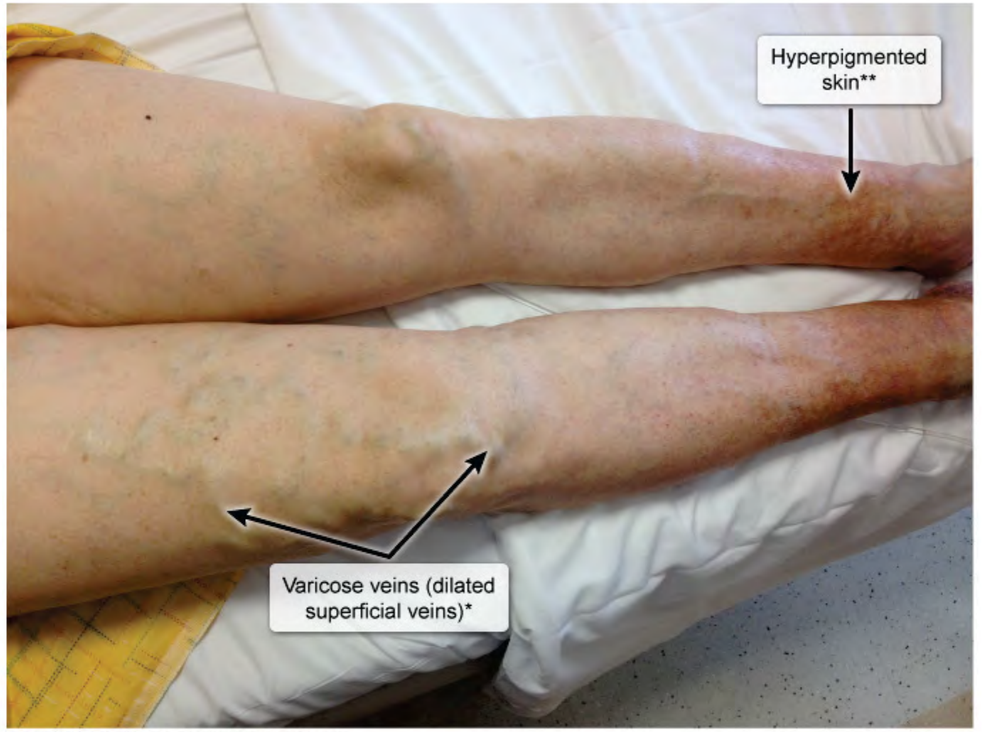
Varicose veins



*Due to chronically elevated venous pressure.
**Due to hemosiderin deposition in dermis.

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extremities. Blood from the deep veins then travels upward and eventually drains into the inferior vena cava.

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This patient has dilated superficial veins (ie, **varicose veins**) and associated skin changes consistent with stasis dermatitis. Venous blood normally flows from superficial veins through perforating veins into the deep veins of the extremities. Blood from the deep veins then travels upward and eventually drains into the inferior vena cava. Valves located in these vessels prevent backward flow from deep to superficial veins.

Chronically elevated intraluminal pressure can lead to dilation of the veins (varicose veins) and **incompetence of the valves**. This allows retrograde flow into superficial veins and results in a further increase in venous pressure. The resulting tissue ischemia causes **venous stasis dermatitis** and is associated with poor wound healing.

Common risk factors for varicose veins and stasis dermatitis include obstruction of venous return (eg, obesity, pregnancy) and conditions that damage the venous valves (eg, deep venous thrombosis). Prolonged standing and sedentary lifestyles are also associated with varicose veins.

(Choice A) Atherosclerotic changes of the arterial intima predispose to peripheral arterial disease. Arterial occlusive disease of the lower extremities presents with leg pain during exercise (claudication) and, if severe, ischemic pain at rest and possible distal gangrene.

(Choice B) Increased capillary permeability is associated with anaphylaxis and other allergic reactions, inflammation, and shock. Increased capillary permeability causes edema due to the extravasation of serum proteins into the interstitium. Capillary permeability does not play a role in the development of varicose veins.

(Choice C) Obstruction of lymphoid capillaries produces lymphedema, with marked swelling of the distal limb that may extend proximally. It is initially soft and pitting but eventually becomes firm and nonpitting due to progressive fibrosis and thickening of the overlying skin.

(Choice D) Decreased plasma oncotic pressure results primarily from low plasma protein content (eg, nephrotic syndrome, cirrhosis/liver failure) and results in generalized edema. Cirrhosis with portal hypertension can cause esophageal and anorectal varices as collateral channels from the portal to systemic circulation. However, lower extremity veins do not communicate with the portal circulation.

(Choice E) Narrowing of the arterioles of the vasa vasorum in chronic hypertension can lead to medial ischemia of

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(Choice E) Narrowing of the arterioles of the vasa vasorum in chronic hypertension can lead to medial ischemia of the aorta and contribute to aneurysm formation.

Educational objective:
Chronically elevated venous pressure in the lower extremities can lead to incompetent venous valves and venous dilation (varicose veins). Venous congestion and tissue ischemia can result in venous stasis dermatitis.

Pathophysiology	Cardiovascular System	Chronic venous insufficiency
Subject	System	Topic

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This patient has dilated superficial veins (ie, **varicose veins**) and associated skin changes consistent with stasis derma
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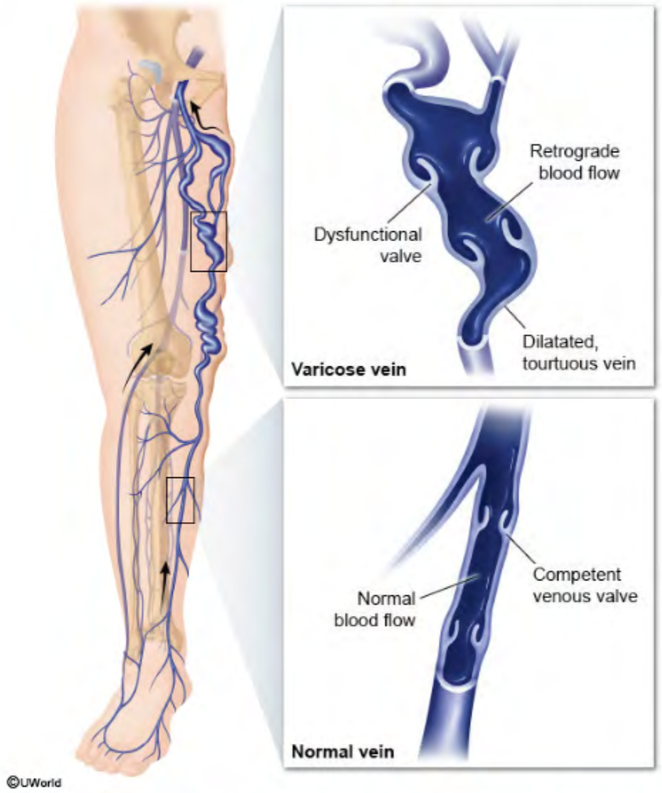

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Chronically elevated venous pressure in the lower extremities can lead to incompetent venous valves and venous

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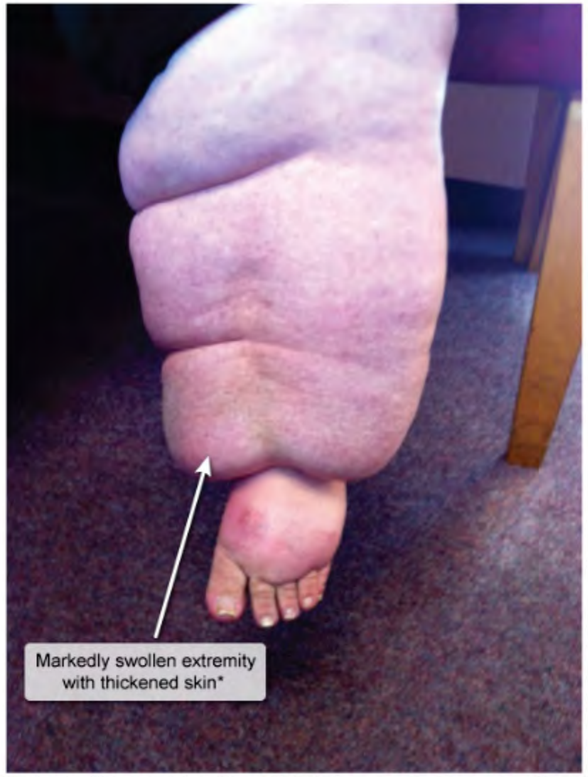
Lymphedema



*Due to interstitial accumulation of lymphatic fluid.

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 67-year-old man comes to the emergency department due to progressive shortness of breath and chest tightness. He has had no lightheadedness or syncope. The patient takes lisinopril for hypertension and metformin for type 2 diabetes mellitus. He has smoked a pack of cigarettes daily for the last 40 years. The blood pressure cuff is inflated to 140 mm Hg and the pressure is released very slowly. At 120 mm Hg, intermittent Korotkoff sounds are heard only during expiration. At 100 mm Hg, Korotkoff sounds are heard throughout the respiratory cycle.

Item 1 of 2

This physical examination finding can be seen in which of the following conditions?

- A. Aortic valve disease
- B. Mitral valve disease
- C. Myocardial ischemia
- D. Pericardial disease
- E. Peripheral vascular disease

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Item 2 of 2

The patient is tachypneic and unable to speak in full sentences. Examination reveals prolonged expiration and prominent bilateral wheezing. Heart sounds are normal. Chest imaging shows a normal-sized heart and hyperinflated lungs with a flattened diaphragm. Bedside echocardiogram reveals no intrapericardial fluid accumulation or pericardial thickening. Which of the following physiologic changes is most likely to provide immediate relief in this patient?

- A. cAMP accumulation in smooth muscle cells
- B. Cell membrane stabilization of mast cells
- C. Inhibition of eosinophil degranulation
- D. Interrupted histamine receptor firing in epithelial cells
- E. Opening of calcium channels in smooth muscle cells

Submit

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Item 1 of 2

This physical examination finding can be seen in which of the following conditions?

- ✗ A. Aortic valve disease (24%)
- B. Mitral valve disease (9%)
- C. Myocardial ischemia (5%)
- ✓ D. Pericardial disease (44%)
- E. Peripheral vascular disease (15%)

Incorrect
Correct answer
D

44%
Answered correctly

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Explanation

Systemic arterial pressure normally falls by <10 mm Hg during normal inspiration. Pulsus paradoxus refers to an

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Explanation

Systemic arterial pressure normally falls by <10 mm Hg during normal inspiration. Pulsus paradoxus refers to an exaggerated drop (**>10 mm Hg**) in systolic blood pressure during **inspiration**. It is detected by inflating a blood pressure cuff above systolic pressure and gradually deflating it. The difference between the systolic pressure at which Korotkoff sounds first become audible during expiration and the pressure at which they are heard throughout all phases of respiration quantifies pulsus paradoxus (20 mm Hg in this patient).

Inspiration causes an increase in systemic venous return, resulting in increased right heart volumes. Under normal conditions, this results in expansion of the right ventricle into the pericardial space with little impact on the left side of the heart. However, in conditions that impair expansion into the pericardial space (eg, acute cardiac tamponade), the increased right ventricular volume occurring with inspiration leads to **bowing of the interventricular septum** toward the left ventricle. This leads to a decrease in left ventricular (LV) end-diastolic volume and stroke volume, with a resultant decrease in systolic pressure during inspiration.

(Choice A) Fixed LV outflow tract obstruction (eg, valvular aortic stenosis) can cause pulsus parvus et tardus, which is palpable as a slow-rising, low-amplitude pulse due to diminished stroke volume (pulsus parvus) and prolonged LV ejection time (pulsus tardus).

(Choice B) Patients with chronic severe mitral regurgitation have a bounding pulse with a brisk upstroke due to increased LV ejection volumes. Arterial pulses are overall reduced in amplitude in patients with mitral stenosis due to reduced stroke volume.

(Choice C) Myocardial ischemia or infarction can lead to decreased systemic blood pressure or hypotension due to reduced cardiac output, but it would not be expected to cause pulsus paradoxus.

(Choice E) Peripheral vascular disease causes decreased pulse amplitude within the affected extremities. This is most commonly observed in the lower extremities, which will have diminished or absent distal pulses caused by widespread peripheral arterial disease.

Educational objective:

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(Choice E) Peripheral vascular disease causes decreased pulse amplitude within the affected extremities. This is most commonly observed in the lower extremities, which will have diminished or absent distal pulses caused by widespread peripheral arterial disease.

Educational objective:
Pulsus paradoxus is defined by a decrease in systolic blood pressure of >10 mm Hg with inspiration. It is most commonly seen in patients with cardiac tamponade but can also occur in severe asthma, chronic obstructive pulmonary disease, and constrictive pericarditis.

References

- Pulsus paradoxus.

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Explanation

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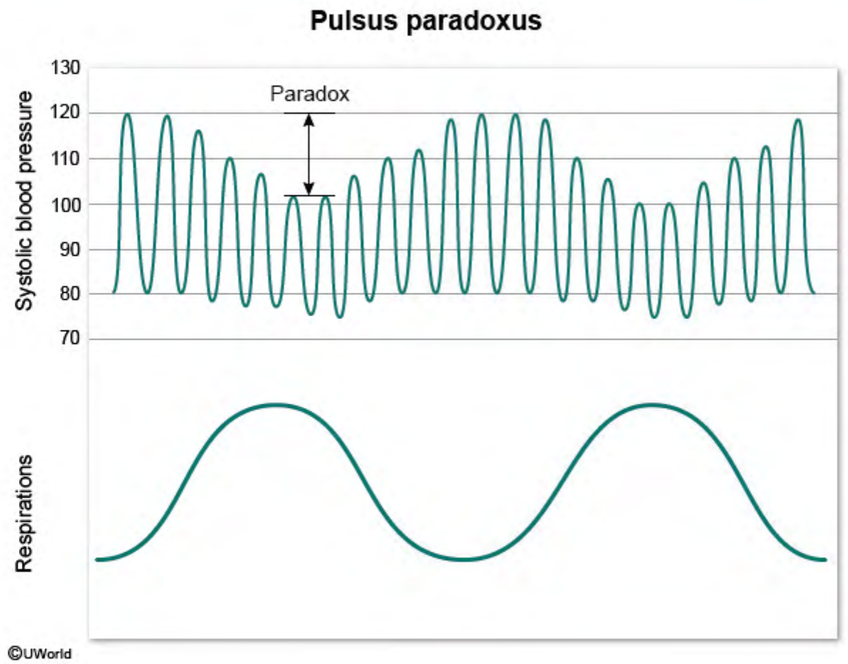

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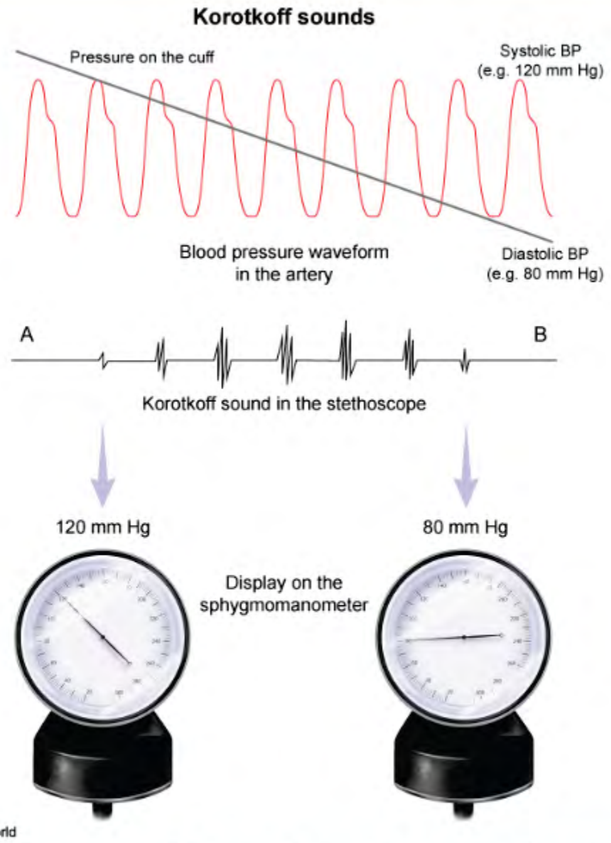

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Educational objective:

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Item 2 of 2

The patient is tachypneic and unable to speak in full sentences. Examination reveals prolonged expiration and prominent bilateral wheezing. Heart sounds are normal. Chest imaging shows a normal-sized heart and hyperinflated lungs with a flattened diaphragm. Bedside echocardiogram reveals no intrapericardial fluid accumulation or pericardial thickening. Which of the following physiologic changes is most likely to provide immediate relief in this patient?

- ✓ A. cAMP accumulation in smooth muscle cells (57%)
- ✗ B. Cell membrane stabilization of mast cells (10%)
- C. Inhibition of eosinophil degranulation (7%)
- D. Interrupted histamine receptor firing in epithelial cells (9%)
- E. Opening of calcium channels in smooth muscle cells (15%)

Incorrect
Correct answer
A

57%
Answered correctly

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Time Spent

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Version

Explanation

This patient's presentation with dyspnea, tachypnea, prolonged expiration, and bilateral wheezing is suggestive of acute exacerbation of **obstructive pulmonary disease** (asthma or COPD). Acute obstructive pulmonary exacerbations are primarily treated with **beta-adrenergic agonists**, which produce relaxation of bronchial smooth muscle by stimulating the beta-2-adrenergic receptor. This receptor is a G_s protein-coupled receptor that activates adenylyl cyclase and **increases intracellular cAMP** concentrations.

Asthma and COPD exacerbation are the most frequent causes of pulsus paradoxus in the absence of significant

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A

Explanation

This patient's presentation with dyspnea, tachypnea, prolonged expiration, and bilateral wheezing is suggestive of acute exacerbation of **obstructive pulmonary disease** (asthma or COPD). Acute obstructive pulmonary exacerbations are primarily treated with **beta-adrenergic agonists**, which produce relaxation of bronchial smooth muscle by stimulating the beta-2-adrenergic receptor. This receptor is a G_s protein-coupled receptor that activates adenylyl cyclase and **increases intracellular cAMP** concentrations.

Asthma and COPD exacerbation are the most frequent causes of pulsus paradoxus in the absence of significant pericardial disease (pericardial effusion or constrictive pericarditis). There is a small normal variation in intrathoracic pressure with respiration, which drops 2-5 mm Hg below the atmospheric pressure during inspiration. In patients with severe asthma or COPD exacerbation, this drop in intrathoracic pressure becomes greatly exaggerated and is transmitted to extrathoracic structures. This leads to an excessive drop in blood pressure with inspiration that is detected as pulsus paradoxus.

(Choice B) Cromolyn sodium inhibits mast cell degranulation and release of histamine and leukotrienes. It is used occasionally for management of chronic asthma but is not effective for immediate relief of acute asthmatic or COPD exacerbations.

(Choice C) Eosinophil degranulation would be impaired by corticosteroids, which play an important role in treatment of acute asthma or COPD exacerbations. However, beta agonists provide immediate relief, whereas corticosteroids would take several hours to a few days to take effect.

(Choice D) Histamine is one of several mediators released in response to a hypersensitivity reaction that can lead to bronchial smooth muscle contraction. However, antihistamine therapy is not an effective treatment for asthma or COPD exacerbation.

(Choice E) Calcium channel agonists are not used in acute treatment of asthma or COPD exacerbation. On the

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muscle by stimulating the beta-2-adrenergic receptor. This receptor is a G_s protein-coupled receptor that activates adenylyl cyclase and **increases intracellular cAMP** concentrations.

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(Choice D) Histamine is one of several mediators released in response to a hypersensitivity reaction that can lead to bronchial smooth muscle contraction. However, antihistamine therapy is not an effective treatment for asthma or COPD exacerbation.

(Choice E) Calcium channel agonists are not used in acute treatment of asthma or COPD exacerbation. On the contrary, increased calcium influx could potentially worsen bronchial smooth muscle contraction.

Educational objective:
Asthma and chronic obstructive pulmonary disease (COPD) exacerbation are the most frequent causes of pulsus paradoxus in the absence of significant pericardial disease. Beta-adrenergic agonists control acute asthma and COPD exacerbations by causing bronchial smooth muscle relaxation via increased intracellular cAMP.

References

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A 75-year-old man is brought to the emergency department after his family found him to be lethargic and confused. The patient has a history of hypertension, coronary artery disease, and heart failure. Temperature is 35 C (95 F), blood pressure is 78/46 mm Hg, and pulse is 110/min. The patient is admitted to the intensive care unit and a pulmonary artery catheter is placed, which demonstrates the following readings:

Cardiac output	high
Central venous pressure	low
Pulmonary capillary wedge pressure	low
Mixed venous O_2 saturation	high
Systemic vascular resistance	low

Which of the following is the most likely cause of this patient's hypotension?

- A. Bacterial infection
- B. Gastrointestinal bleeding
- C. Heart failure
- D. Hypothermia
- E. Myocardial infarction
- F. Pulmonary embolism

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Cardiac output	high
Central venous pressure	low
Pulmonary capillary wedge pressure	low
Mixed venous O_2 saturation	high
Systemic vascular resistance	low

Which of the following is the most likely cause of this patient's hypotension?

- ✓ A. Bacterial infection (44%)
- B. Gastrointestinal bleeding (21%)
- C. Heart failure (7%)
- D. Hypothermia (14%)
- E. Myocardial infarction (2%)
- F. Pulmonary embolism (9%)

Correct

44% Answered correctly

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Explanation

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Hemodynamic measurements in shock				
Parameter	**Hypovolemic shock**	**Cardiogenic shock**	**Obstructive shock**	**Distributive shock**
CVP (right-sided preload)	↓	↑	↑	↓
PCWP (left-sided preload)	↓	↑	↓*	↓
Cardiac index (LV output)	↓	↓	↓	↑**
SVR (afterload)	↑	↑	↑	↓
SvO_2	↓	↓	↓	↑**

*In tamponade, left-sided preload is decreased, but measured PCWP is paradoxically increased due to external compression by pericardial fluid.

**Cardiac index & SvO_2 are usually decreased in neurogenic shock due to impaired sympathetic reflexes.

CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **SvO_2** = mixed venous oxygen saturation; **SVR** = systemic vascular resistance.

This patient with severe **hypotension** and evidence of **end-organ dysfunction** (eg, confusion) is in **shock**, which is a state of **inadequate oxygen delivery** to the organs and tissues of the body. Left untreated, shock can rapidly lead to multiorgan failure and death; therefore, prompt diagnosis and treatment are essential. Pulmonary arterial

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This patient with severe **hypotension** and evidence of **end-organ dysfunction** (eg, confusion) is in **shock**, which is a state of **inadequate oxygen delivery** to the organs and tissues of the body. Left untreated, shock can rapidly lead to multiorgan failure and death; therefore, prompt diagnosis and treatment are essential. Pulmonary arterial catheterization can help determine the type of shock to help guide treatment.

The **low systemic vascular resistance** (SVR) in this patient is consistent with the peripheral vasodilation that occurs in **distributive shock**; septic shock (usually due to **bacterial infection**) is the most common type of distributive shock. Central venous pressure (CVP), or right-sided preload, is decreased in septic shock due to widespread vasodilation in response to systemic release of proinflammatory mediators. Pulmonary capillary wedge pressure (PCWP), which is an estimate of left atrial pressure (left-sided preload), is decreased as well due to reduced venous return. A compensatory increase in sympathetic drive increases cardiac contractility and heart rate resulting in **increased cardiac output (CO)**. The high rate of blood flow through the systemic capillaries prevents complete extraction of oxygen by the tissues, resulting in **high mixed venous oxygen saturation**.

(Choice B) Gastrointestinal bleeding can lead to hemorrhagic (hypovolemic) shock characterized by low CVP, PCWP, and CO, with a compensatory increase in SVR.

(Choices C and E) Myocardial infarction can lead to heart failure and cardiogenic shock, characterized by high PCWP and CVP. CO decreases, with a compensatory increase in SVR.

(Choice D) Moderate to severe hypothermia (ie, <32 C) can produce hypotension due to bradycardia, but mild hypothermia does not typically lead to shock. Septic shock can present with either hyper- or hypothermia; **hypothermic presentation** may be due to cytokine-induced dysregulation of temperature control in the hypothalamus and is associated with poorer outcomes.

(Choice F) Acute pulmonary embolism is a potential cause of obstructive shock. Blood flow through the pulmonary circulation is impeded, resulting in reduced left-sided preload (low PCWP) and consequently reduced CO with a compensatory increase in SVR. Blood backs up in the capacitance veins leading to high CVP.

Educational objective:

Septic shock can present with either hyper- or hypothermia. The initial disturbance is peripheral vasodilation

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Educational objective:
Septic shock can present with either hyper- or hypothermia. The initial disturbance is peripheral vasodilation leading to decreased systemic vascular resistance, decreased central venous pressure, and decreased pulmonary capillary wedge pressure. A compensatory increase in sympathetic drive causes an increase in cardiac output; the resulting high flow rates lead to incomplete oxygen extraction in the tissues, resulting in high mixed venous oxygen saturation.

References

- Pulmonary artery catheterization in critically ill patients: a prospective analysis of outcome changes associated

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Educational objective:
Septic shock can present with either hyper- or hypothermia. The initial disturbance is peripheral vasodilation

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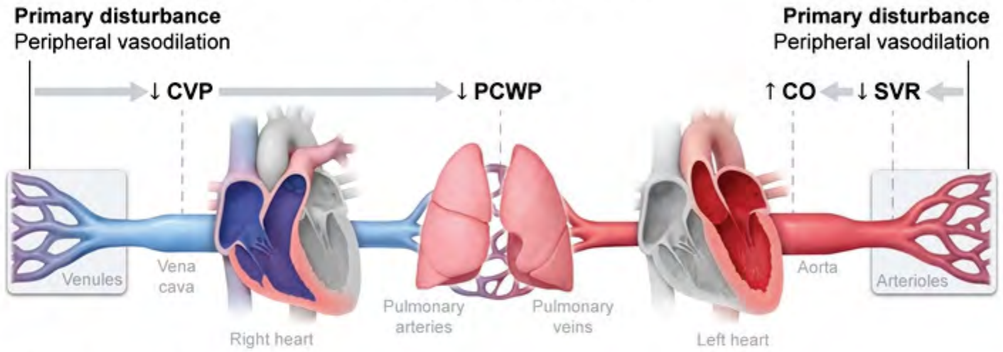
Septic (distributive) shock



CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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Educational objective:
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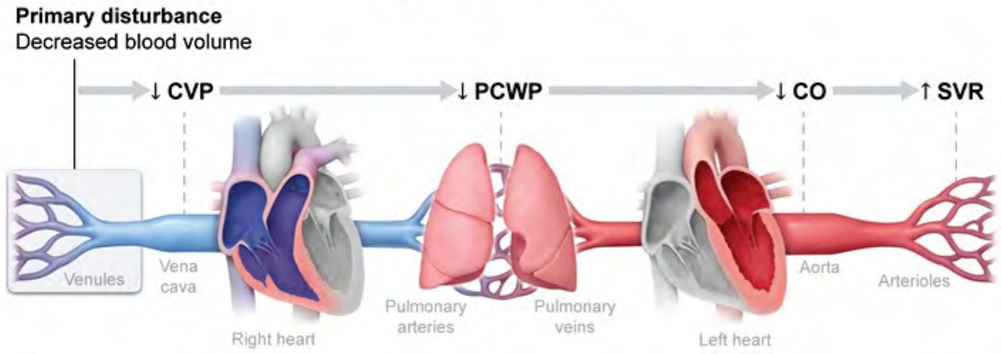
Hypovolemic shock



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Educational objective:
Septic shock can present with either hyper- or hypothermia. The initial disturbance is peripheral vasodilation

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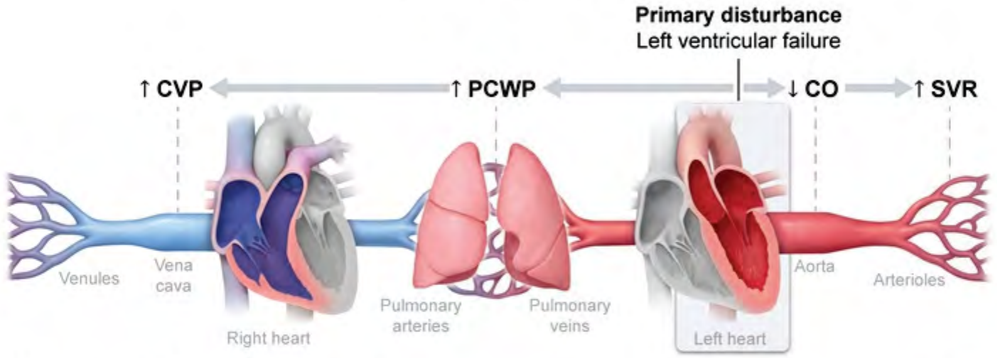
Cardiogenic shock



CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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catheterization can help determine the type of shock to help guide treatment.

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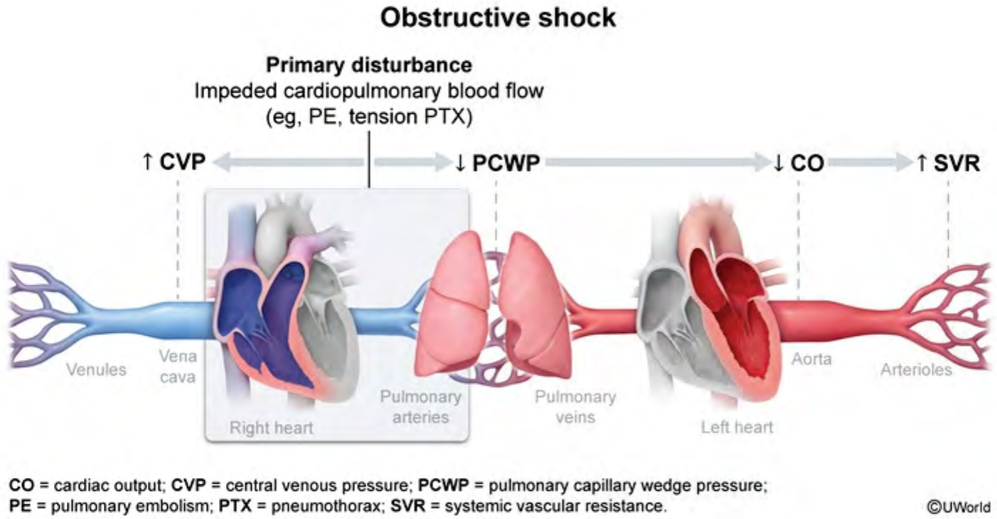

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A 43-year-old man comes to the emergency department due to a 3-day history of persistent headaches. The patient has a history of hypertension and has had poor medical follow-up. Blood pressure is 224/115 mm Hg and pulse is 67/min. He appears mildly confused during the physical examination, but no focal neurologic deficits are noted. Funduscopic examination shows bilateral papilledema. Serum creatinine is 1.4 mg/dL. An intravenous medication is initiated that causes arteriolar dilation while also improving renal perfusion and increasing natriuresis. Which of the following agents is most likely being used in this patient?

- A. Esmolol
- B. Fenoldopam
- C. Hydralazine
- D. Nitroglycerin
- E. Phenylephrine

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- A. Esmolol (5%)
- ✓ B. Fenoldopam (52%)
- C. Hydralazine (34%)
- D. Nitroglycerin (6%)
- E. Phenylephrine (1%)

Correct

52% Answered correctly

03 secs Time Spent

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Explanation

Hypertensive crisis	
Hypertensive urgency	• Severe hypertension (≥180/120 mm Hg) • No evidence of end-organ damage
	• Severe hypertension with end-organ damage: ◦ Cardiovascular: angina, myocardial infarction, aortic dissection

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Hypertensive crisis	
Hypertensive urgency	• Severe hypertension (≥180/120 mm Hg) • No evidence of end-organ damage
Hypertensive emergency	• Severe hypertension with end-organ damage: ◦ Cardiovascular: angina, myocardial infarction, aortic dissection ◦ CNS: encephalopathy, cerebral infarct/hemorrhage ◦ Kidney: acute hypertensive nephrosclerosis ◦ Lung: pulmonary edema ◦ Retina: papilledema, flame-shaped hemorrhages, exudates

This patient has **severe hypertension**. In addition, he has clinical evidence of secondary **end-organ damage**, including encephalopathy (eg, confusion), papilledema, and acute kidney injury (eg, elevated creatinine). This presentation is consistent with **hypertensive emergency** and requires aggressive treatment with immediate but controlled blood pressure reduction to minimize organ damage. Intravenous agents are preferred in hypertensive emergency due to their rapid action and dose titration ability.

Fenoldopam is a short-acting, selective, peripheral **dopamine-1 receptor agonist** with little to no effect on alpha- or beta-adrenergic receptors. Dopamine-1 receptor stimulation activates adenylyl cyclase and raises intracellular cyclic AMP, resulting in vasodilation of most arterial beds with a corresponding decrease in systemic blood pressure. **Renal vasodilation** is particularly prominent and leads to **increased renal perfusion**, increased urine output, and **natriuresis** (ie, sodium excretion). This makes fenoldopam especially beneficial in patients with hypertensive emergency and renal insufficiency.

(Choice A) Esmolol is a short-acting, cardioselective beta-1 receptor antagonist. It works by reducing heart rate and myocardial contractility. Esmolol is not a vasodilator and does not increase renal perfusion.

(Choice C) Hydralazine is a direct arteriolar vasodilator; however, it does not dilate renal arterioles and may decrease renal perfusion and encourage sodium and fluid retention. It is not often used in hypertensive

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(Choice C) Hydralazine is a direct arteriolar vasodilator; however, it does not dilate renal arterioles and may decrease renal perfusion and encourage sodium and fluid retention. It is not often used in hypertensive emergency because it is associated with reflex sympathetic activation, resulting in increased heart rate and contractility.

(Choice D) Nitroglycerin is a rapid-acting venodilator that decreases preload and cardiac output. It causes only minimal arterial dilation and does not improve renal perfusion. Nitroglycerin is primarily used to reduce myocardial oxygen demand in acute coronary syndrome.

(Choice E) Phenylephrine is an alpha-adrenergic agonist that causes an increase in systemic vascular resistance due to arterial vasoconstriction. It is used in patients with hypotension or shock and is contraindicated in hypertensive emergency.

Educational objective:
Fenoldopam is a selective peripheral dopamine-1 receptor agonist. It causes vasodilation of systemic and renal arterioles to lower blood pressure while also increasing renal perfusion, urine output, and natriuresis, making it useful in the treatment of hypertensive emergency in patients with renal insufficiency.

References

- Fenoldopam.

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A 45-year-old man is brought to a rural emergency department due to severe chest pain, sweating, and nausea. The symptoms began suddenly an hour ago. He has no significant medical history. His father died at age 50 after experiencing sudden-onset chest pain. The patient smokes a pack of cigarettes daily. He does not take any medications and has no known drug allergies. Examination shows normal heart sounds and breath sounds. ECG shows sinus tachycardia with ST segment elevation in leads II, III, and aVF. Medical management for the patient's acute condition is initiated. After initial treatment, the chest pain decreases in intensity and a reperfusion complex ventricular arrhythmia emerges. The arrhythmia is asymptomatic and resolves spontaneously. Which of the following drugs is most likely responsible for rapid reperfusion in this patient?

- A. Alteplase
- B. Apixaban
- C. Argatroban
- D. Aspirin
- E. Heparin
- F. Prasugrel
- G. Rosuvastatin

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- ✓ A. Alteplase (71%)
- B. Apixaban (1%)
- C. Argatroban (2%)
- D. Aspirin (9%)
- E. Heparin (11%)
- F. Prasugrel (2%)
- G. Rosuvastatin (0%)

Correct

71% Answered correctly

50 secs Time Spent

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Explanation

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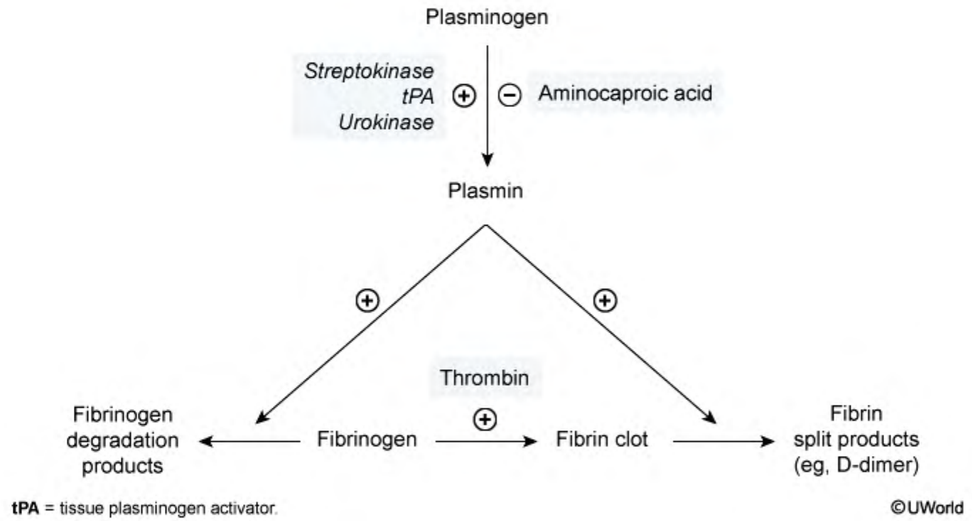
G. Rosuvastatin (0%)

Exhibit Display

Fibrinolytic pathway



tPA = tissue plasminogen activator.

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of fibrin clot and often restoration of myocardial perfusion; some patients develop a self-limiting reperfusion-related arrhythmia (most commonly an accelerated idioventricular rhythm).

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tPA = tissue plasminogen activator.

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This patient had an **ST-elevation myocardial infarction** (STEMI) and, following medical treatment, experienced symptomatic improvement and reperfusion-related arrhythmia; this is most likely the result of **fibrinolytic (thrombolytic) therapy**. Fibrinolytic agents (eg, **alteplase**) are indicated in patients with acute STEMI who cannot receive percutaneous coronary intervention in a timely manner. Administration of these agents leads to breakdown of fibrin clot and often restoration of myocardial perfusion; some patients develop a self-limiting reperfusion-related arrhythmia (most commonly an accelerated idioventricular rhythm).

In the **fibrinolytic pathway**, tissue plasminogen activator (tPA) is released by the endothelium and cleaves plasminogen to form plasmin. Plasmin then acts to both degrade individual fibrinogen molecules and break down fibrin clot that has already formed. The breakdown of fibrin clot leads to elevated levels of fibrin split products (eg, d-dimer) in the blood.

Pharmacologic fibrinolytic agents that are used clinically include streptokinase and recombinant forms of tPA (eg, alteplase, tenecteplase). All of these agents inherently increase bleeding risk and are contraindicated in patients with increased risk of bleeding complications (eg, recent hemorrhagic stroke).

(Choices B and C) Apixaban is a direct factor Xa inhibitor used in the treatment and prevention of venous thromboembolism, and argatroban is a direct thrombin inhibitor used to treat heparin-induced thrombocytopenia. Both of these drugs are anticoagulants; neither causes fibrinolysis or is typically used in the treatment of STEMI.

(Choices D and F) Aspirin inhibits cyclooxygenase to reduce thromboxane levels, and P2Y12 receptor blockers (eg, clopidogrel, prasugrel) prevent the binding of adenosine diphosphate to platelets; both actions lead to irreversible inhibition of platelet aggregation. These drugs are used together in acute STEMI to prevent the development of additional platelet thrombus; however, they do not cause lysis of existing thrombus.

(Choice E) Heparin potentiates the activity of antithrombin III to inhibit thrombin and factor Xa and exert an anticoagulation effect. Heparin is used in acute STEMI to prevent further progression of clot formation, but it does not cause thrombolysis.

(Choice G) Statins (eg, rosuvastatin) are used for acute STEMI as they may stabilize atherosclerotic plaques,

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(Choice G) Statins (eg, rosuvastatin) are used for acute STEMI as they may stabilize atherosclerotic plaques, reduce vascular inflammation, and promote long-term regression of atherosclerosis. However, they do not cause thrombolysis.

Educational objective:
In the fibrinolytic pathway, tissue plasminogen activator (tPA) converts plasminogen to plasmin, which then breaks down fibrin clot. The administration of a tPA analogue (eg, alteplase, tenecteplase, streptokinase) triggers fibrinolysis and can restore myocardial perfusion in patients with ST-elevation myocardial infarction who cannot undergo timely percutaneous coronary intervention.

References

- 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary: a

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of fibrin clot and often restoration of myocardial perfusion; some patients develop a self-limiting reperfusion-related arrhyth

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fibrinolysis and can restore myocardial perfusion in patients with ST-elevation myocardial infarction who cannot

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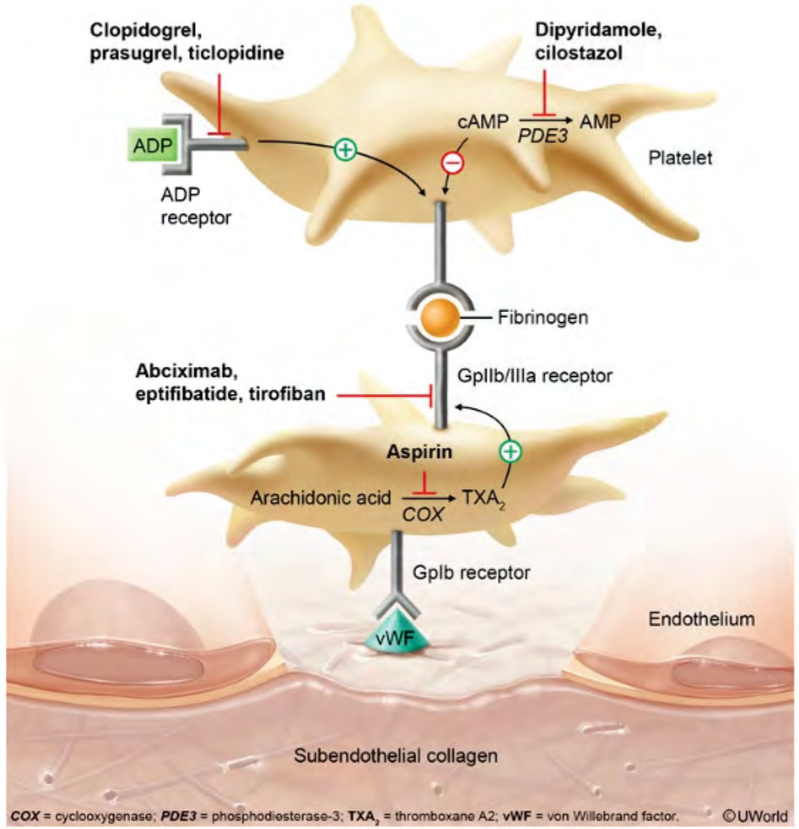
Antiplatelet medications



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A 23-year-old woman is brought to the emergency department with a chest wound. She was in her house during a severe thunderstorm when a large tree branch fell through a window. The window was shattered and large fragments of wood and glass struck the patient. She did not lose consciousness, but her family found her bleeding profusely and rushed her to the hospital. Blood pressure is 80/50 mm Hg and pulse is 130/min. The patient appears to be in severe distress. Physical examination shows a deep penetrating wound in the fourth intercostal space along the left sternal border. Smaller lacerations are noted across the face and forearms. Which of the following structures is most likely injured in this patient?

- A. Inferior vena cava
- B. Left atrium
- C. Left ventricle
- D. Pulmonary trunk
- E. Right ventricle

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- A. Inferior vena cava (1%)
- ✗ B. Left atrium (6%)
- C. Left ventricle (20%)
- D. Pulmonary trunk (6%)
- ✓ E. Right ventricle (65%)

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Correct answer
E

65%
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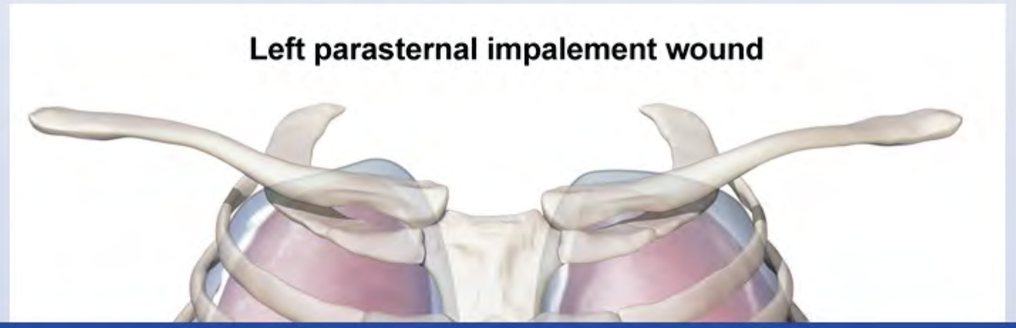
Explanation



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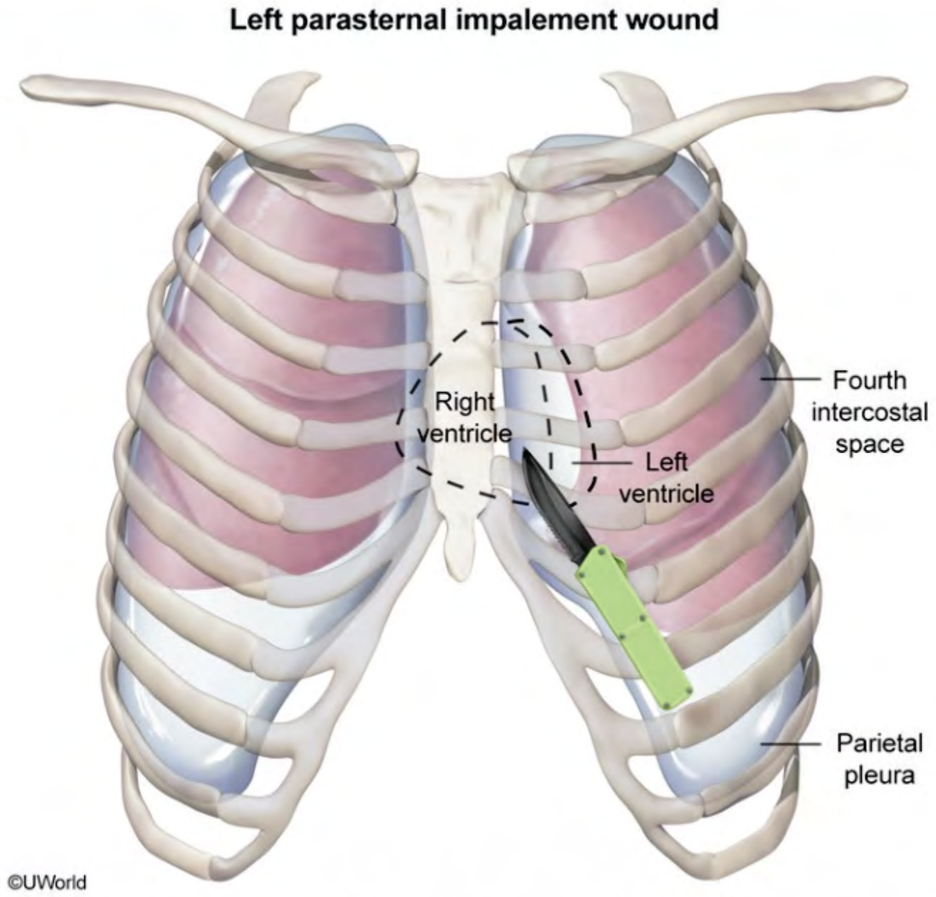

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A penetrating injury at the left sternal border in the fourth intercostal space (level of the nipple) will pass through the following layers (in order):

1. Skin and subcutis
2. Pectoralis major muscle
3. External intercostal membrane
4. Internal intercostal muscle
5. Internal thoracic artery and veins
6. Transversus thoracis muscle
7. Parietal pleura
8. Pericardium
9. Right ventricular myocardium

The **right ventricle** composes the majority of the **anterior surface of the heart** and is at risk of injury with penetrating trauma at the mid- and lower-left sternal border. The parietal pleura would be injured as well, but the left lung itself would not be punctured as there is no middle lobe on the left side, and the superior lobe of the left lung is displaced laterally by the cardiac impression.

(Choice A) The inferior vena cava (IVC) passes through the right side of the central tendon of the diaphragm at the level of T8. A penetrating wound to the back at the immediate right of the vertebral bodies could strike the IVC.

(Choice B) The left atrium is located at the base of the heart (posterior surface, opposite the apex) and makes up most of the heart's posterior surface. Only the auricle of the left atrium is visible anteriorly (protruding between the pulmonary trunk and the left ventricle).

(Choice C) The left ventricle composes the left lateral aspect of the heart. A stab wound angled slightly medially in the fourth intercostal space at the midclavicular line could strike the left ventricle, but only after passing through the bulk of the left lung.

(Choice D) The pulmonary trunk could be pierced by a penetrating injury to the second intercostal space at the left sternal border.

Educational objective:

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1. Skin and subcutis
2. Pectoralis major muscle
3. External intercostal membrane
4. Internal intercostal muscle
5. Internal thoracic artery and veins
6. Transversus thoracis muscle
7. Parietal pleura
8. Pericardium
9. Right ventricular myocardium

The **right ventricle** composes the majority of the **anterior surface of the heart** and is at risk of injury with penetrating trauma at the mid- and lower-left sternal border. The parietal pleura would be injured as well, but the left lung itself would not be punctured as there is no middle lobe on the left side, and the superior lobe of the left lung is displaced laterally by the cardiac impression.

(Choice A) The inferior vena cava (IVC) passes through the right side of the central tendon of the diaphragm at the level of T8. A penetrating wound to the back at the immediate right of the vertebral bodies could strike the IVC.

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(Choice D) The pulmonary trunk could be pierced by a penetrating injury to the second intercostal space at the left sternal border.

Educational objective:
The right ventricle composes most of the heart's anterior surface. A deep, penetrating injury at the left sternal border in the fourth intercostal space would puncture the right ventricle.

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A 45-year-old man who was recently started on medication for paroxysmal atrial fibrillation undergoes stress ECG testing. The patient exercises on a treadmill for 9 minutes and reaches 98% of age-predicted maximal heart rate without chest pain or ST-segment changes. His pre-test resting QRS complex duration is 95 msec (normal: 80-120 msec) and corrected QT (QTc) interval duration is 410 msec (normal ≤440 msec). ECG findings recorded at his maximally achieved heart rate show a QRS complex duration of 125 msec and QTc interval duration of 400 msec. Based on the patient's test results, which of the following medications is most likely being used to treat his atrial fibrillation?

- A. Atenolol
- B. Digoxin
- C. Dofetilide
- D. Flecainide
- E. Verapamil

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- ✗ A. Atenolol (10%)
- B. Digoxin (10%)
- C. Dofetilide (18%)
- ✓ D. Flecainide (44%)
- E. Verapamil (15%)

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Correct answer
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Answered correctly

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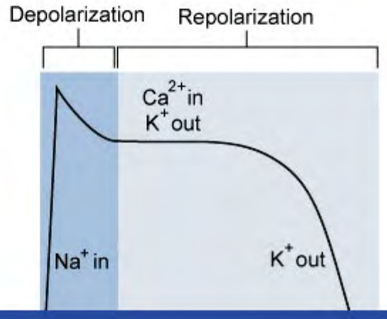
Explanation



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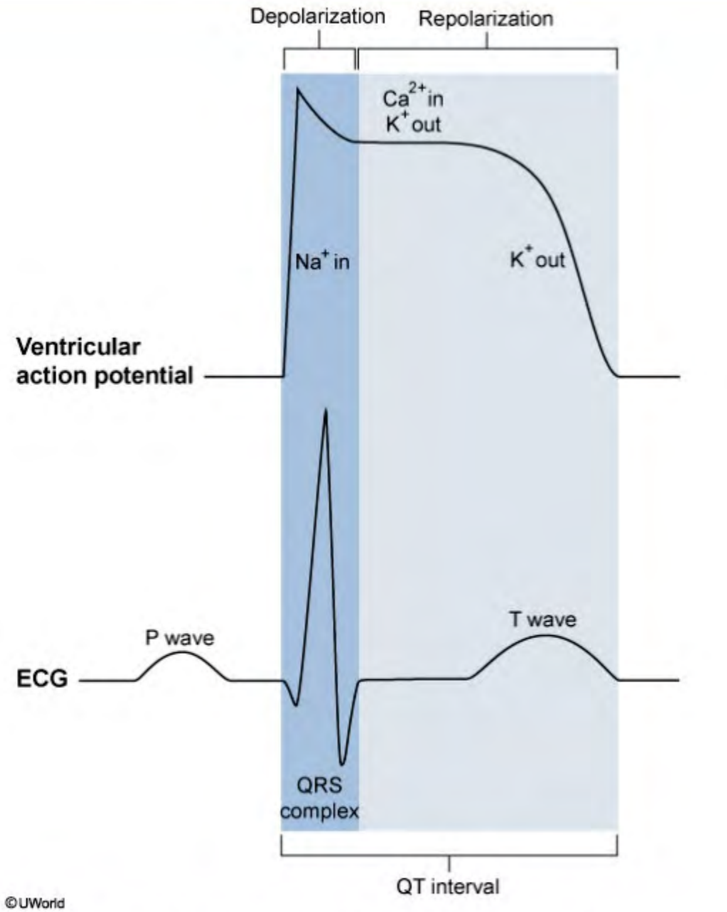

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The **QRS complex duration** is typically slightly **reduced during exercise** in response to the increase in cardiac conduction velocity that accompanies faster heart rates. However, this patient's QRS duration is normal at rest and prolonged at near-maximal heart rate. This suggests that the drug used to treat his atrial fibrillation lengthens the QRS duration in a rate-dependent manner, which is consistent with a drug exhibiting strong use-dependence such as flecainide.

Flecainide is a class 1C antiarrhythmic that is typically used to treat supraventricular tachycardias such as those caused by atrial fibrillation. Class 1C antiarrhythmics bind to the fast sodium channels responsible for phase 0 depolarization of cardiac myocytes, blocking the inward sodium current. This **prolongs the QRS duration** with little effect on total action potential or QT interval duration. Class 1C drugs are the slowest of the class 1 agents to dissociate from the sodium channel, and do so primarily during diastole. This results in a phenomenon known as **use-dependence**, in which their sodium-blocking effects intensify as the heart rate increases due to less time for the medication to dissociate from the receptor.

(Choice A) Selective β1 blockers such as atenolol do not significantly affect QRS duration. However, their use during exercise ECG testing can limit the maximal heart rate that is achieved.

(Choice B) Digoxin can cause false-positive ST-segment depression during exercise ECG testing as well as lower the maximal achievable heart rate. It does not significantly affect QRS duration.

(Choice C) Dofetilide is a class III antiarrhythmic drug that blocks the outward potassium current during repolarization. As a result, it increases the QT interval duration but does not affect QRS duration. Class III agents demonstrate reverse use-dependence (the slower the heart rate, the more the QT interval is prolonged).

(Choice E) Verapamil (a class IV antiarrhythmic) is a calcium channel blocker that slows sinoatrial and atrioventricular nodal conduction. It also increases coronary blood flow and reduces myocardial oxygen demand, which can mask ischemia during stress testing. Verapamil does not affect QRS duration.

Educational objective:
Class 1C antiarrhythmics such as flecainide are potent sodium channel blockers that have increased effect at faster heart rates (use-dependence). This makes them more effective at treating tachyarrhythmias, but can also cause prolonged QRS duration (a proarrhythmic effect) at higher heart rates.

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Class I (sodium channel blocking) antiarrhythmics			
	Specific agents	**Inhibition of phase 0 depolarization**	**Effect on length of action potential**
Class IA	Quinidine, procainamide, disopyramide	Intermediate	Prolonged
Class IB	Lidocaine, mexiletine	Weak	Shortened
Class IC	Flecainide, propafenone	Strong	No change

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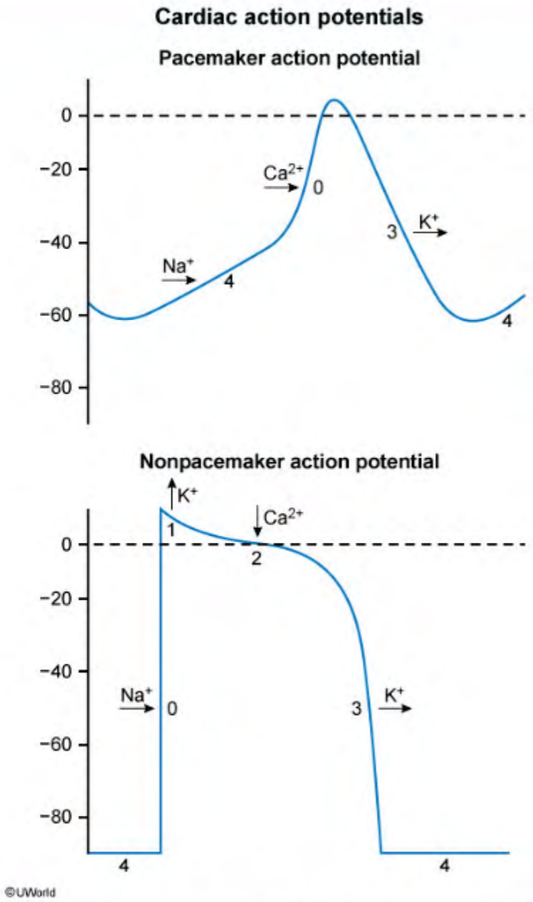

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A 46-year-old man with a 30-pack-year smoking history develops right leg pain and swelling after returning from an overseas trip. He is diagnosed with a lower extremity deep venous thrombosis and is started on intravenous heparin. Shortly after being hospitalized, he develops right-sided weakness and facial droop. CT scan of the head reveals a left middle cerebral artery stroke. Which of the following physical examination findings is most likely to be present in this patient?

- A. Diastolic decrescendo-type murmur that decreases following amyl nitrite inhalation
- B. Ejection-type systolic murmur that increases on standing
- C. Presystolic murmur that disappears with atrial fibrillation
- D. Midsystolic click that occurs earlier with inspiration
- E. Wide splitting of S2 that does not change with respiration

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- ✗ A. Diastolic decrescendo-type murmur that decreases following amyl nitrite inhalation (6%)
- B. Ejection-type systolic murmur that increases on standing (9%)
- C. Presystolic murmur that disappears with atrial fibrillation (15%)
- D. Midsystolic click that occurs earlier with inspiration (7%)
- ✓ E. Wide splitting of S2 that does not change with respiration (60%)

Incorrect
Correct answer
E

60%
Answered correctly

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Explanation

A cerebrovascular event (eg, transient ischemic attack, stroke) in the setting of known venous thromboembolic disease is suspicious for **paradoxical embolism**. Paradoxical emboli originate in the systemic venous circulation (lower or upper extremities) and enter the systemic arterial circulation via an intracardiac or intrapulmonary shunt. They can occur in patients with **patent foramen ovale, atrial septal defects**, ventricular septal defects, or large pulmonary arteriovenous malformations.

Atrial septal defects with left-to-right shunting typically cause **wide and fixed splitting** (no change with respiration) of the second heart sound (S2). Patients may also have a mid-systolic ejection murmur over the left upper sternal border resulting from increased flow across the pulmonic valve and a mid-diastolic rumble due to increased flow across the tricuspid valve. Importantly, a paradoxical embolism can occur even in patients with net left-to-right

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Explanation

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(Choice A) An early diastolic decrescendo murmur is characteristic of aortic regurgitation. Inhaled amyl nitrite produces marked vasodilatation, which reduces systemic arterial pressure and regurgitant flow (lessening the murmur).

(Choice B) A systolic ejection murmur that increases in intensity with standing can be heard in patients with hypertrophic cardiomyopathy (due to a decrease in left ventricular outflow tract size). In contrast, the murmur of valvular aortic stenosis decreases in intensity upon standing.

(Choice C) Mitral or tricuspid valve stenosis can cause a diastolic murmur with presystolic accentuation due to atrial contraction. With mild stenosis, the murmur may only be audible during the accentuation phase in late diastole; atrial fibrillation may cause the murmur to disappear completely.

(Choice D) The midsystolic click of mitral valve prolapse occurs earlier with inspiration due to the reduced left ventricular volume caused by decreased venous return to the left heart.

Educational objective:
Paradoxical embolism occurs when a thrombus from the venous system crosses into the arterial circulation via an

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disease is suspicious for **paradoxical embolism**. Paradoxical emboli originate in the systemic venous circulation (lower or upper extremities) and enter the systemic arterial circulation via an intracardiac or intrapulmonary shunt. They can occur in patients with **patent foramen ovale, atrial septal defects**, ventricular septal defects, or large pulmonary arteriovenous malformations.

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(Choice D) The midsystolic click of mitral valve prolapse occurs earlier with inspiration due to the reduced left ventricular volume caused by decreased venous return to the left heart.

Educational objective:
Paradoxical embolism occurs when a thrombus from the venous system crosses into the arterial circulation via an abnormal connection between the right and left cardiac chambers (eg, patent foramen ovale, atrial septal defect, or ventricular septal defect). Atrial left-to-right shunts cause wide and fixed splitting of S2 and can facilitate paradoxical embolism due to periods of transient shunt reversal (eg, during straining or coughing).

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Explanation

Exhibit Display

Variation in intensity of cardiac murmurs

Maneuver	Change in cardiac parameters	Murmurs that increase	Murmurs that decrease
Inspiration	↑ RV preload, ↓ LV preload	Most right-sided murmurs (↑ RV blood volume)	Most left-sided murmurs (↓ LV blood volume)
Valsalva strain phase	↓ RV & LV preload	MVP & HCM (↓ LV blood volume)	Most valvular murmurs (↓ flow through valve)
Abrupt standing			
Squatting	↑ RV & LV preload, ↑ LV afterload	Most valvular murmurs (↑ flow through valve)	MVP & HCM (↑ LV blood volume)
Passive leg raise	↑ RV & LV preload		
Hand grip	↑ LV afterload	AR, MR & VSD (↑ aortic & LV pressure)	MVP & HCM (↑ LV blood volume) AS (↓ transvalvular pressure gradient)

AR = aortic regurgitation; **AS** = aortic stenosis; **HCM** = hypertrophic cardiomyopathy; **LV** = left ventricular; **MR** = mitral regurgitation; **MVP** = mitral valve prolapse; **RV** = right ventricular; **VSD** = ventricular septal defect.

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Paradoxical embolism occurs when a thrombus from the venous system crosses into the arterial circulation via an

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A 28-year-old man comes to the emergency department with a 3-day history of increasing chest tightness and intermittent sharp chest pains. He also reports mild dyspnea, especially on exertion. The patient has no prior chronic medical conditions but had an upper respiratory illness a week ago that resolved without treatment. ECG reveals sinus tachycardia and low voltage QRS complexes that vary in the amplitude from beat to beat. Which of the following is the most likely chest x-ray finding in this patient?

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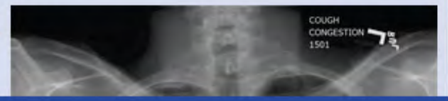

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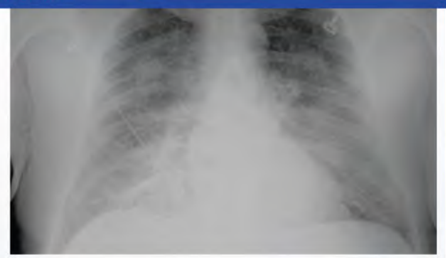

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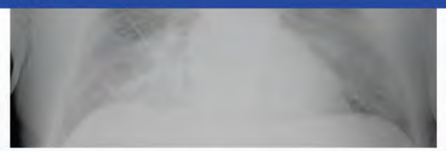

D.

(4%)

✕ E.

(4%)

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Correct answer
A

62%
Answered correctly

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Correct answer
A

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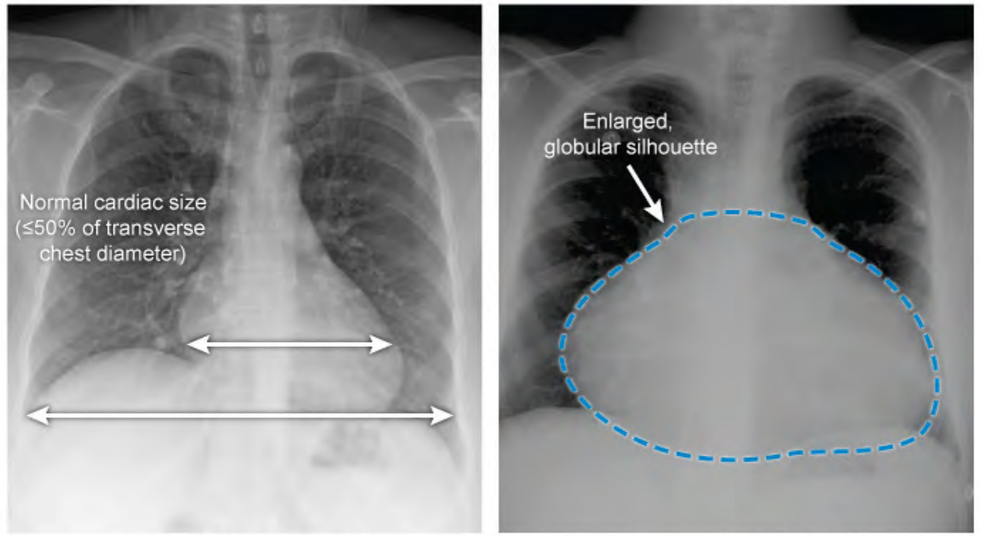
Pericardial effusion



Normal cardiac silhouette

Enlarged cardiac silhouette & clear lung fields

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As in this patient, viral pericarditis can be **complicated by pericardial effusion** due to fibrinous or serofibrinous pericardial inflammation. Symptoms of pericardial effusion may include a sensation of **chest tightness** or fullness

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This patient with a recent history of **upper respiratory infection** has likely developed **viral pericarditis** complicated by pericardial effusion. Viral infection is the most common cause of acute pericarditis, with coxsackievirus (types A and B) and echovirus being the most common organisms. Patients with pericarditis typically experience chest pain, which may be intermittent (as in this patient) but is often pleuritic and/or positional (eg, relieved by leaning forward).

As in this patient, viral pericarditis can be **complicated by pericardial effusion** due to fibrinous or serofibrinous pericardial inflammation. Symptoms of pericardial effusion may include a sensation of **chest tightness** or fullness and **dyspnea on exertion** (due to decreased diastolic ventricular filling that leads to decreased stroke volume). In addition, pericardial effusion often causes the following clinical findings:

- **Tachycardia** to compensate for decreased ventricular filling
- **Low voltage QRS** on ECG and diminished heart sounds to auscultation due to pericardial fluid accumulation increasing the distance between the heart and the ECG leads or stethoscope
- **Electrical alternans** on ECG due to the swinging motion of the heart in the fluid-filled pericardial cavity, causing a beat-to-beat variation in the QRS axis

Patients with pericardial effusion classically have an **enlarged, globular cardiac silhouette** on **chest x-ray**, which occurs when progressive pericardial stretching over days to weeks (ie, subacute course) allows large volumes (eg, up to 1-2 L) of pericardial fluid to accumulate. **Clear lung fields** are typically seen because the increase in pericardial pressure (due to pericardial fluid accumulation) affects the lower pressure, right-sided heart chambers more than the left-sided heart chambers. When right-sided obstruction to blood flow is greater than left-sided obstruction, pulmonary edema is unlikely to develop.

(Choice B) Hyperinflated lungs with increased bronchial markings are commonly seen in patients with chronic obstructive pulmonary disease.

(Choice C) Bilateral patchy infiltrates (perihilar > peripheral) are concerning for pulmonary edema. A normal cardiac silhouette may be present (as in this x-ray) due to an acute cardiac process (eg, MI) that causes acute systolic dysfunction and pulmonary congestion. In contrast, pulmonary edema in the setting of cardiomegaly is

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increasing the distance between the heart and the ECG leads or stethoscope

- **Electrical alternans** on ECG due to the swinging motion of the heart in the fluid-filled pericardial cavity, causing a beat-to-beat variation in the QRS axis

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(Choice D) Absence of right-sided, peripheral lung markings surrounding a small, collapsed lung is consistent with a large right-sided pneumothorax.

(Choice E) Blunting of the right costophrenic angle and loss of diaphragmatic contour are characteristic of a pleural effusion.

Educational objective:
Acute viral pericarditis is commonly complicated by pericardial effusion. Classic features of pericardial effusion include tachycardia and ECG findings of low voltage QRS and electrical alternans. Chest x-ray classically reveals an enlarged cardiac silhouette with clear lungs.

References

- Diagnosis and management of pericardial effusion.

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Normal cardiac silhouette

Enlarged cardiac silhouette & clear lung fields

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Pericardial effusion	
Etiology	• Acute pericarditis (eg, viral, bacterial, autoimmune) • Cardiac injury (eg, MI, cardiac surgery) • Malignancy (eg, metastatic spread of noncardiac tumors) • Renal failure with uremia
Clinical features	• Dyspnea, chest discomfort, fatigue • Possible cardiac tamponade* (eg, hypotension, JVD, ↓ heart sounds) • ECG: ↓ QRS voltage ± electrical alternans • CXR: enlarged cardiac silhouette** & clear lungs

*Acute or large effusions.
**With subacute & large effusions.
CXR = chest x-ray; **JVD** = jugular venous distension; **MI** = myocardial infarction.

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coxsackievirus (types A and B) and echovirus being the most common organisms. Patients with pericarditis typically experience chest pain, which may be intermittent (as in this patient) but is often pleuritic and/or positional (eg, re

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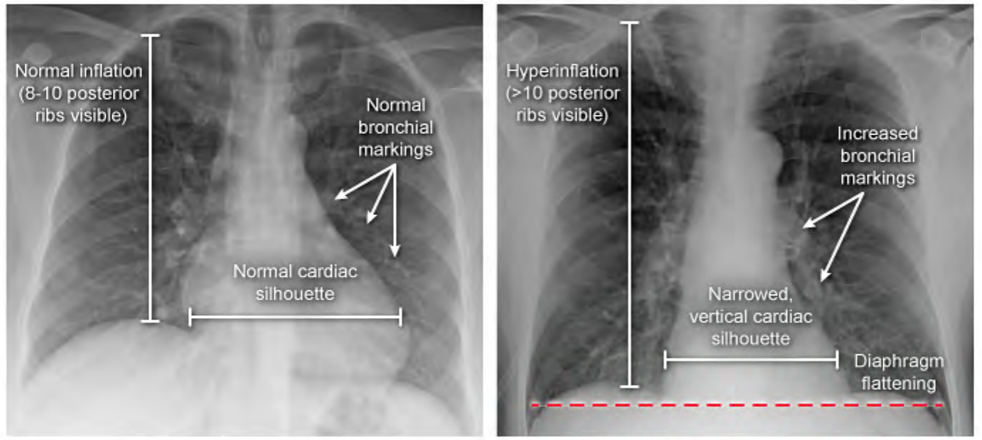
Chronic obstructive pulmonary disease



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Normal chest x-ray

Chronic obstructive pulmonary disease

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As in this patient, viral pericarditis can be **complicated by pericardial effusion** due to fibrinous or serofibrinous pericardial inflammation. Symptoms of pericardial effusion may include a sensation of **chest tightness** or fullness and dy
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Educational objective:

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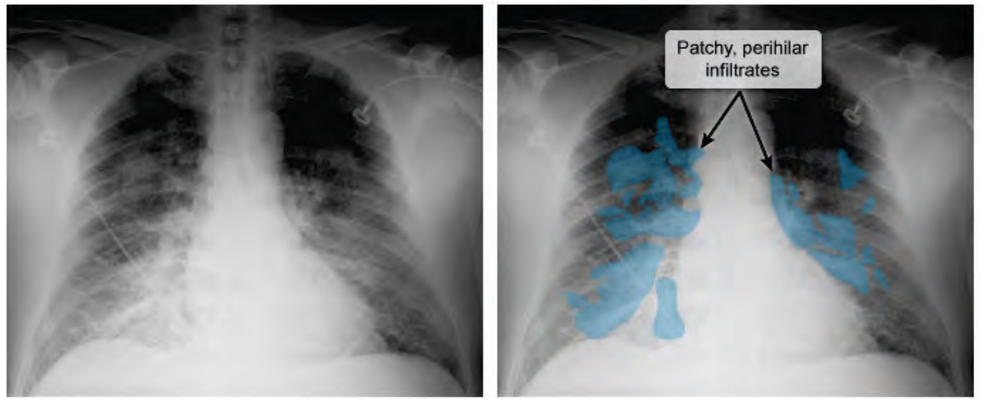
Bilateral pulmonary edema



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- **Tachycardia** to compensate for decreased ventricular filling
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include tachycardia and ECG findings of low voltage QRS and electrical alternans. Chest x-ray classically reveals an enlarged cardiac silhouette with clear lungs.

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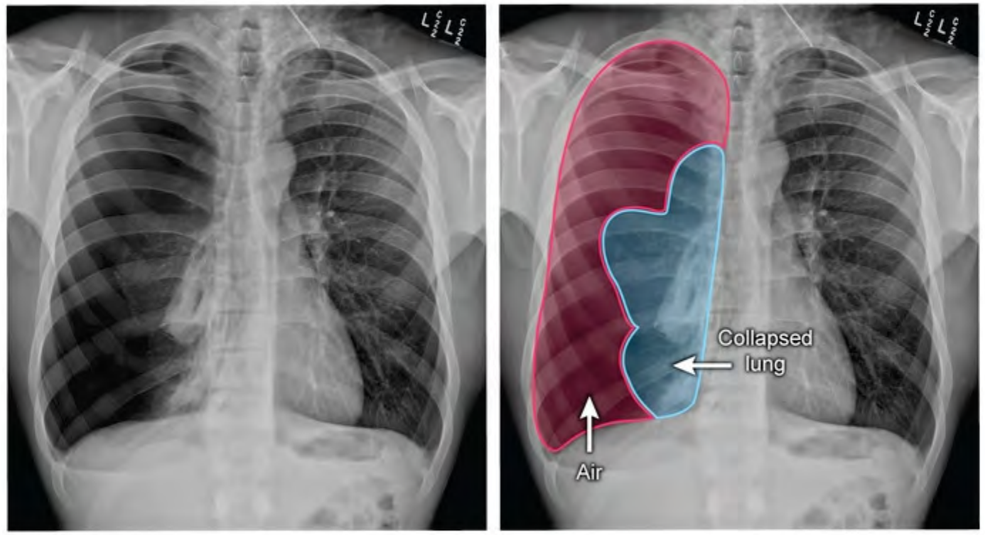
Large right pneumothorax



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addition, pericardial effusion often causes the following clinical findings:

- **Tachycardia** to compensate for decreased ventricular filling
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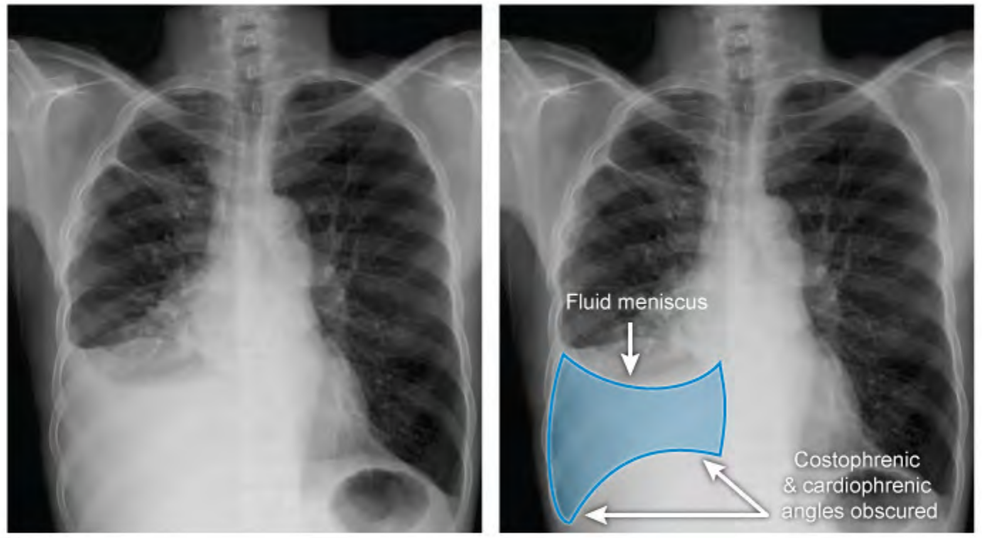
include tachycardia and ECG findings of low voltage QRS and electrical alternans. Chest x-ray classically reveals an enlarged cardiac silhouette with clear lungs.

Exhibit Display

Pleural effusion



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A 46-year-old man comes to the office with chest pain and dyspnea on exertion. He has no known medical problems and leads a sedentary lifestyle. He is a lifetime nonsmoker. Noninvasive cardiac testing is nondiagnostic. Left and right heart catheterization is planned. During the procedure, the catheter records periodic pressure changes with a maximum of 25 mm Hg and minimum of 2 mm Hg. The catheter is advanced further, and then shows periodic pressure changes with a maximum of 25 mm Hg and a minimum of 10 mm Hg. Assuming the results of the procedure are normal, the first set of readings was most likely obtained from which of the following locations?

- A. Left atrium
- B. Left ventricle
- C. Pulmonary artery
- D. Right atrium
- E. Right ventricle

Submit

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A 46-year-old man comes to the office with chest pain and dyspnea on exertion. He has no known medical problems and leads a sedentary lifestyle. He is a lifetime nonsmoker. Noninvasive cardiac testing is nondiagnostic. Left and right heart catheterization is planned. During the procedure, the catheter records periodic pressure changes with a maximum of 25 mm Hg and minimum of 2 mm Hg. The catheter is advanced further, and then shows periodic pressure changes with a maximum of 25 mm Hg and a minimum of 10 mm Hg. Assuming the results of the procedure are normal, the first set of readings was most likely obtained from which of the following locations?

- A. Left atrium (8%)
- ✗ B. Left ventricle (3%)
- C. Pulmonary artery (14%)
- D. Right atrium (34%)
- ✓ E. Right ventricle (38%)

Incorrect
Correct answer
E

38%
Answered correctly

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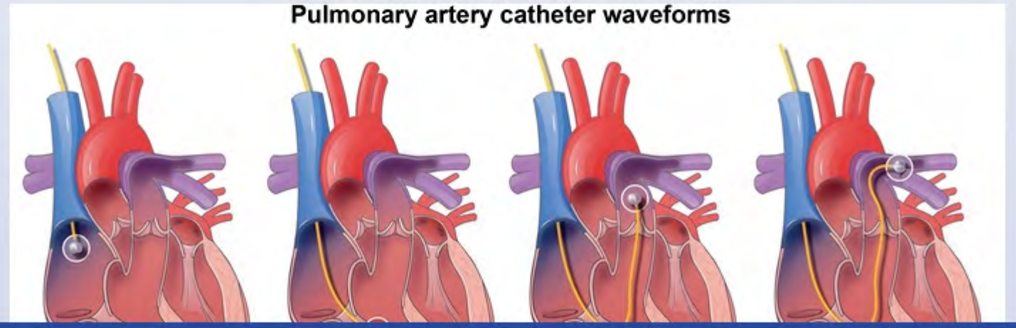
Explanation



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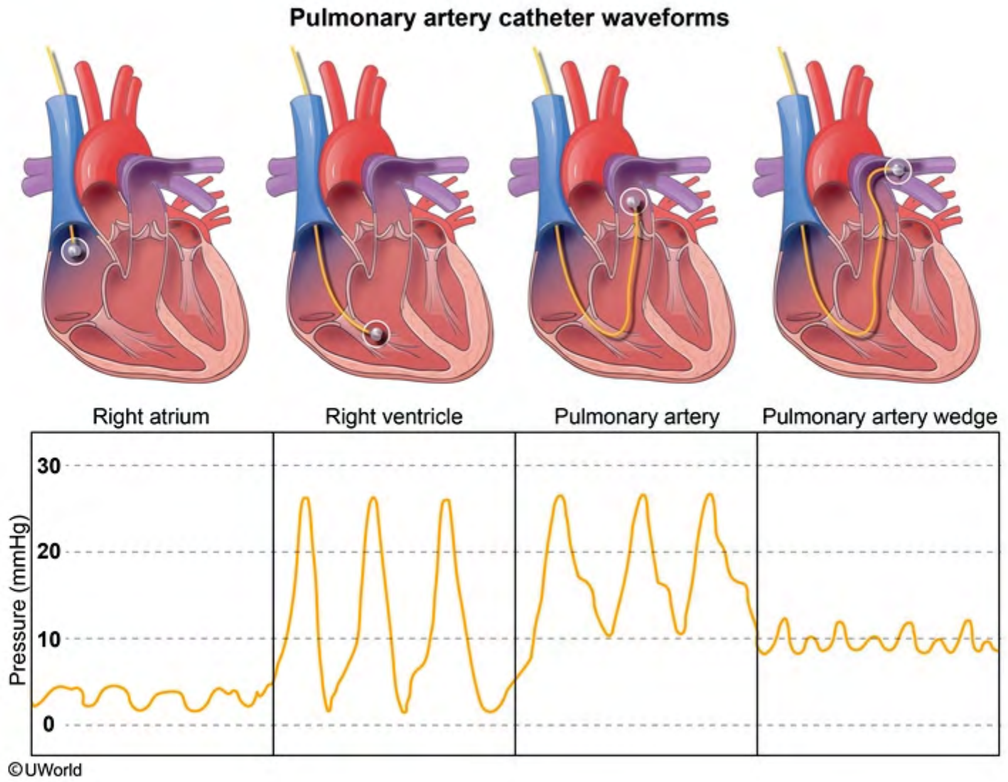

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Cardiac catheterization is performed to evaluate for cardiac etiologies and obtain direct measurements of intracardiac pressures in patients with unexplained symptoms. In general, right-sided pressures are much lower than those of the left side as the right ventricle is pumping blood into a much lower resistance pulmonary vasculature.

A balloon-tipped pulmonary artery (PA) catheter (attached distally to a pressure transducer) is inserted into the subclavian or internal jugular vein. When the PA catheter enters the superior vena cava (SVC), a venous waveform appears, and the balloon is inflated with air and the catheter is advanced. The location of the catheter tip is determined by the pressure readings as follows:

1. The venous pressure waveform of the SVC appears as small-amplitude oscillations. The pressure remains unchanged as the catheter is advanced into the right atrium (RA). Normal pressure in the SVC and RA is 1-6 mm Hg **(Choice D)**.
2. As the catheter is advanced across the tricuspid valve into the right ventricle (RV), a pulsatile waveform appears. The peak (systolic) pressure is the strength of right ventricular contraction, and the lowest (diastolic) pressure is just below the RA pressure. RV systolic pressure is normally 15-30 mm Hg.
3. As the catheter advances across the pulmonic valve into a main pulmonary artery, the pressure waveform shows a sudden increase in diastolic pressure with no change in systolic pressure. This rise in diastolic pressure is due to pulmonary capillary resistance and backward transmission of left atrial pressure. The PA diastolic pressure is normally 6-12 mm Hg **(Choice C)**.
4. When the catheter is advanced into the distal PA branches, a venous type waveform appears; this is called the PA occlusion pressure or pulmonary capillary wedge pressure (PCWP). This pressure is reflective of venous pressure in the left heart (left atrial pressure and left ventricular diastolic pressure) **(Choice A)**. PCWP is usually equal to PA diastolic pressure (6-12 mm Hg).

(Choice B) Left-sided pressures are much higher than right heart pressures in order to maintain systemic perfusion. The normal left ventricular systolic pressure is approximately 90-140 mm Hg with a left ventricular end-

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tip is determined by the pressure readings as follows:

1. The venous pressure waveform of the SVC appears as small-amplitude oscillations. The pressure remains unchanged as the catheter is advanced into the right atrium (RA). Normal pressure in the SVC and RA is 1-6 mm Hg **(Choice D)**.
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(Choice B) Left-sided pressures are much higher than right heart pressures in order to maintain systemic perfusion. The normal left ventricular systolic pressure is approximately 90-140 mm Hg with a left ventricular end-diastolic pressure of 6-12 mm Hg.

Educational objective:
Right-sided pressures in the heart are lower than left-sided pressures due to lower resistance in the pulmonary vasculature. Right ventricular diastolic pressure is similar to right atrial/central venous pressure (1-6 mm Hg), whereas pulmonary artery diastolic pressure is slightly higher (6-12 mm Hg) due to resistance to flow in the pulmonary circulation.

Physiology	Cardiovascular System	Cardiac catheterization
Subject	System	Topic

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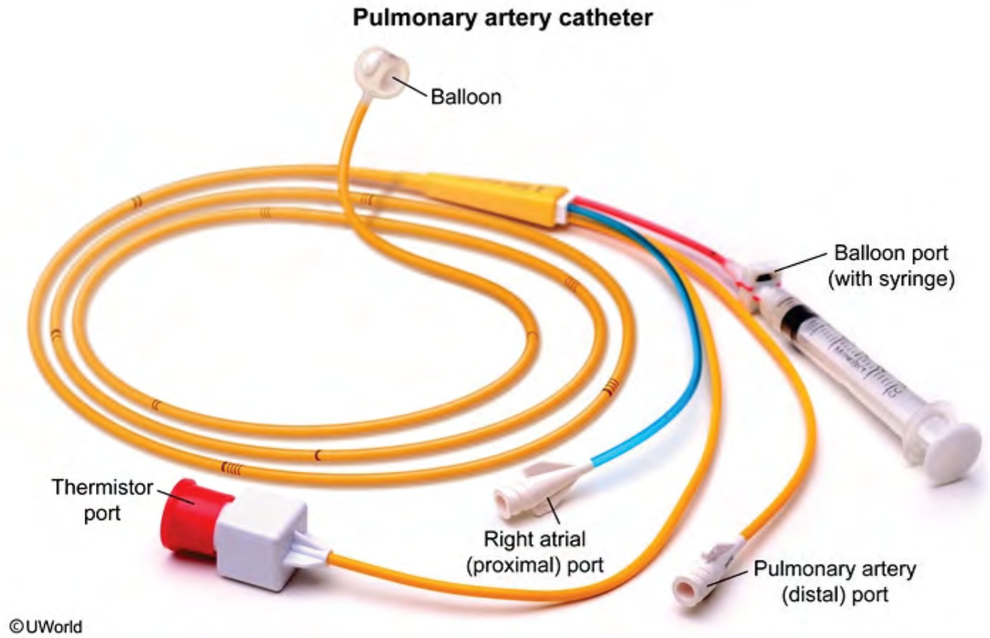

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(Choice B) Left-sided pressures are much higher than right heart pressures in order to maintain systemic perfusion. The normal left ventricular systolic pressure is approximately 90-140 mm Hg with a left ventricular end-

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Normal hemodynamic findings	
Location	**Pressure (mm Hg)**
Right atrium	Mean: 1-6
Right ventricle	Systolic: 15-30 Diastolic: 1-6
Pulmonary artery	Systolic: 15-30 Diastolic: 6-12
Left atrial/PCWP/LVEDP	Mean: 6-12
LVEDP = left ventricular end-diastolic pressure; **PCWP** = pulmonary capillary wedge pressure.	

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(Choice B) Left-sided pressures are much higher than right heart pressures in order to maintain systemic perfusion. The normal left ventricular systolic pressure is approximately 90-140 mm Hg with a left ventricular end-

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An 18-year-old man suddenly collapses during a high school soccer game. There was no preceding collision or trauma. Immediate resuscitation is initiated but he dies before the emergency medical service arrives. The patient had recently experienced occasional exertion-related chest discomfort and dyspnea. Otherwise, he had no significant medical history. He took no medications and did not use tobacco, alcohol, or illicit drugs. Which of the following is most likely to be seen on autopsy?

- A. Atherosclerotic narrowing of the left anterior descending artery
- B. Dilation of all cardiac chambers with thin ventricular walls
- C. Extracellular deposits and stiff ventricular walls
- D. Myocardial hypertrophy with patchy interstitial fibrosis
- E. Thrombus at the bifurcation of the main pulmonary artery

Submit

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An 18-year-old man suddenly collapses during a high school soccer game. There was no preceding collision or trauma. Immediate resuscitation is initiated but he dies before the emergency medical service arrives. The patient had recently experienced occasional exertion-related chest discomfort and dyspnea. Otherwise, he had no significant medical history. He took no medications and did not use tobacco, alcohol, or illicit drugs. Which of the following is most likely to be seen on autopsy?

- A. Atherosclerotic narrowing of the left anterior descending artery (1%)
- ✕ B. Dilation of all cardiac chambers with thin ventricular walls (12%)
- C. Extracellular deposits and stiff ventricular walls (4%)
- ✓ D. Myocardial hypertrophy with patchy interstitial fibrosis (79%)
- E. Thrombus at the bifurcation of the main pulmonary artery (1%)

Incorrect
Correct answer
D

79%
Answered correctly

03 secs
Time Spent

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Version

Explanation

Sudden cardiac death (SCD) is most often precipitated by cardiac **arrhythmia** (eg, ventricular tachycardia/fibrillation) that leads to markedly decreased cardiac output and impaired coronary artery perfusion. In older adults (eg, age ≥35), myocardial ischemia due to atherosclerotic coronary artery disease **(Choice A)** is the most common cause of SCD. However, young adults who experience SCD usually have underlying structural heart disease; **hypertrophic cardiomyopathy** (HCM) is one of the most common precipitating diseases. Most patients with HCM are asymptomatic, but some may experience exertional dyspnea, fatigue, chest pain, or syncope.

HCM is an autosomal dominant genetic disorder caused by mutation of one of several genes coding for cardiac sarcomere proteins. It is characterized by **cardiomyocyte hypertrophy** (predominantly affecting the **septum**) and

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Explanation

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HCM is an autosomal dominant genetic disorder caused by mutation of one of several genes coding for cardiac sarcomere proteins. It is characterized by **cardiomyocyte hypertrophy** (predominantly affecting the **septum**) and **myofiber disarray** consisting of an irregular arrangement of abnormally shaped cardiomyocytes and increased **interstitial fibrosis**. This structural disarray leads to conduction abnormalities that predispose to ventricular arrhythmia and SCD.

(Choice B) Dilated cardiac chambers and thin ventricular walls are consistent with dilated cardiomyopathy (DCM), a less common cause of SCD than HCM in young adults. A patient with DCM would likely have symptoms of heart failure (eg, lower extremity swelling, dyspnea, orthopnea) and would not be capable of participating in strenuous activities such as soccer.

(Choice C) Extracellular deposits and stiff ventricular walls suggest restrictive cardiomyopathy due to amyloidosis or hemochromatosis. Although restrictive cardiomyopathy can cause SCD, there is usually a preceding course of progressive and symptomatic heart failure.

(Choice E) Thrombus at the bifurcation of the main pulmonary artery (ie, saddle pulmonary embolism) is a potential cause of SCD; however, a preceding history of exertion-related dyspnea and chest discomfort would not be expected.

Educational objective:
Hypertrophic cardiomyopathy is a common cause of sudden cardiac death (SCD) in young adults. Histologic features include cardiomyocyte hypertrophy and myofiber disarray with increased interstitial fibrosis. The structural

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tachycardia/fibrillation) that leads to markedly decreased cardiac output and impaired coronary artery perfusion. In older adults (eg, age ≥35), myocardial ischemia due to atherosclerotic coronary artery disease **(Choice A)** is the most common cause of SCD. However, young adults who experience SCD usually have underlying structural heart disease; **hypertrophic cardiomyopathy** (HCM) is one of the most common precipitating diseases. Most patients with HCM are asymptomatic, but some may experience exertional dyspnea, fatigue, chest pain, or syncope.

HCM is an autosomal dominant genetic disorder caused by mutation of one of several genes coding for cardiac sarcomere proteins. It is characterized by **cardiomyocyte hypertrophy** (predominantly affecting the **septum**) and **myofiber disarray** consisting of an irregular arrangement of abnormally shaped cardiomyocytes and increased **interstitial fibrosis**. This structural disarray leads to conduction abnormalities that predispose to ventricular arrhythmia and SCD.

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Educational objective:
Hypertrophic cardiomyopathy is a common cause of sudden cardiac death (SCD) in young adults. Histologic features include cardiomyocyte hypertrophy and myofiber disarray with increased interstitial fibrosis. The structural disarray creates a substrate for ventricular arrhythmia (eg, ventricular tachycardia, ventricular fibrillation) that can lead to SCD.

References

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tachycardia/fibrillation) that leads to markedly decreased cardiac output and impaired coronary artery perfusion. In

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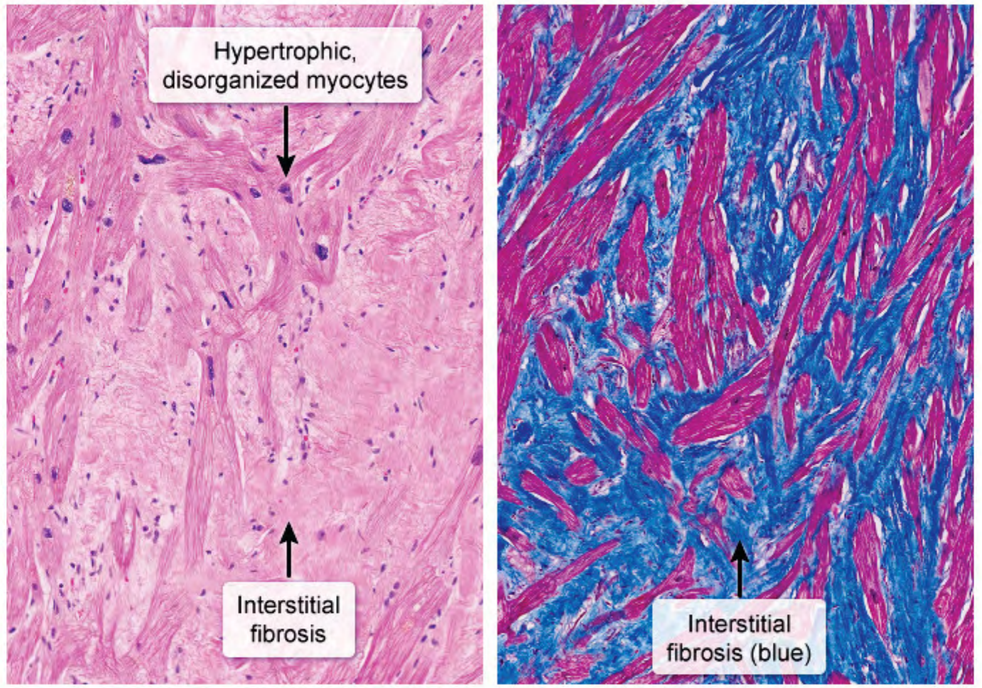
Exhibit Display

Hypertrophic cardiomyopathy



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Hematoxylin & eosin stain

Trichrome stain

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A 55-year-old man comes to the office for follow-up of hypertension and coronary heart disease. He has been on several medications in the past with suboptimal blood pressure control. The physician considers adding verapamil to the patient's current regimen, as its vasodilatory properties make it useful for controlling systemic hypertension. Verapamil also affects cardiac contractility but has a minimal effect on skeletal muscle. Which of the following properties of skeletal muscle is responsible for its resistance to the medication?

- A. Calmodulin-independent excitation-contraction coupling
- B. Decreased troponin affinity to intracellular calcium
- C. Elaborate T-tubular system
- D. Lack of internal automaticity
- E. Little dependence on extracellular calcium influx

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- A. Calmodulin-independent excitation-contraction coupling (17%)
- ✗ B. Decreased troponin affinity to intracellular calcium (3%)
- C. Elaborate T-tubular system (11%)
- D. Lack of internal automaticity (15%)
- ✓ E. Little dependence on extracellular calcium influx (52%)

Incorrect
Correct answer
E

52%
Answered correctly

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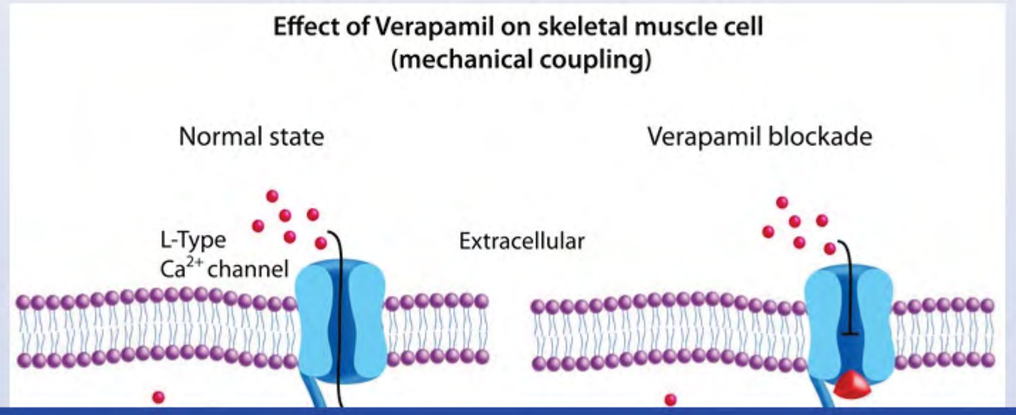
Explanation



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A 55-year-old man comes to the office for follow-up of hypertension and coronary heart disease. He has been on
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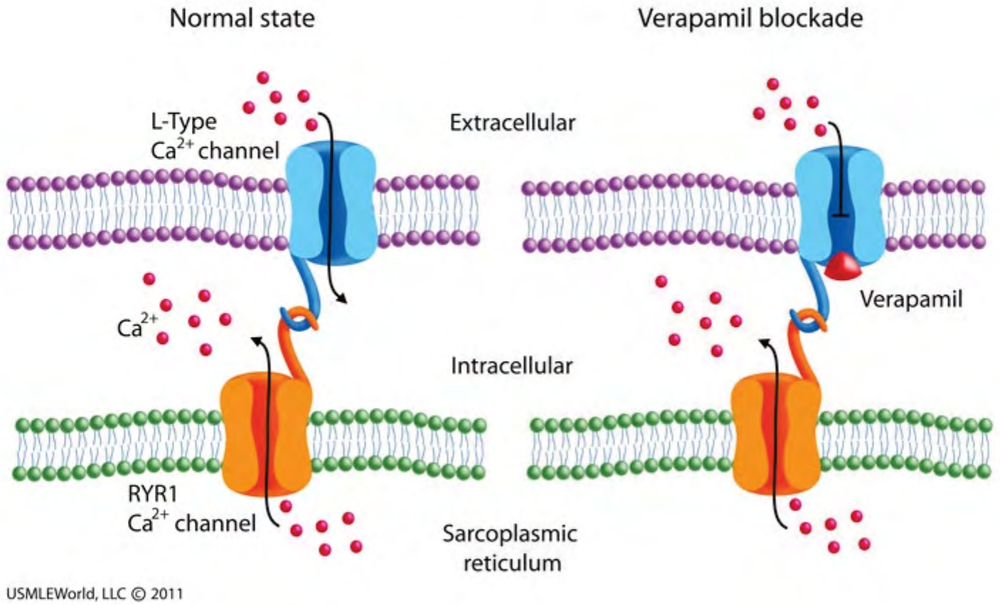
Exhibit Display

Effect of Verapamil on skeletal muscle cell (mechanical coupling)



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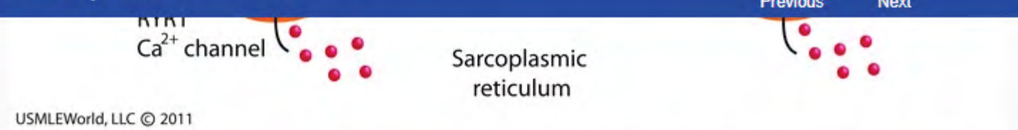

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Calcium plays an important role in excitation-contraction coupling in cardiac, smooth, and skeletal muscle cells. Myocyte depolarization causes activation of L-type calcium channels on the plasma membrane. In **cardiac and smooth muscle cells**, this leads to an **influx of extracellular calcium** that activates sarcoplasmic RyR channels, inducing the release of additional calcium from the sarcoplasmic reticulum (calcium-induced calcium release). The released calcium binds to troponin (cardiac muscle) or calmodulin (smooth muscle), allowing actin and myosin to interact and cause muscle contraction.

In contrast, the L-type calcium channels in **skeletal muscle** directly interact with RyR calcium channels to release calcium from the sarcoplasmic reticulum. This **mechanical coupling** between the receptors allows sarcoplasmic calcium release to occur without significant influx of calcium across the plasma membrane. **Verapamil** is a nondihydropyridine calcium channel blocker that acts by **blocking L-type calcium channels**, preventing the initial calcium influx into cardiac myocytes and vascular smooth muscle cells. Because skeletal muscle is not dependent on extracellular calcium influx, calcium channel blockers do not affect skeletal muscle contractility.

(Choice A) Troponin (but not calmodulin) is responsible for contraction of both cardiac and skeletal muscle. Unlike skeletal muscle, cardiac muscle is affected by verapamil due to its dependence on extracellular calcium to induce calcium release from the sarcoplasmic reticulum.

(Choice B) A rise in intracellular calcium concentration results in a structural change in the troponin complex that allows actin and myosin to interact and cause skeletal muscle contraction. Calcium channel blockers do not affect troponin's affinity to intracellular calcium.

(Choice C) An elaborate T tubule system is present in skeletal and cardiac muscle but not in smooth muscle. T tubules are a communication of the extracellular space that allows the depolarization impulse to reach deep into the cell, nearer to the myofilaments. They are in close proximity to the sarcoplasmic reticulum and therefore facilitate rapid release of intracellular calcium stores.

(Choice D) Unlike cardiac myocytes and specialized smooth muscle tissue (eg, gastrointestinal tract, ureters), skeletal muscle does not display automaticity and contracts only in response to the neurotransmitter acetylcholine.

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(Choice B) A rise in intracellular calcium concentration results in a structural change in the troponin complex that allows actin and myosin to interact and cause skeletal muscle contraction. Calcium channel blockers do not affect troponin's affinity to intracellular calcium.

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(Choice D) Unlike cardiac myocytes and specialized smooth muscle tissue (eg, gastrointestinal tract, ureters), skeletal muscle does not display automaticity and contracts only in response to the neurotransmitter acetylcholine. However, the mechanical coupling between L-type and RyR calcium channels is responsible for skeletal muscle's resistance to verapamil.

Educational objective:
Contraction initiation in cardiac and smooth muscle cells is dependent on extracellular calcium influx through L-type calcium channels, which can be prevented by calcium channel blockers (eg, verapamil). Skeletal muscle is resistant to calcium channel blockers, as calcium release by the sarcoplasmic reticulum is triggered by a mechanical interaction between L-type and RyR calcium channels.

References

- Role of ryanodine receptor subtypes in initiation and formation of calcium sparks in arterial smooth muscle:

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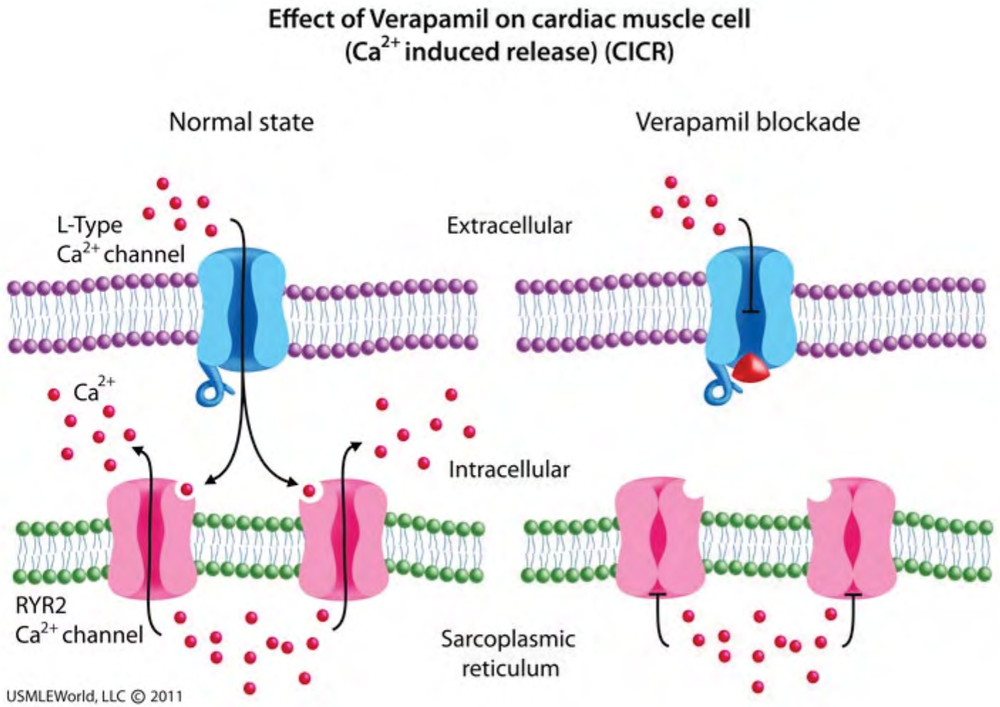
RYR1
Ca^{2+} channel

Sarcoplasmic

Exhibit Display

Effect of Verapamil on Cardiac Muscle Cell | Effect of Verapamil on Smooth Muscle Cell



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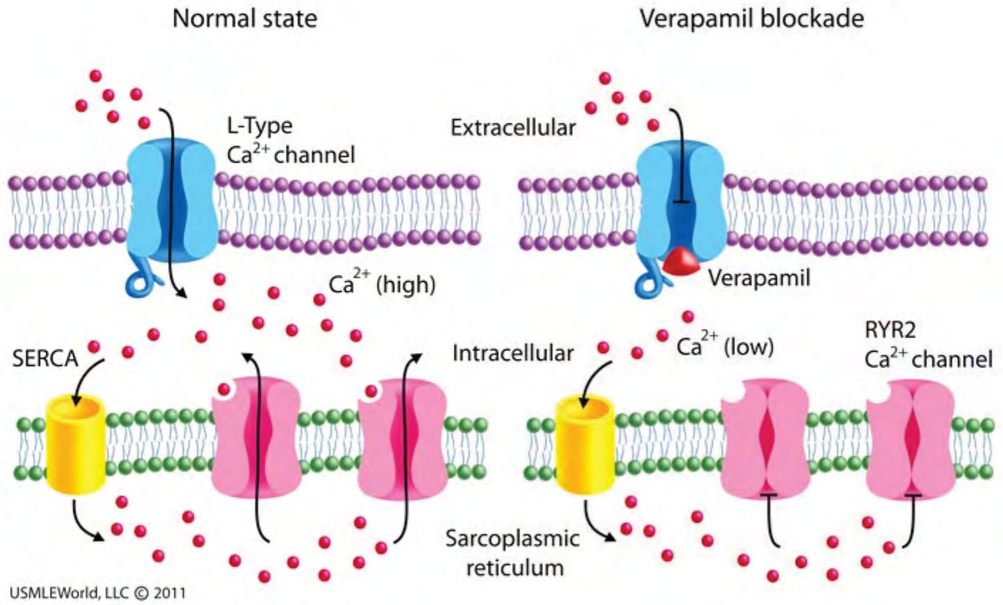
Ca^{2+} channel

Sarcoplasmic

Exhibit Display

Effect of Verapamil on Cardiac Muscle Cell | Effect of Verapamil on Smooth Muscle Cell

Effect of Verapamil on smooth muscle cell (through luminal Ca^{2+})



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(Choice D) Unlike cardiac myocytes and specialized smooth muscle tissue (eg, gastrointestinal tract, ureters), skeletal muscle does not display automaticity and contracts only in response to the neurotransmitter acetylcholine.

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A 54-year-old man comes to the emergency department due to worsening shortness of breath for the last 3 days. His symptoms initially occurred with exertion but are now present at rest. The patient could not sleep last night because of a suffocating cough each time he tried to lie down. He considers himself generally healthy and states, "I've never had to see a doctor for any problems." Family history is remarkable for asthma and hypertension. Blood pressure is 162/86 mm Hg, pulse is 92/min, and respirations are 26/min. An x-ray of the chest is shown in the exhibit. Which of the following is the most likely diagnosis?

- A. Acute decompensated heart failure
- B. Acute respiratory distress syndrome
- C. Idiopathic pulmonary fibrosis
- D. Obstructive pulmonary disease
- E. Pulmonary arterial hypertension
- F. Tension pneumothorax

Submit

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Exhibit Display

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- ✓ A. Acute decompensated heart failure (76%)
- B. Acute respiratory distress syndrome (8%)
- ✗ C. Idiopathic pulmonary fibrosis (3%)
- D. Obstructive pulmonary disease (2%)
- E. Pulmonary arterial hypertension (7%)
- F. Tension pneumothorax (1%)

Incorrect
Correct answer
A

76%
Answered correctly

32 secs
Time Spent

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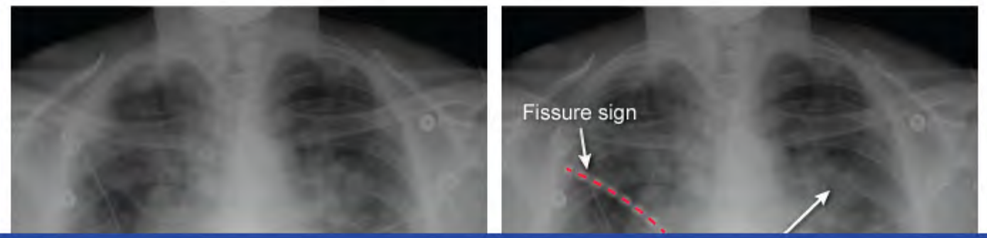
Explanation

Decompensated heart failure



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A. Acute decompensated heart failure (76%)

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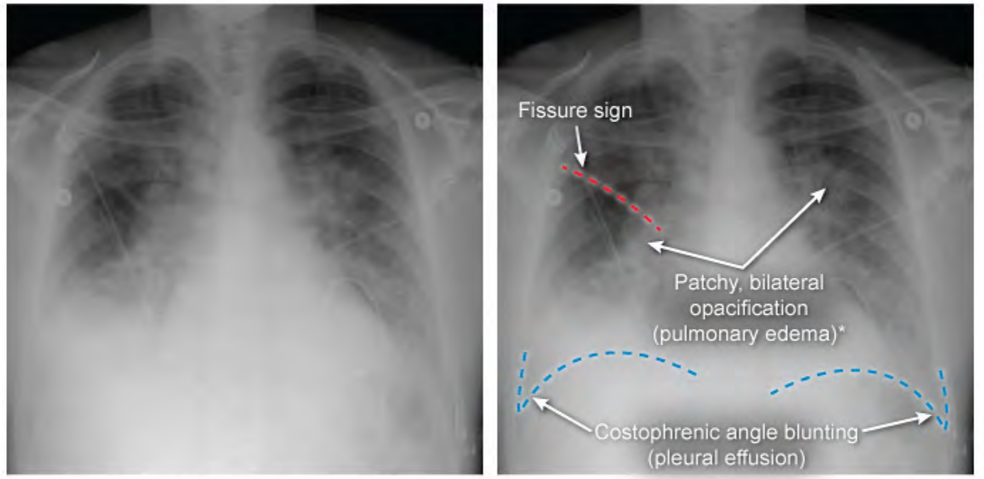
Decompensated heart failure



*Transudate in pulmonary interstitial & alveolar spaces

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(pleural effusion)
*Transudate in pulmonary interstitial & alveolar spaces
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*Transudate in pulmonary interstitial & alveolar spaces

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This patient has progressive dyspnea and orthopnea (cough when lying down), along with chest x-ray findings of prominent pulmonary vessels, patchy **bilateral airspace opacities**, blunting of the costophrenic angles (**pleural effusions**), and a **fissure sign** (created by fluid trapped between the right upper and middle lobe). These findings are consistent with **acute decompensated heart failure** (ADHF); chronic hypertension is a common cause of heart failure due to concentric left ventricular hypertrophy and resulting diastolic dysfunction.

In ADHF, increased atrial and ventricular filling pressures are transmitted to the pulmonary vasculature, causing fluid transudation into pulmonary interstitial and alveolar spaces (**cardiogenic pulmonary edema**). The chest x-ray may also show Kerley B lines—short, horizontal lines perpendicular to the pleural surface that represent edema of the interlobular septa. Cardiomegaly (cardiac-to-thoracic width ratio >50%) is also common in heart failure, but it is difficult to assess on anteroposterior view due to magnification of the heart.

(Choice B) Acute respiratory distress syndrome (ARDS), or noncardiogenic pulmonary edema, results from endothelial injury and pulmonary capillary fluid leakage; it is typically preceded by a precipitating event such as sepsis, pneumonia, or trauma. The appearance on chest x-ray is similar to ADHF, but pleural effusions are typically less prominent with ARDS (mostly alveolar opacification is seen), and ADHF is more likely given the absence of an inciting event in the patient's history.

(Choice C) The chest x-ray in idiopathic interstitial fibrosis shows diffuse, reticular opacities typically associated with decreased lung volumes. Most patients have slowly progressive dyspnea and a nonproductive cough.

(Choice D) Obstructive pulmonary diseases (eg, chronic obstructive pulmonary disease) are associated with hyperinflated lungs and a flattened diaphragm. Asthma is an exception as the chest x-ray is typically normal between exacerbations.

(Choice E) The chest x-ray in pulmonary arterial hypertension reveals enlargement of the pulmonary arteries and right ventricle and absence of pulmonary edema.

(Choice F) On chest x-ray, pneumothorax is demonstrated by a white, visceral pleural line with an absence of peripheral pulmonary markings. The mediastinum is often shifted away from the affected side in tension pneumothorax.

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heart failure due to concentric left ventricular hypertrophy and resulting diastolic dysfunction.

In ADHF, increased atrial and ventricular filling pressures are transmitted to the pulmonary vasculature, causing fluid transudation into pulmonary interstitial and alveolar spaces (**cardiogenic pulmonary edema**). The chest x-ray may also show Kerley B lines—short, horizontal lines perpendicular to the pleural surface that represent edema of the interlobular septa. Cardiomegaly (cardiac-to-thoracic width ratio >50%) is also common in heart failure, but it is difficult to assess on anteroposterior view due to magnification of the heart.

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(Choice E) The chest x-ray in pulmonary arterial hypertension reveals enlargement of the pulmonary arteries and right ventricle and absence of pulmonary edema.

(Choice F) On chest x-ray, pneumothorax is demonstrated by a white, visceral pleural line with an absence of peripheral pulmonary markings. The mediastinum is often shifted away from the affected side in tension pneumothorax.

Educational objective:
The chest x-ray in acute decompensated heart failure typically shows prominent pulmonary vessels; patchy, bilateral airspace opacification; and blunting of the costophrenic angles due to pleural effusions.

Pathology	Cardiovascular System	Acute heart failure
Subject	System	Topic

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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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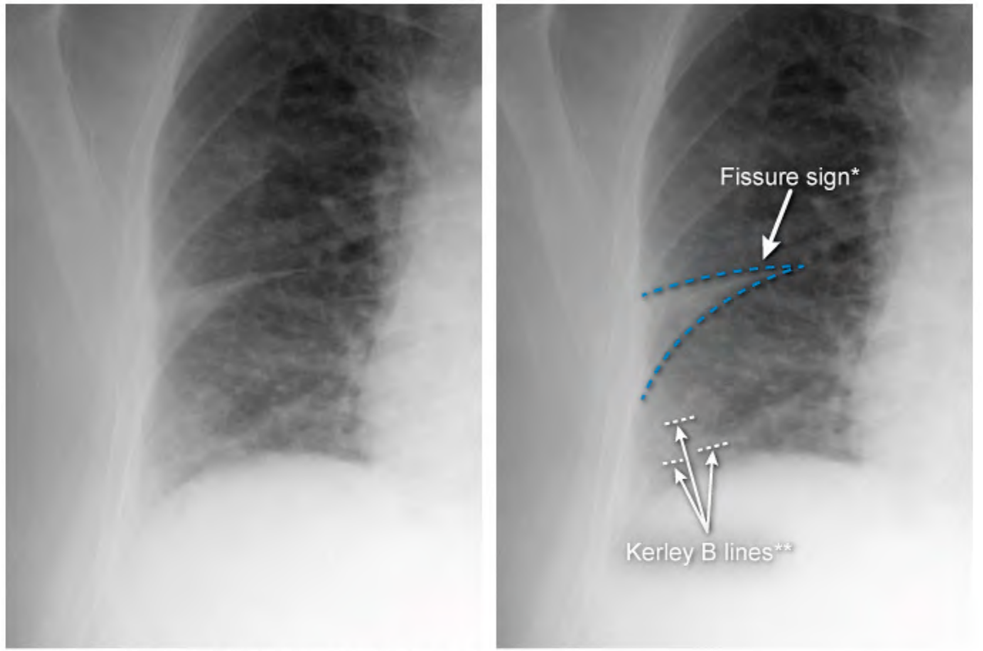
Congestive heart failure



*Intrapleural fluid between the right upper and middle lung lobes
**Thickening of the interlobular septa (connective tissue between secondary pulmonary lobules)

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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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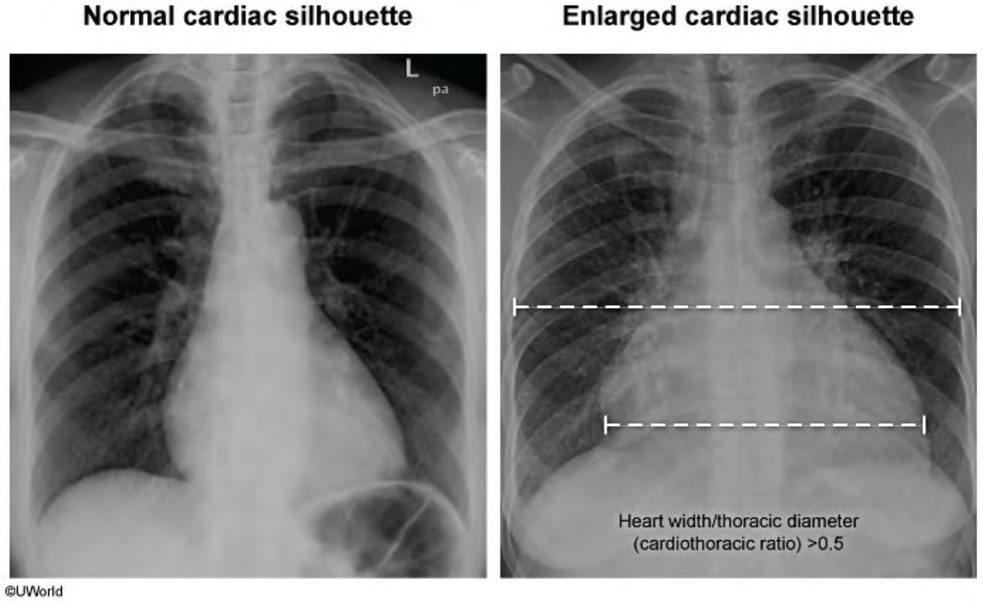

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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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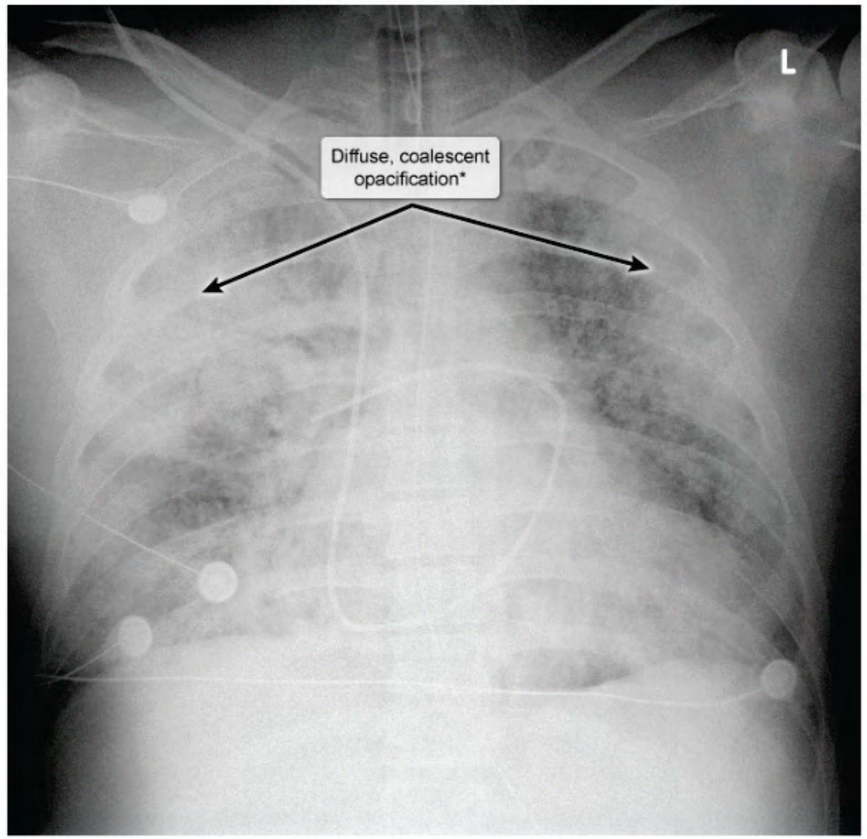
Acute respiratory distress syndrome



*Exudate due to diffuse alveolar endothelial damage (eg, sepsis, pneumonia, trauma)

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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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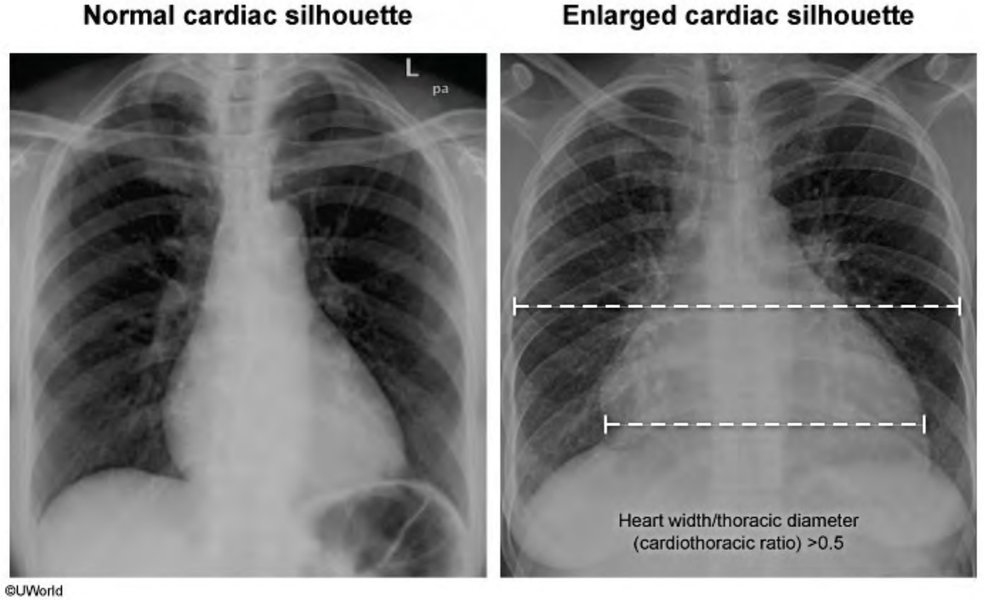

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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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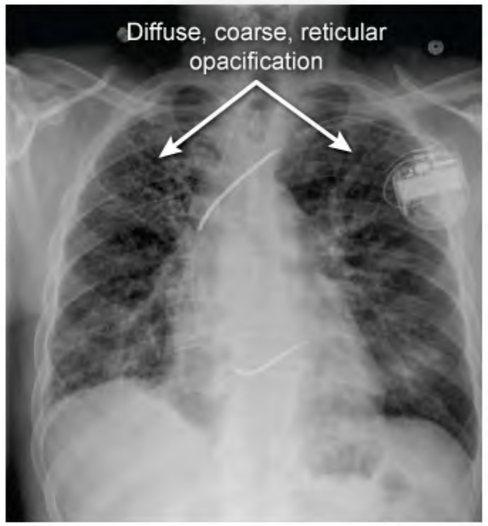
Interstitial lung disease



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(pleural effusion)

*Transudate in pulmonary interstitial & alveolar spaces

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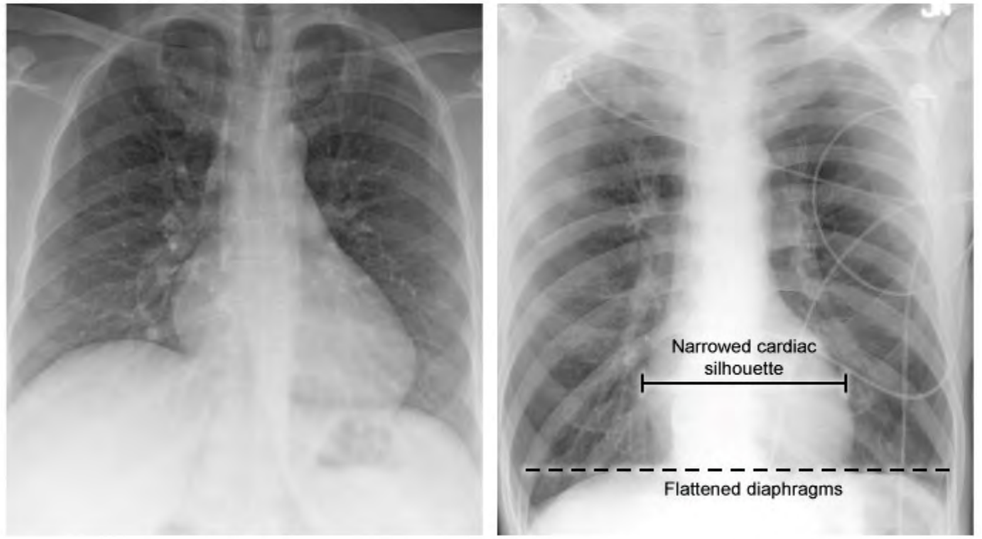
Chronic obstructive pulmonary disease (COPD)



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Normal chest x-ray

Lung hyperinflation in COPD

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prominent pulmonary vessels, patchy **bilateral airspace opacities**, blunting of the costophrenic angles (**pleural effusions**), and a **fissure sign** (created by fluid trapped between the right upper and middle lobe). These findings are co
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The chest x-ray in acute decompensated heart failure typically shows prominent pulmonary vessels; patchy, bilateral airspace opacification; and blunting of the costophrenic angles due to pleural effusions.

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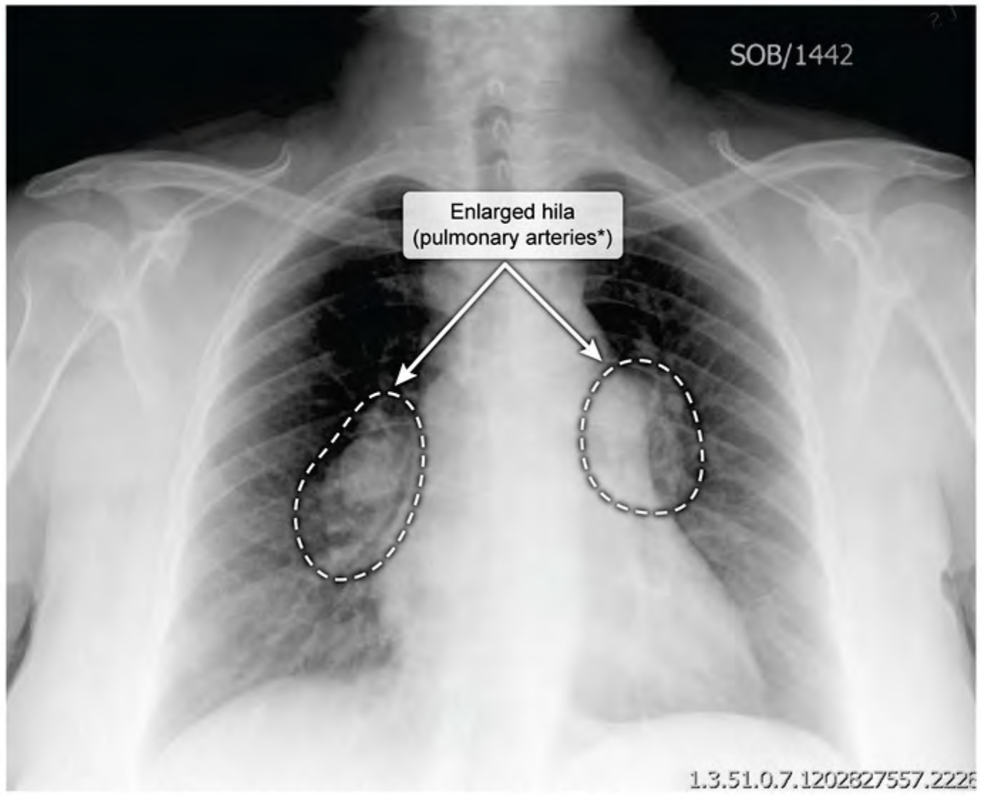
Pulmonary arterial hypertension



* Muscular hypertrophy in pulmonary arteries/arterioles elevates right-sided heart pressures

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heart failure due to concentric left ventricular hypertrophy and resulting diastolic dysfunction.

In ADHF, increased atrial and ventricular filling pressures are transmitted to the pulmonary vasculature, causing fluid tr
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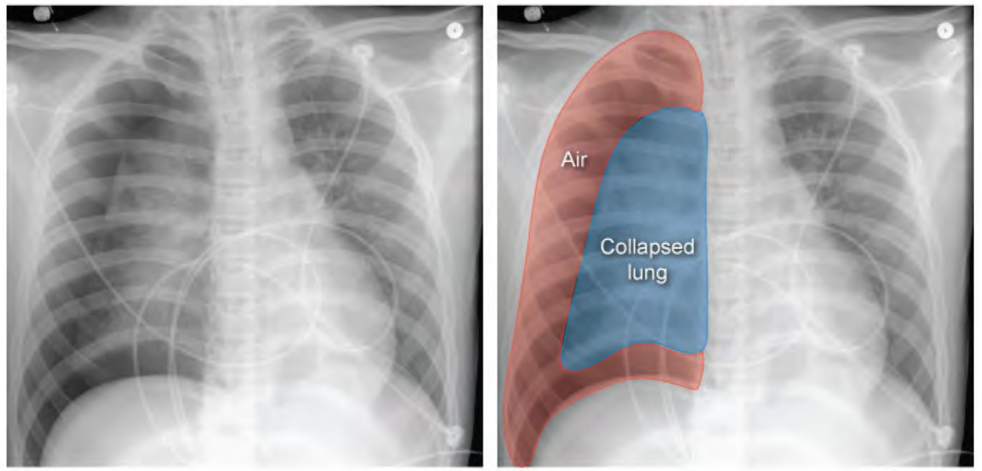
Pneumothorax



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Pathology	Cardiovascular System	Acute heart failure
Subject	System	Topic

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A 71-year-old man comes to the emergency department due to sudden-onset vision loss in his left eye for one hour. He has a history of coronary artery disease, hypertension, and type 2 diabetes mellitus. Blood pressure is 145/80 mm Hg and pulse is 72/min. On examination, the patient can see only hand motions through the left eye. Funduscopic evaluation of the eye shows a cherry-red spot in the macula with surrounding retinal whitening. Cranial nerve examination is otherwise unremarkable. There is a left-sided neck bruit on cardiovascular examination. Which of the following is the most likely path of the embolus causing this patient's symptoms?

- A. External carotid artery, facial artery, ophthalmic artery, retinal artery
- B. External carotid artery, ophthalmic artery, retinal artery
- C. External carotid artery, temporal artery, retinal artery
- D. Internal carotid artery, anterior cerebral artery, retinal artery
- E. Internal carotid artery, ophthalmic artery, retinal artery

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A 71-year-old man comes to the emergency department due to sudden-onset vision loss in his left eye for one hour. He has a history of coronary artery disease, hypertension, and type 2 diabetes mellitus. Blood pressure is 145/80 mm Hg and pulse is 72/min. On examination, the patient can see only hand motions through the left eye. Funduscopic evaluation of the eye shows a cherry-red spot in the macula with surrounding retinal whitening. Cranial nerve examination is otherwise unremarkable. There is a left-sided neck bruit on cardiovascular examination. Which of the following is the most likely path of the embolus causing this patient's symptoms?

- A. External carotid artery, facial artery, ophthalmic artery, retinal artery (7%)
- ✗ B. External carotid artery, ophthalmic artery, retinal artery (19%)
- C. External carotid artery, temporal artery, retinal artery (6%)
- D. Internal carotid artery, anterior cerebral artery, retinal artery (8%)
- ✓ E. Internal carotid artery, ophthalmic artery, retinal artery (57%)

Incorrect
Correct answer
E

57%
Answered correctly

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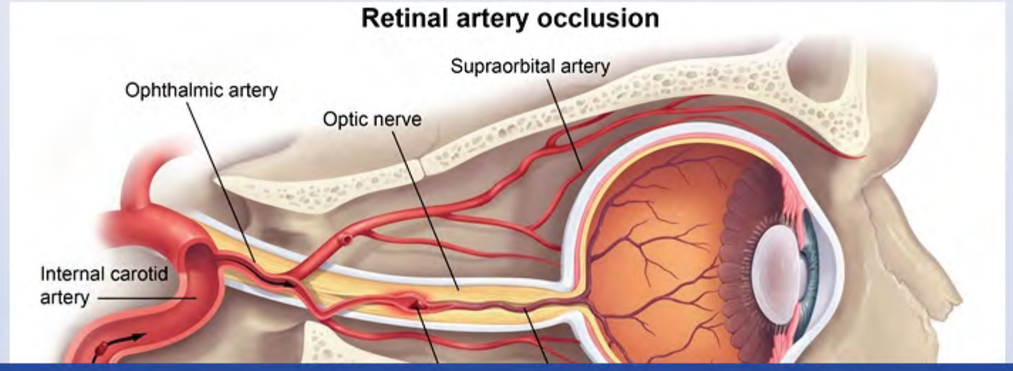
Explanation



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A. External carotid artery, facial artery, ophthalmic artery, retinal artery (7%)

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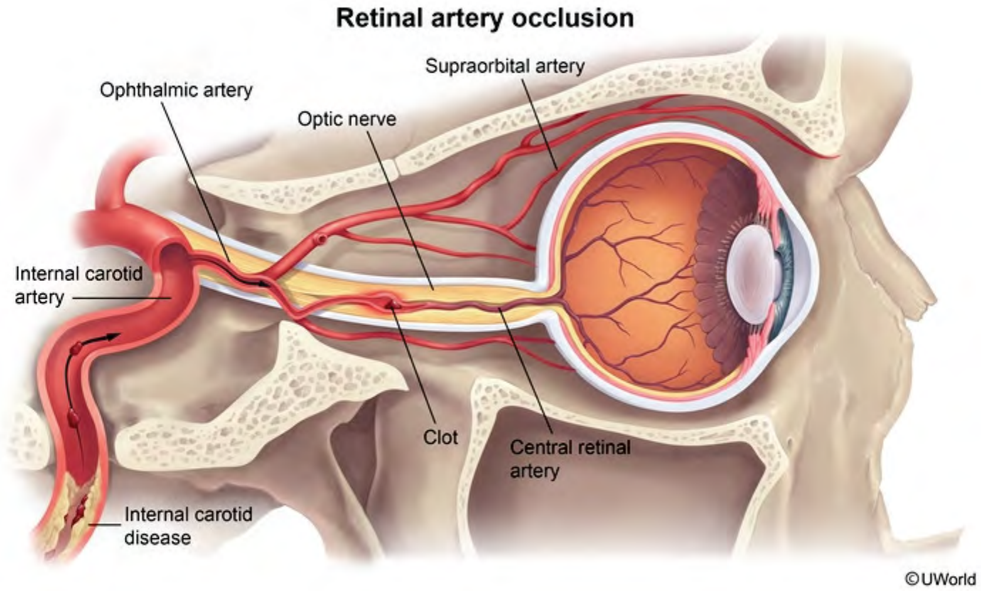

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Retinal artery occlusion (RAO) is an important cause of acute, painless, monocular vision loss. Thromboembolic

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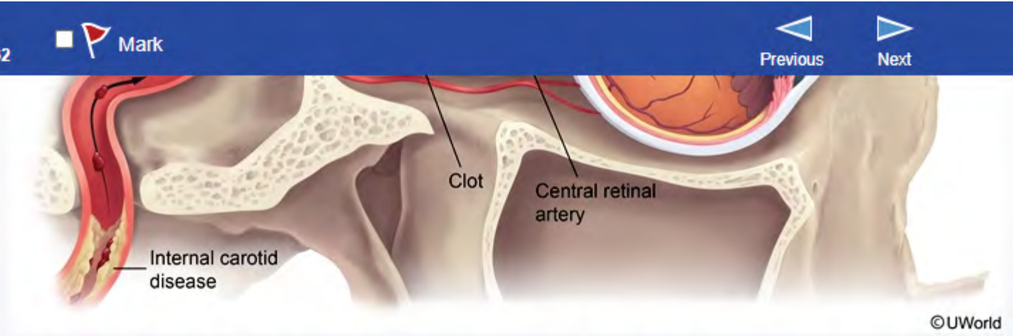

Retinal artery occlusion (RAO) is an important cause of acute, painless, monocular vision loss. Thromboembolic complications of atherosclerosis in the **internal carotid** are the most common cause of RAO. The retinal artery is one of the first branches of the **ophthalmic artery**, which receives its supply from the internal carotid **(Choice B)**. The retinal artery then travels within the retinal nerve to supply the inner retina and the surface of the optic nerve.

(Choices A and C) There are a number of anastomoses between branches of the ophthalmic artery and branches of the external carotid, including the facial artery and temporal artery, but neither branch anastomoses with the retinal artery or supplies the retina.

(Choice D) The anterior cerebral artery is a branch off the circle of Willis that supplies blood to the midline portions of the frontal and superior medial parietal lobes. Its occlusion can lead to loss of strength and sensation in the contralateral lower extremity, incontinence, and transcortical motor aphasia.

Educational objective:
Retinal artery occlusion is a cause of acute, painless, monocular vision loss. It is usually caused by thromboembolic complications of atherosclerosis traveling from the internal carotid artery and through the ophthalmic artery.

References

- Orbital vascular anatomy.
- Ocular ischemic syndrome - a systematic review.

Anatomy	Cardiovascular System	Retinal artery occlusion
Subject	System	Topic

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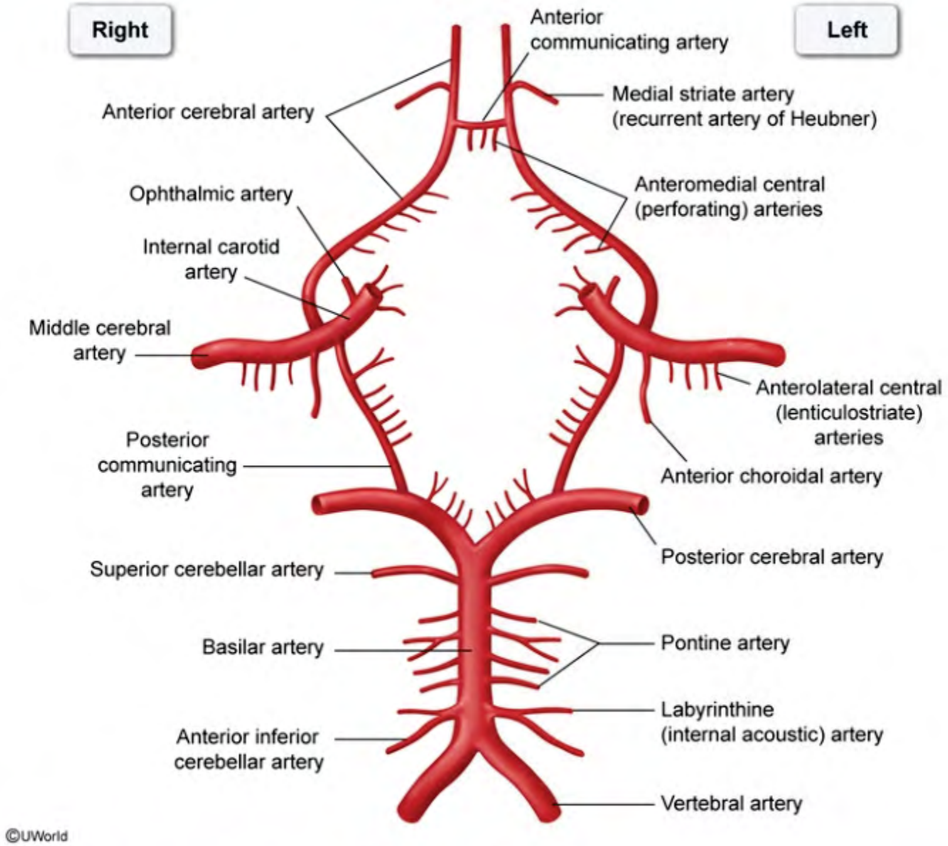
Circle of Willis



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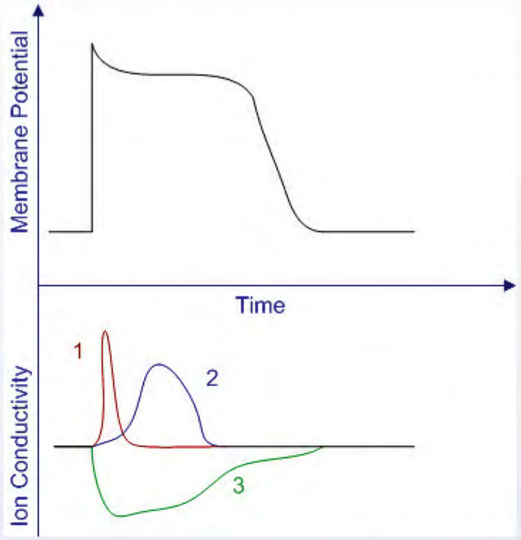
Membrane potential changes in an isolated cardiac muscle cell are recorded along with ion movements across the cell membrane (see graph below).



Which of the following ion sequences corresponds to regions 1-2-3 on the graph, respectively?

- A. Potassium, sodium, calcium
- B. Sodium, calcium, potassium
- C. Calcium, sodium, chloride
- D. Calcium, sodium, potassium
- E. Sodium, potassium, calcium

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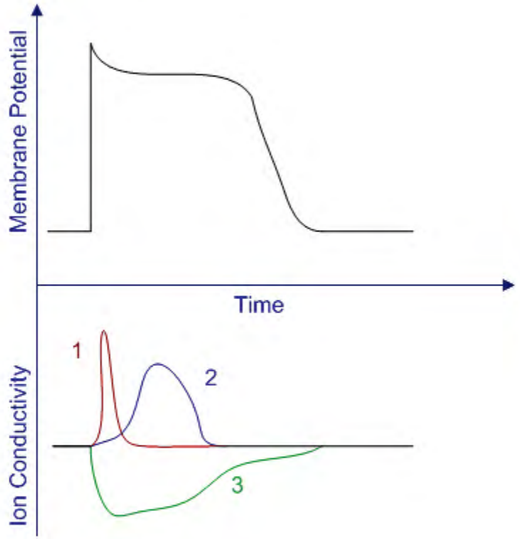

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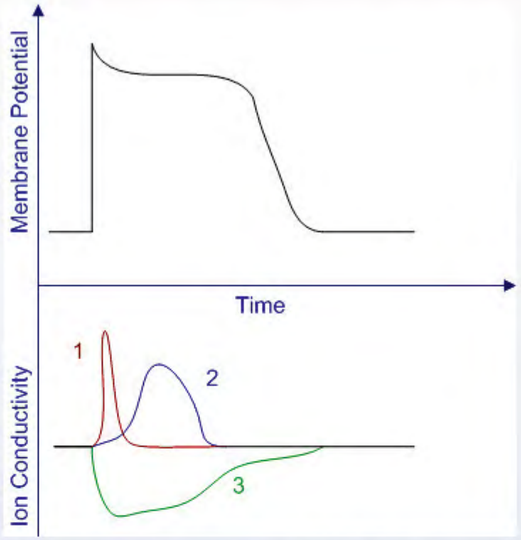
Membrane potential changes in an isolated cardiac muscle cell are recorded along with ion movements across the cell membrane (see graph below).



Which of the following ion sequences corresponds to regions 1-2-3 on the graph, respectively?

- A. Potassium, sodium, calcium (1%)
- ✓ B. Sodium, calcium, potassium (87%)
- C. Calcium, sodium, chloride (0%)
- D. Calcium, sodium, potassium (3%)
- E. Sodium, potassium, calcium (7%)

Correct

87%
Answered correctly

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Explanation

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E. Sodium, potassium, calcium (7 %)

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   channels. Efflux of K^+ from the cell restores the membrane resting potential.

In sum, the cardiac muscle action potential includes the following three key events: Net ion influx (corresponding

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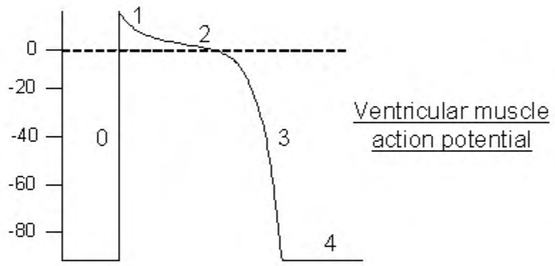

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The action potential of ventricular and non-nodal conduction cells within the heart consists of four phases.

1. **Phase 4: Resting potential (diastole).** Resting potential is determined largely by membrane permeability to K^+ ions when in the resting state. The resting potential of cardiac myocytes is approximately -90mV, while the resting potential of skeletal myocytes is approximately -75mV. The highly negative resting potential of cardiac myocytes reduces the risk of arrhythmias, as a larger stimulus is needed to excite the cells.
2. **Phase 0: Rapid depolarization.** As in skeletal myocytes, the action potential onset occurs when voltage-gated Na^+ channels open and Na^+ ions rush into the cell.
3. **Phase 1: Initial rapid repolarization.** This phase is associated with the rapid closure of Na^+ channels.
4. **Phase 2: Plateau.** This phase is a distinctive feature of the cardiac action potential not seen in other tissues. It is characterized by the opening of L-type dihydropyridine-sensitive Ca^{++} channels and the closure of some K^+ channels. Consequently, the membrane becomes highly permeable to Ca^{++} ions and minimally permeable to K^+ ions.
5. **Phase 3: Late rapid repolarization.** This phase features closure of Ca^{++} channels and the opening of K^+ channels. Efflux of K^+ from the cell restores the membrane resting potential.

In sum, the cardiac myocyte action potential includes the following three key events: Na^+ ion influx (corresponding to the red curve on the ion conductivity graph); Ca^{++} ion influx (corresponding to the blue curve on the ion conductivity graph); and decreased K^+ permeability during the action potential upstroke (corresponding to the green curve on the ion conductivity graph).

(Choices A, C, D, and E) The other ion sequences listed do not correlate with the action potential phases observed in cardiac myocytes.

Educational Objective:
The cardiac myocyte action potential consists of rapid depolarization (phase 0), initial rapid repolarization (phase 1), plateau (phase 2), late rapid repolarization (phase 3), and resting potential (phase 4). The action potential is associated with increased membrane permeability to Na^+ and Ca^{++} and decreased permeability to K^+.

Physiology	Cardiovascular System	Resting membrane potential and action potential
Subject	System	Topic

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A 64-year-old man loses consciousness near the entrance to an emergency room. A physician rushes to the patient and palpates a strong pulse along the inner side of the left sternocleidomastoid muscle. The vessel palpated by the doctor is a derivative of which of the following aortic arches?

- A. First
- B. Second
- C. Third
- D. Fourth
- E. Sixth

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A 64-year-old man loses consciousness near the entrance to an emergency room. A physician rushes to the patient and palpates a strong pulse along the inner side of the left sternocleidomastoid muscle. The vessel palpated by the doctor is a derivative of which of the following aortic arches?

- ✗ A. First (7%)
- B. Second (23%)
- ✓ C. Third (51%)
- D. Fourth (12%)
- E. Sixth (5%)

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Correct answer
C

51%
Answered correctly

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Explanation

Arches & associated cranial nerves		
Pharyngeal and aortic arch	**Cranial nerve**	**Key aortic arch derivatives**
1	Trigeminal (CN V)	Maxillary artery (portion)
2	Facial (CN VII)	Stapedial artery (regresses)
3	Glossopharyngeal (CN IX)	Common carotid artery, proximal internal

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Arches & associated cranial nerves		
Pharyngeal and aortic arch	**Cranial nerve**	**Key aortic arch derivatives**
1	Trigeminal (CN V)	Maxillary artery (portion)
2	Facial (CN VII)	Stapedial artery (regresses)
3	Glossopharyngeal (CN IX)	Common carotid artery, proximal internal carotid artery
4	Superior laryngeal branch of vagus (CN X)	True aortic arch, subclavian arteries
5	Obliterated	Obliterated
6	Recurrent laryngeal branch of CN X	Pulmonary arteries, ductus arteriosus

During embryonic development, the head, neck, and upper thorax region develop from a set of arches known as **pharyngeal arches**. Each arch is associated with a cranial nerve and an aortic arch derivative. The vessel palpated in this patient is the **common carotid artery**, which (along with the proximal portion of the internal carotid artery) is derived from the **third aortic arch** (ie, third pharyngeal arch). The third pharyngeal arch also gives rise to the glossopharyngeal nerve (CN IX), parts of the hyoid bone, and the stylopharyngeus muscle.

(Choice A) The first pharyngeal arch is associated with the trigeminal nerve (CN V). This pharyngeal arch gives rise to the tensor tympani, muscles of mastication, maxilla, and inner ear bones (eg, incus, malleus). The

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During embryonic development, the head, neck, and upper thorax region develop from a set of arches known as **pharyngeal arches**. Each arch is associated with a cranial nerve and an aortic arch derivative. The vessel palpated in this patient is the **common carotid artery**, which (along with the proximal portion of the internal carotid artery) is derived from the **third aortic arch** (ie, third pharyngeal arch). The third pharyngeal arch also gives rise to the glossopharyngeal nerve (CN IX), parts of the hyoid bone, and the stylopharyngeus muscle.

(Choice A) The first pharyngeal arch is associated with the trigeminal nerve (CN V). This pharyngeal arch gives rise to the tensor tympani, muscles of mastication, maxilla, and inner ear bones (eg, incus, malleus). The associated first aortic arch regresses almost completely, although it does give rise to a portion of the maxillary artery.

(Choice B) The second pharyngeal arch is associated with the facial nerve (CN VII). This pharyngeal arch gives rise to the muscles of facial expression as well as some ear and hyoid structures. The stapedial artery is a derivative of the associated second aortic arch, but this vessel typically regresses.

(Choice D) The fourth pharyngeal arch is associated with the superior laryngeal branch of the vagus nerve (CN X). This pharyngeal arch gives rise to many of the muscles of the pharynx and soft palate as well as a few of the laryngeal muscles. The associated fourth aortic arch gives rise to part of the true aortic arch and subclavian arteries.

(Choice E) The sixth pharyngeal arch is associated with the recurrent laryngeal branches of CN X. This pharyngeal arch gives rise to most of the muscles of the larynx, and the associated aortic arch gives rise to the pulmonary arteries and the ductus arteriosus.

Educational objective:
The common carotid artery and the proximal portion of the internal carotid artery are derived from the third aortic arch. The third aortic arch is associated with the third pharyngeal arch, which gives rise to the glossopharyngeal nerve (CN IX), parts of the hyoid bone, and the stylopharyngeus muscle.

Embryology	Cardiovascular System	Embryologic derivatives
Subject	System	Topic

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	3	Glossopharyngeal (CN IX)	artery, proximal internal

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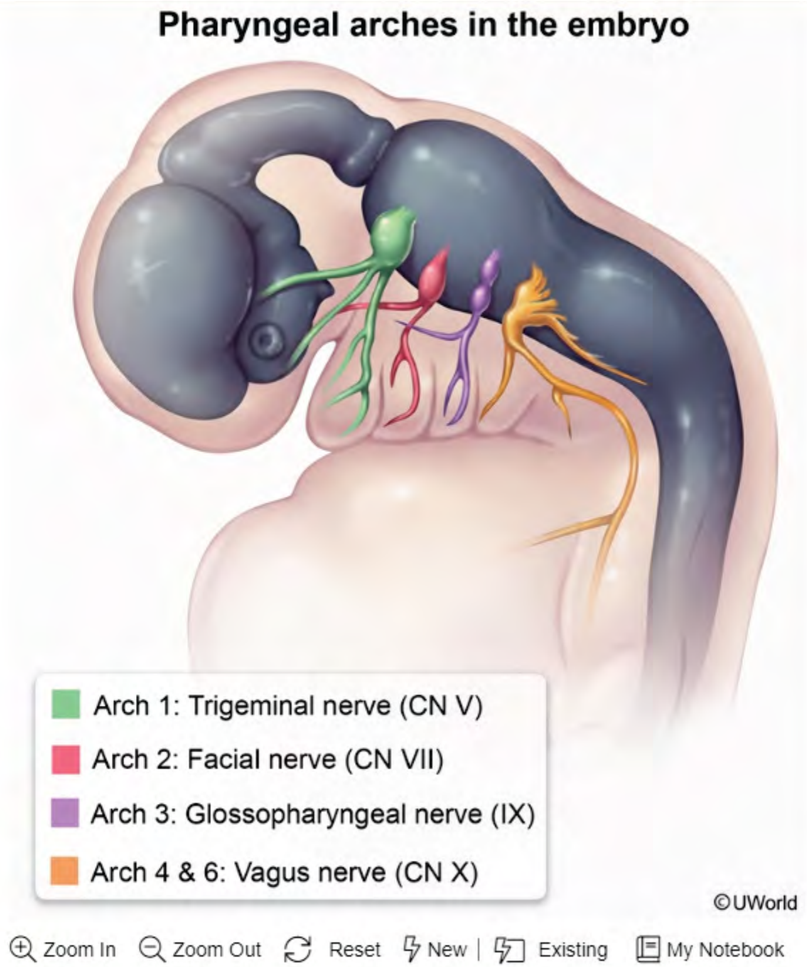

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(Choice E) The sixth pharyngeal arch is associated with the recurrent laryngeal branches of CN X. This

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A 16-year-old previously healthy boy is brought to the office due to palpitations. He was watching a documentary at a movie theater when his heart suddenly began to race and he became lightheaded. The patient says that he has felt "rapid heartbeats" in the past and thought that they were due to stress from school examinations, but he has never had associated lightheadedness before. He has no chest pain or dyspnea. Physical examination is within normal limits. Resting ECG is shown in the exhibit. Which of the following is most likely present in this patient?

- A. Accessory bypass tract connecting the atria and ventricles
- B. Cardiac conduction system degeneration below the level of the atrioventricular node
- C. Ectopic atrial foci that serve as the dominant pacemaker
- D. Impaired generation of impulses in the sinoatrial node
- E. Prolonged ventricular action potential due to myocyte ion channel abnormality

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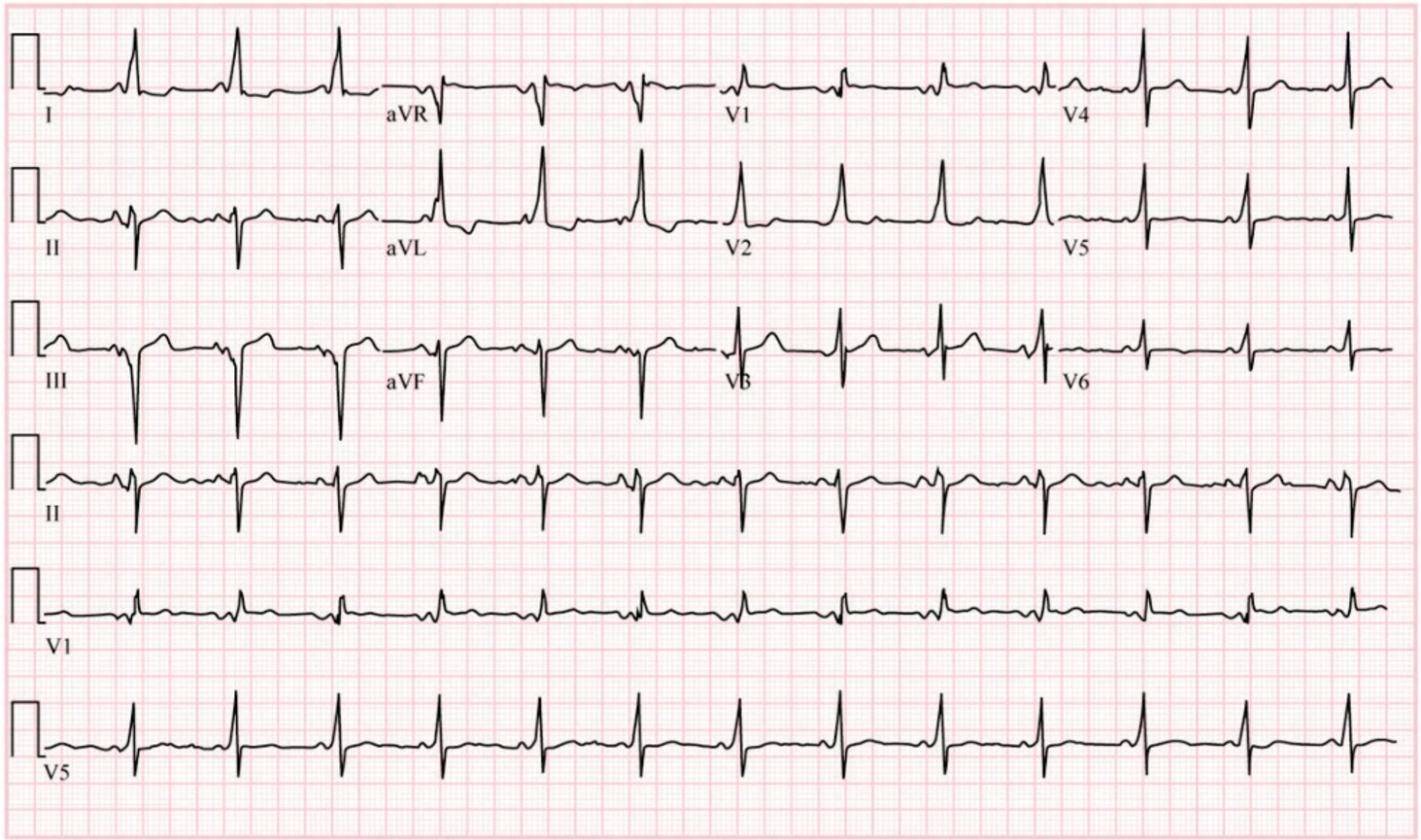

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- ✓ A. Accessory bypass tract connecting the atria and ventricles (61%)
- B. Cardiac conduction system degeneration below the level of the atrioventricular node (8%)
- C. Ectopic atrial foci that serve as the dominant pacemaker (13%)
- D. Impaired generation of impulses in the sinoatrial node (7%)
- E. Prolonged ventricular action potential due to myocyte ion channel abnormality (9%)

Correct

61% Answered correctly

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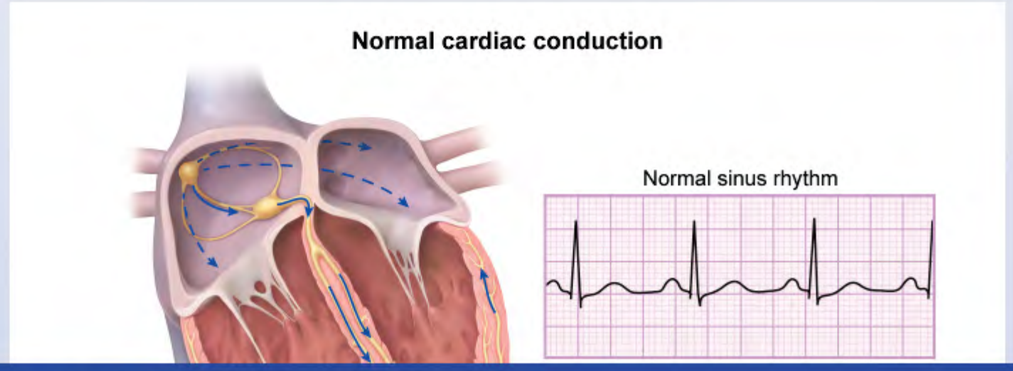
Explanation



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C. Ectopic atrial foci that serve as the dominant pacemaker (13%)

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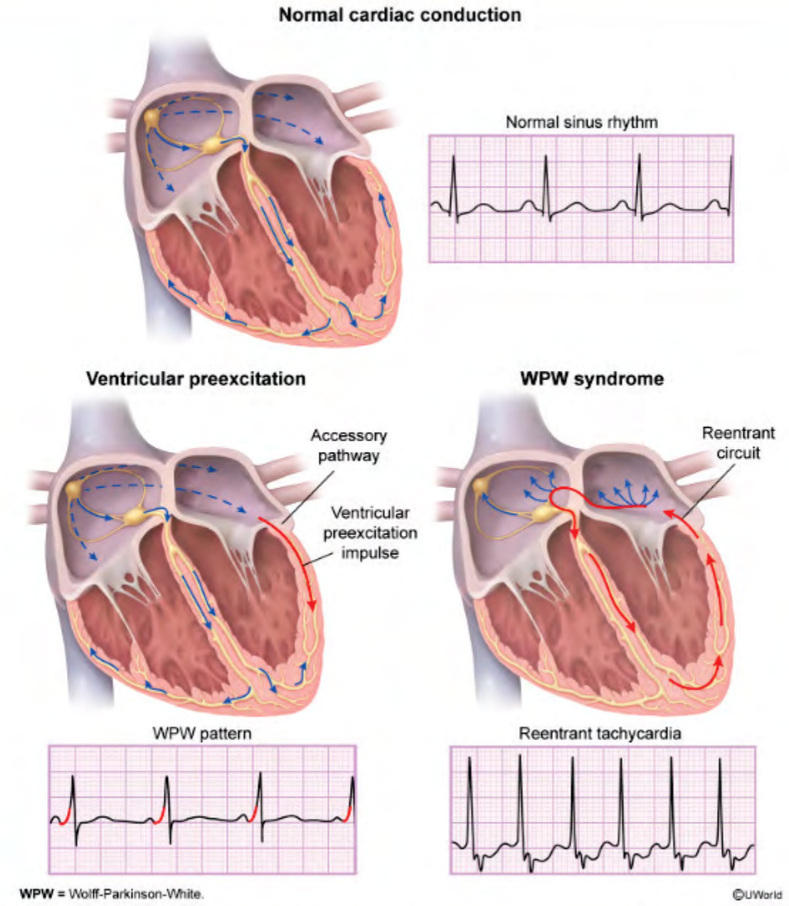

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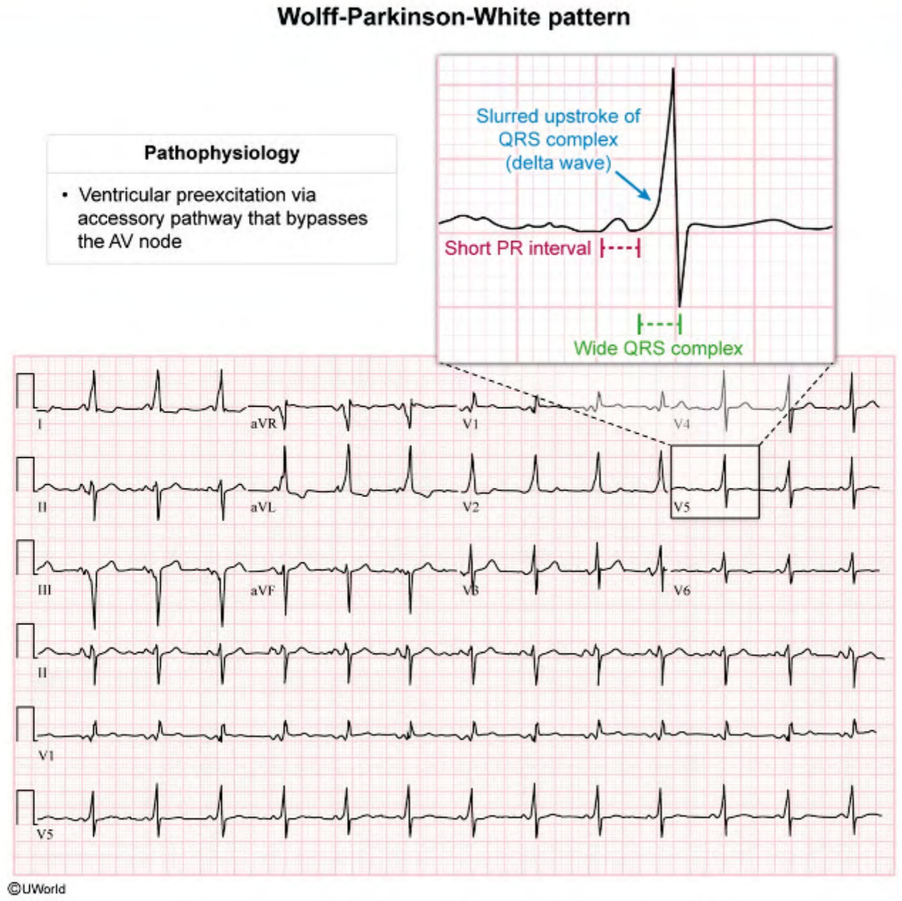

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(Choice D) Impaired generation of impulses in the sinoatrial node can occur with sick sinus syndrome, which usually affects elderly patients. It appears on ECG as a delayed P wave or the absence of a P wave with the QRS

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WPW = Wolff-Parkinson-White.

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This patient's resting ECG shows the characteristic **Wolff-Parkinson-White (WPW)** pattern, which consists of the following triad:

- **Short PR** interval, generally <120 msec
- Slurred and broad upstroke of the QRS complex (**delta wave**)
- **QRS widening**, often with ST segment/T wave changes

These findings are created by an **accessory pathway** (bundle of Kent), which allows ventricular depolarization to begin before the normal depolarization impulse can pass through the atrioventricular node (ie, **ventricular preexcitation**). Many patients with WPW pattern on ECG remain asymptomatic, but some develop paroxysmal tachyarrhythmias (ie, episodes of palpitations and lightheadedness); these patients are considered to have WPW syndrome.

Atrioventricular reentrant tachycardia (AVRT), a form of paroxysmal supraventricular tachycardia, is the most common arrhythmia seen with WPW syndrome. It results when a premature atrial or ventricular contraction creates a reentrant circuit involving the accessory pathway and the atrioventricular node. Notably, the characteristic findings of WPW pattern are not evident on ECG during AVRT and are best demonstrated during normal sinus rhythm.

(Choice B) Cardiac conduction system degeneration below the level of the atrioventricular node (eg, His Purkinje system) leads to variable degrees of conduction block and typically presents with symptomatic bradycardia.

(Choice C) Patients with wandering atrial pacemaker or ectopic atrial rhythm have ectopic atrial foci that replace the sinus node as the dominant pacemaker; P waves of varying morphology are seen on ECG. This patient has normal, uniform P waves suggesting normal sinus node function.

(Choice D) Impaired generation of impulses in the sinoatrial node can occur with sick sinus syndrome, which usually affects elderly patients. It appears on ECG as a delayed P wave or the absence of a P wave with the QRS complex formed by a junctional escape beat.

(Choice E) Congenital long QT syndrome is caused by mutations in the genes encoding cardiac sodium or potassium channels. The prolonged repolarization interval (eg, QTc > 450 msec) puts patients at risk for

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WPW = Wolff-Parkinson-White.

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(Choice E) Congenital long QT syndrome is caused by mutations in the genes encoding cardiac sodium or potassium channels. The prolonged repolarization interval (eg, QTc > 450 msec) puts patients at risk for

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Atrioventricular reentrant tachycardia (AVRT)

Pathophysiology	Key features
• Accessory pathway connecting atria & ventricles creates a macrorentrant circuit • Usually triggered by a PAC or PVC • Characteristic arrhythmia of WPW syndrome	• Rate 140-250 bpm • Regular R-R intervals • Retrograde P waves often visible • Usually narrow QRS complexes

PAC = premature atrial contraction; **PVC** = premature ventricular contraction; **WPW** = Wolff-Parkinson-White.

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(Choice E) Congenital long QT syndrome is caused by mutations in the genes encoding cardiac sodium or potassium channels. The prolonged repolarization interval (eg, QTc > 450 msec) puts patients at risk for developing polymorphic ventricular tachycardia (eg, torsades de pointes).

Educational objective:
Wolff-Parkinson-White (WPW) pattern is characterized by a shortened PR interval, slurred initial upstroke of the QRS complex (delta wave), and widening of the QRS complex. It is caused by an accessory pathway that bypasses the atrioventricular node, leading to ventricular preexcitation. Some patients develop paroxysmal tachyarrhythmias (eg, atrioventricular reentrant tachycardia) due to reentry of electrical impulses through the accessory conduction pathway (ie, WPW syndrome).

References

- Wolff Parkinson White syndrome.

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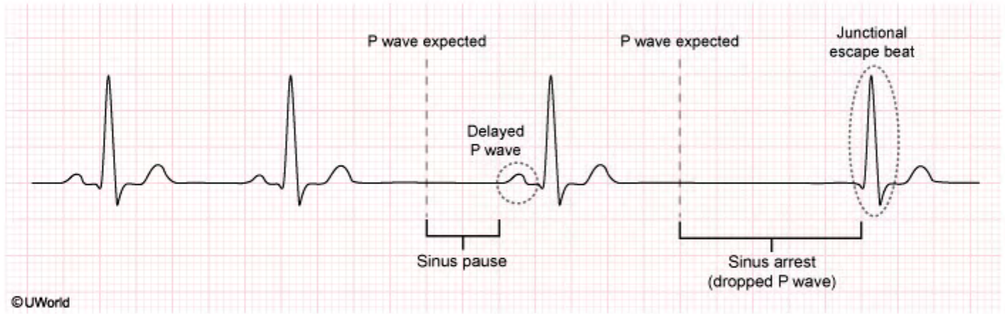

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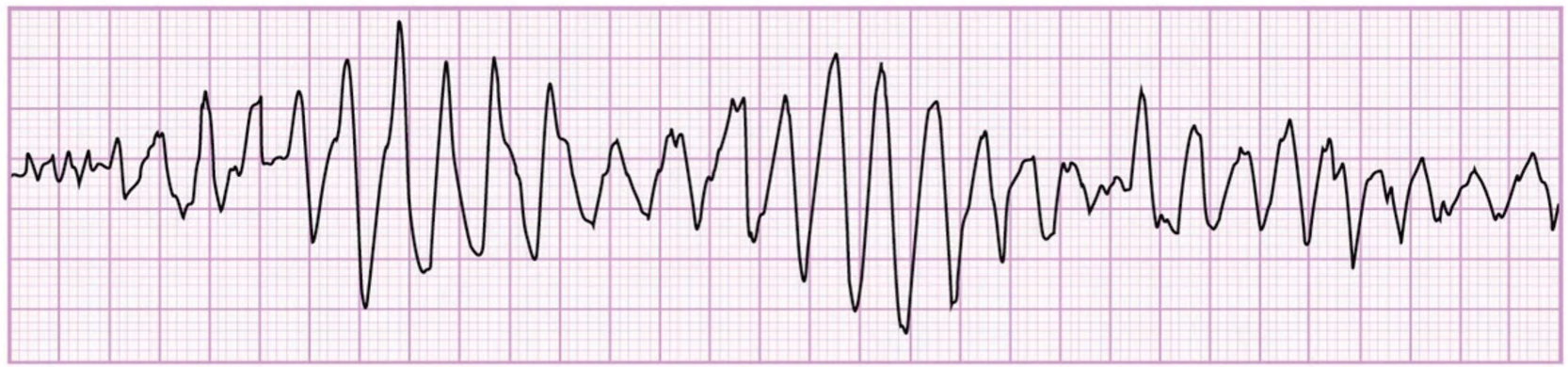
Torsade de pointes



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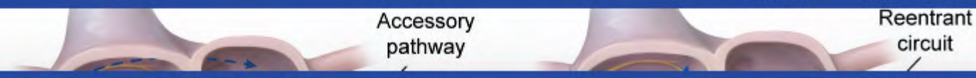

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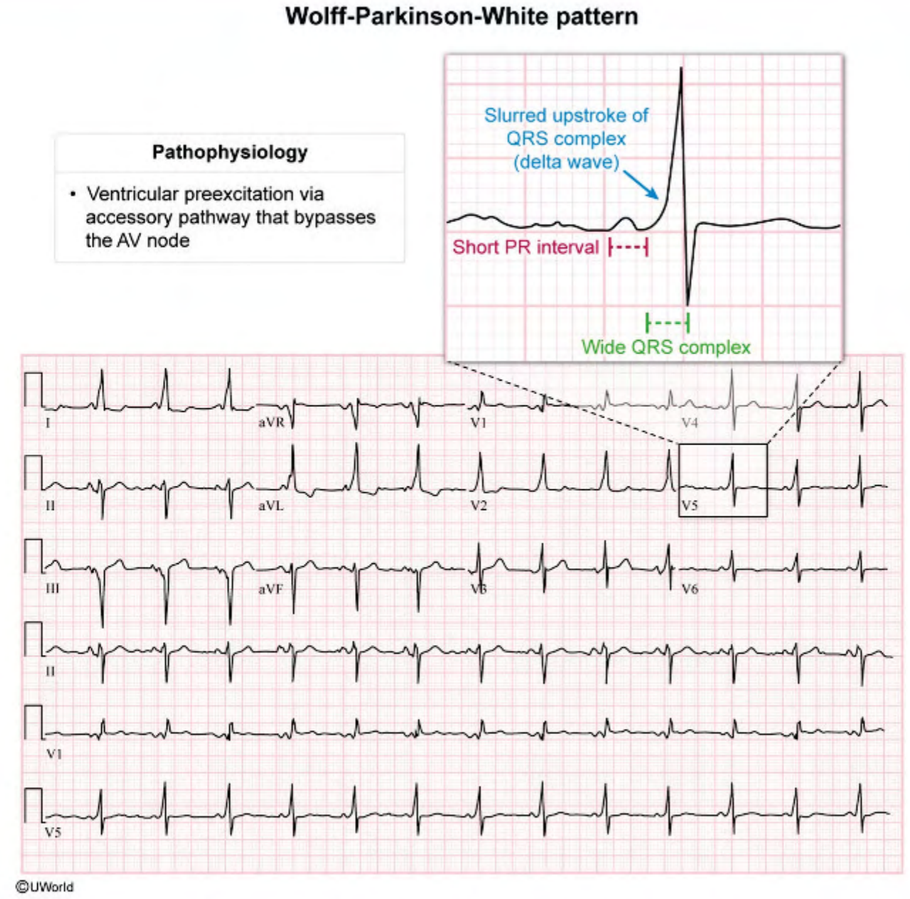

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A 42-year-old woman is brought to the hospital due to right-sided weakness and difficulty speaking. She has a longstanding history of a diastolic murmur, but her medical follow-up has been poor. She does not use tobacco, alcohol, or illicit drugs. A CT scan of the brain reveals a large ischemic stroke involving the left middle cerebral artery distribution. The patient dies 2 days later due to progressive neurologic deterioration. At autopsy, exploration of the left atrium shows diffuse fibrous thickening and distortion of the mitral valve leaflets, commissural fusion at the leaflet edges, and narrowing of the mitral valve orifice. This finding is most likely the result of which of the following conditions?

- A. Congenital heart disease
- B. Degenerative valvular calcinosis
- C. Infective endocarditis
- D. Late syphilis
- E. Rheumatic fever
- F. Rheumatoid arthritis
- G. Rupture of the papillary muscle

Submit

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- ✗ A. Congenital heart disease (3%)
- B. Degenerative valvular calcinosis (7%)
- C. Infective endocarditis (12%)
- D. Late syphilis (1%)
- ✓ E. Rheumatic fever (72%)
- F. Rheumatoid arthritis (0%)
- G. Rupture of the papillary muscle (1%)

Incorrect
Correct answer
E

72%
Answered correctly

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Time Spent

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Explanation

This patient's presentation suggests **mitral stenosis** (MS) due to rheumatic heart disease. Fibrous thickening and fusion of the valve leaflets in chronic rheumatic heart disease following acute **rheumatic fever** is the most common cause of MS, accounting for up to 99% of cases. There is often a latency period of 10-20 years between the initial episode of rheumatic fever and symptomatic MS, with most patients manifesting during the fourth or fifth decade of life.

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Mitral stenosis can cause atrial enlargement, which may lead to **atrial fibrillation** and/or atrial **mural thromboses**. Thrombi dislodged from the wall of the left atrium may later cause an embolic stroke, which likely occurred in this patient. Cardiac auscultation in **mitral stenosis** often reveals a loud first heart sound (S1), an early diastolic sound (opening snap), followed by a **mid-diastolic murmur** from turbulent flow across the mitral valve.

(Choice A) Congenital endocardial cushion defects can result in mitral regurgitation. However, congenital mitral valve stenosis is rare and untreated newborns and infants typically have a grim prognosis.

(Choice B) Degenerative calcific deposits most commonly develop in the mitral valve annulus in women over age 60 and generally do not impair valve function. This patient's autopsy did not show any peripherally located nodular calcific deposits.

(Choice C) Embolization of vegetations due to infective endocarditis can cause a stroke or septic infarct resulting in a brain abscess. However, infective endocarditis is usually associated with large friable vegetations on the valve cusps along with destruction (rather than fibrosis) of the valve leaflets.

(Choice D) Tertiary syphilis may result in aortitis, aortic aneurysm, and/or aortic regurgitation. Mitral valve lesions are not commonly observed.

(Choice F) Although cardiac involvement is uncommon in rheumatoid arthritis, patients can develop pericarditis and myocarditis. Rheumatoid nodules can also develop in the cardiac tissues (including valvular structures), but these are rarely symptomatic.

(Choice G) Rupture of papillary muscle (whether due to bacterial endocarditis, a connective tissue disease, or myocardial infarction) leads to prolapse of the valve leaflets and mitral regurgitation (holosystolic or early systolic murmur).

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(Choice G) Rupture of papillary muscle (whether due to bacterial endocarditis, a connective tissue disease, or myocardial infarction) leads to prolapse of the valve leaflets and mitral regurgitation (holosystolic or early systolic murmur).

Educational objective:
Rheumatic mitral stenosis is characterized by diffuse fibrous thickening and distortion of the mitral valve leaflets along with commissural fusion at the leaflet edges. Patients often present with a diastolic murmur, dyspnea, and fatigue and are at increased risk of atrial fibrillation and thromboembolism (eg, stroke).

Pathology	Cardiovascular System	Mitral stenosis
Subject	System	Topic

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A 78-year-old man is being evaluated for headaches that are relieved by over-the-counter acetaminophen. He takes no other medications and is a lifelong nonsmoker. The patient's blood pressure is 180/70 mm Hg and pulse is 75/min and regular. During a prior office visit, blood pressure was 175/68 mm Hg. Physical examination shows an S4 but is otherwise unremarkable. CT scan of the head reveals no significant abnormalities. Which of the following age-related changes best explains this patient's blood pressure readings?

- A. Decreased diameter of the aortic lumen
- B. Decreased stroke volume
- C. Increased arterial collagen deposition
- D. Increased renal arterial resistance
- E. Increased sympathetic tone

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- ✗ A. Decreased diameter of the aortic lumen (26%)
- B. Decreased stroke volume (8%)
- ✓ C. Increased arterial collagen deposition (42%)
- D. Increased renal arterial resistance (17%)
- E. Increased sympathetic tone (5%)

Incorrect
Correct answer
C

42%
Answered correctly

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Explanation

Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
Mild concentric LVH*	• Response to cardiomyocyte dropout & ↑ afterload • Resting EF, SV & cardiac output maintained • ↓ Maximal cardiac output
Conduction cell	• Slightly ↓ resting heart rate

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Explanation

Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
Mild concentric LVH*	• Response to cardiomyocyte dropout & ↑ afterload • Resting EF, SV & cardiac output maintained • ↓ Maximal cardiac output
Conduction cell degeneration	• Slightly ↓ resting heart rate • ↓ Maximal heart rate
Reduced baroreceptor sensitivity & adrenergic responsiveness	• ↑ Orthostasis • ↓ Heart rate & contractility response • ↑ Circulating catecholamines
*A soft S4 is a normal finding in the elderly. **EF** = ejection fraction; **HTN** = hypertension; **LVH** = left ventricular hypertrophy; **SV** = stroke volume.	

This patient's blood pressure readings show a pattern of elevated systolic blood pressure (SBP) with relatively low diastolic blood pressure (DBP) and elevated pulse pressure, consistent with **isolated systolic hypertension** (ISH). ISH is often seen in **elderly patients** (eg, age >65), representing 60%-80% of hypertension cases in this population.

Aging is associated with increased aortic stiffness caused by endothelial dysfunction and a change in extracellular matrix composition (ie, **replacement of elastin with collagen**). These changes **reduce aortic compliance**, which **increases pulse pressure** (pulse pressure is directly related to stroke volume and inversely related to aortic compliance). In youth, ample aortic compliance results in delayed retrograde reflection of pressure waves during ventricular systole, with the waves returning to the heart after aortic valve closure and assisting in coronary artery

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Reduced aortic compliance also causes less blood volume to be retained in the arterial system between left ventricular contractions, **slightly decreasing DBP**.

(Choice A) The aortic lumen slightly increases (not decreases) in diameter with age. Nonetheless, it is the compliance of the large arteries (rather than the diameter) that affects blood pressure. The diameter of the small arteries (ie, arterioles) determines systemic vascular resistance and is a primary determinant of DBP.

(Choice B) A decrease in stroke volume decreases pulse pressure, leading to reduced SBP. However, stroke volume is generally maintained with age; a physiologic decrease does not occur.

(Choice D) Renal arterial resistance can increase with age due to the development of renal artery atherosclerosis. This causes hypertension by activating the renin-angiotensin-aldosterone system to cause sodium retention and arteriolar vasoconstriction, resulting in increased DBP (and increased SBP due to unchanged pulse pressure) rather than ISH.

(Choice E) Increased sympathetic tone causes increased DBP (arteriolar vasoconstriction) and increased pulse

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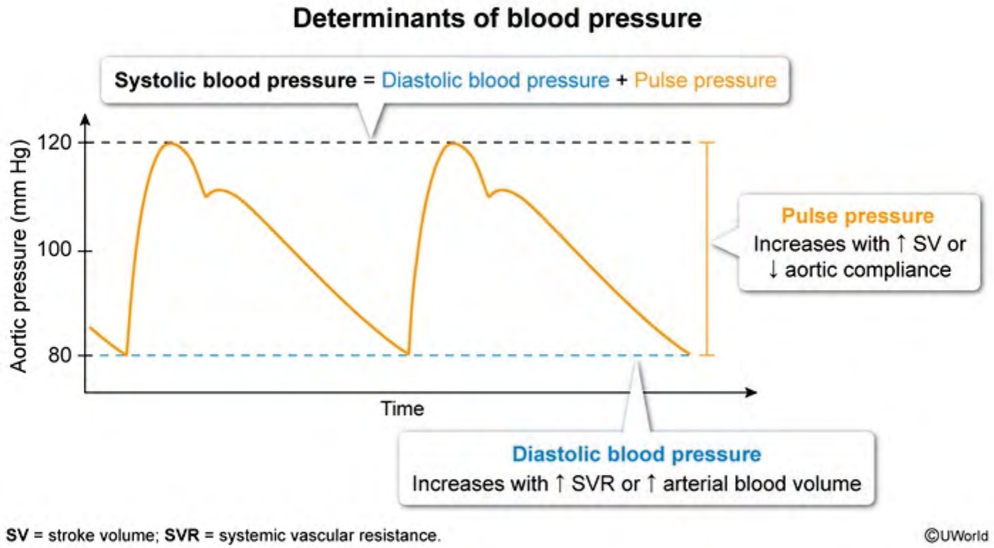

SV = stroke volume; **SVR** = systemic vascular resistance.

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compliance). In youth, ample aortic compliance results in delayed retrograde reflection of pressure waves during ventricular systole, with the waves returning to the heart after aortic valve closure and assisting in coronary artery perfusion. The reduced compliance that occurs with age hastens the retrograde reflection of pressure waves, causing the waves to return prior to aortic valve closure and increase the load on the left ventricle (ie, **increased SBP**).

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(Choice A) The aortic lumen slightly increases (not decreases) in diameter with age. Nonetheless, it is the compliance of the large arteries (rather than the diameter) that affects blood pressure. The diameter of the small arteries (ie, arterioles) determines systemic vascular resistance and is a primary determinant of DBP.

(Choice B) A decrease in stroke volume decreases pulse pressure, leading to reduced SBP. However, stroke volume is generally maintained with age; a physiologic decrease does not occur.

(Choice D) Renal arterial resistance can increase with age due to the development of renal artery atherosclerosis. This causes hypertension by activating the renin-angiotensin-aldosterone system to cause sodium retention and arteriolar vasoconstriction, resulting in increased DBP (and increased SBP due to unchanged pulse pressure) rather than ISH.

(Choice E) Increased sympathetic tone causes increased DBP (arteriolar vasoconstriction) and increased pulse pressure (increased contractility leading to increased stroke volume). Increased sympathetic tone often contributes to hypertension in patients age <50, but sympathetic tone decreases with age due to reduced responsiveness of adrenergic receptors.

Educational objective:
The isolated systolic hypertension commonly seen in elderly patients results from age-related stiffening (ie, replacement of elastin with collagen) and reduced compliance of the aorta and other large arteries.

References

- Systolic hypertension.

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A 72-year-old man is brought to the emergency department with waxing and waning chest pressure over the past several hours. The patient says he occasionally gets a similar sensation that lasts 5-10 minutes when he walks briskly up the stairs. Medical history is significant for hypertension and hyperlipidemia; he has been nonadherent with prescribed medical therapy and follow-up visits. The patient has a 25-pack-year smoking history. Blood pressure is 211/105 mm Hg, and pulse is 88/min and regular. Chest examination reveals no heart murmurs, and the lungs are clear. ECG shows sinus rhythm and nonspecific ST-segment changes. A medication is administered intravenously and produces the following hemodynamic effects compared to baseline:

Stroke volume	unchanged
Systemic vascular resistance	decreased
Heart rate	decreased

Which of the following medications was most likely used in this patient?

- A. Hydralazine
- B. Labetalol
- C. Metoprolol
- D. Nitroprusside
- E. Phentolamine

Submit

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Stroke volume	unchanged
Systemic vascular resistance	decreased
Heart rate	decreased

Which of the following medications was most likely used in this patient?

- ✗ A. Hydralazine (11%)
- ✓ B. Labetalol (59%)
- C. Metoprolol (9%)
- D. Nitroprusside (14%)
- E. Phentolamine (4%)

Incorrect
Correct answer
B

59%
Answered correctly

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Time Spent

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Version

Explanation

Systolic blood pressure **≥180 mm Hg** (or diastolic pressure ≥120 mm Hg) along with evidence of associated end-organ damage (eg, chest pressure, ST-segment changes on ECG) constitutes a **hypertensive emergency**. This

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Systolic blood pressure **≥180 mm Hg** (or diastolic pressure ≥120 mm Hg) along with evidence of associated end-organ damage (eg, chest pressure, ST-segment changes on ECG) constitutes a **hypertensive emergency**. This patient likely has underlying coronary artery disease and is experiencing myocardial ischemia due to increased myocardial oxygen demand caused by markedly elevated blood pressure and increased left ventricular afterload.

Management of hypertensive emergency involves prompt and controlled blood pressure reduction to reduce the risk of complications (eg, stroke, aortic dissection, myocardial infarction). **Labetalol** is a **nonselective vasodilatory beta blocker** that blocks beta-1, beta-2, and alpha-1 adrenergic receptors and reduces blood pressure. The **alpha-1 receptor blockade** outweighs beta-2 receptor blockade in vascular smooth muscle to cause **peripheral vasodilation** of both venules and arterioles. Venular vasodilation decreases venous return and arteriolar vasodilation **decreases systemic vascular resistance** (SVR); because the vasodilation is relatively balanced, blood pressure is reduced with **stroke volume unchanged**. Normally, reduced blood pressure stimulates a baroreceptor reflex-mediated increase in heart rate, but the strong beta-1 receptor blockade of labetalol overrides this response, causing overall **decreased heart rate**.

(Choices A, D, and E) Hydralazine is an arterial vasodilator that has little effect on veins. Nitroprusside is a nitric oxide donor that causes balanced arterial and venous vasodilation. Phentolamine is a nonselective alpha receptor blocker that also causes balanced vasodilation. All these drugs reduce blood pressure primarily via a reduction in SVR, but they have no negative chronotropic effects and are associated with a baroreceptor reflex-mediated increase in heart rate. Stroke volume is increased with hydralazine, and unchanged with the balanced vasodilation that occurs with nitroprusside and phentolamine.

(Choice C) Metoprolol is a cardioselective beta blocker that primarily blocks beta-1 receptors; it causes slight blockade of beta-2 receptors and has no effect on alpha receptors. Metoprolol causes no significant change in SVR but facilitates a mild reduction in blood pressure due to a decrease in contractility and heart rate that reduces stroke volume and cardiac output.

Educational objective:
Labetalol is a nonselective vasodilatory beta blocker that blocks beta-1, beta-2, and alpha-1 adrenergic receptors. Alpha-1 receptor blockade outweighs beta-2 receptor blockade to cause peripheral vasodilation with decreased venous return and lower systemic vascular resistance. The relatively balanced vasodilation reduces blood

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Management of hypertensive emergency involves prompt and controlled blood pressure reduction to reduce the risk of complications (eg, stroke, aortic dissection, myocardial infarction). **Labetalol** is a **nonselective vasodilatory beta blocker** that blocks beta-1, beta-2, and alpha-1 adrenergic receptors and reduces blood pressure. The **alpha-1 receptor blockade** outweighs beta-2 receptor blockade in vascular smooth muscle to cause **peripheral vasodilation** of both venules and arterioles. Venular vasodilation decreases venous return and arteriolar vasodilation **decreases systemic vascular resistance** (SVR); because the vasodilation is relatively balanced, blood pressure is reduced with **stroke volume unchanged**. Normally, reduced blood pressure stimulates a baroreceptor reflex-mediated increase in heart rate, but the strong beta-1 receptor blockade of labetalol overrides this response, causing overall **decreased heart rate**.

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Educational objective:
Labetalol is a nonselective vasodilatory beta blocker that blocks beta-1, beta-2, and alpha-1 adrenergic receptors. Alpha-1 receptor blockade outweighs beta-2 receptor blockade to cause peripheral vasodilation with decreased venous return and lower systemic vascular resistance. The relatively balanced vasodilation reduces blood pressure without changing stroke volume. Beta-1 receptor blockade overrides the expected baroreflex-mediated increase in heart rate, resulting in overall decreased heart rate.

Pharmacology Cardiovascular System Malignant hypertension

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Expl

Exhibit Display

Hypertensive crisis	
Hypertensive urgency	• Severe hypertension (≥180/120 mm Hg) • No evidence of end-organ damage
Hypertensive emergency	• Severe hypertension with end-organ damage: ◦ Cardiovascular: angina, myocardial infarction, aortic dissection ◦ CNS: encephalopathy, cerebral infarct/hemorrhage ◦ Kidney: acute hypertensive nephrosclerosis ◦ Lung: pulmonary edema ◦ Retina: papilledema, flame-shaped hemorrhages, exudates

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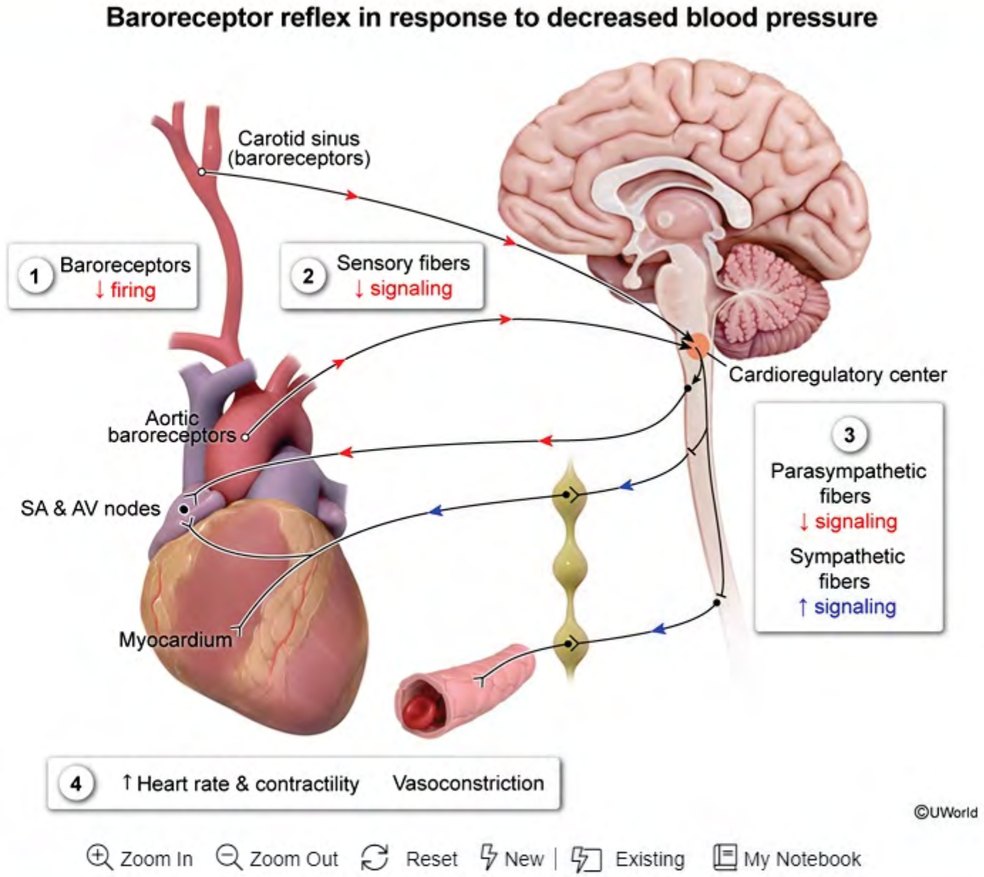

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SVR but facilitates a mild reduction in blood pressure due to a decrease in contractility and heart rate that reduces stroke volume and cardiac output.

Educational objective:

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A 66-year-old man is brought to the hospital due to sudden-onset chest pain and nausea. Medical history includes diabetes mellitus. Blood pressure is 70/60 mm Hg and pulse is 60/min. The lungs are clear on auscultation, and the jugular veins are distended. ECG shows ST-segment elevation in leads II, III, and aVF. Chest x-ray is unremarkable. An inferior wall myocardial infarction is diagnosed. Emergency cardiac catheterization reveals complete occlusion of the proximal right coronary artery. The patient is persistently hypotensive in the cardiac catheterization laboratory. Which of the following hemodynamic findings are most likely to be observed in this patient?

	Cardiac output	Pulmonary capillary wedge pressure	Central venous pressure
○ A.	↓	↓	↓
○ B.	↓	↓	↑
○ C.	↓	↑	↓
○ D.	↑	↓	↓
○ E.	↑	↑	↑

Submit

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	Cardiac output	Pulmonary capillary wedge pressure	Central venous pressure
✗ A.	↓	↓	↓
✓ B.	↓	↓	↑
C.	↓	↑	↓
D.	↑	↓	↓
E.	↑	↑	↑

Incorrect
Correct answer
B

Collecting Statistics

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Time Spent

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Explanation

Right-sided heart failure

Primary disturbance
Right ventricular dysfunction

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A 66-year-old man is brought to the hospital due to sudden-onset chest pain and nausea. Medical history includes
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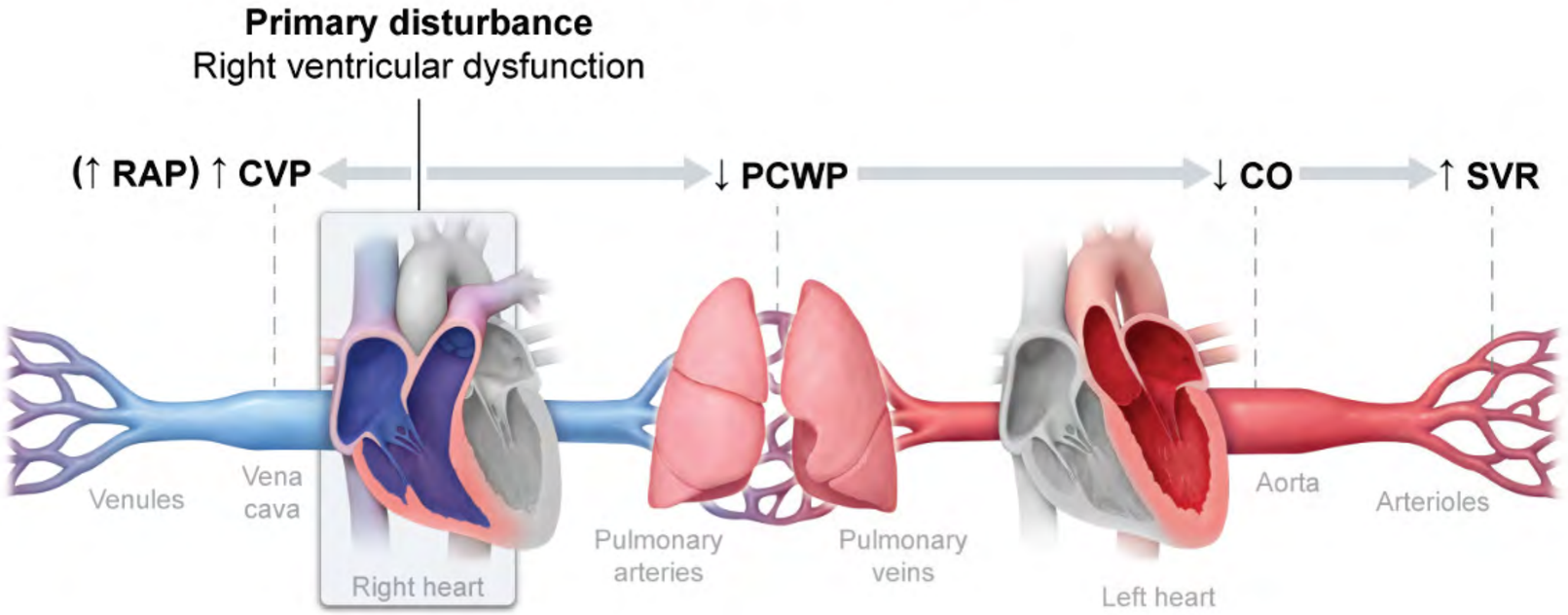
Right-sided heart failure



CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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Right ventricular dysfunction

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PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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This patient has an acute myocardial infarction (MI) affecting the **inferior wall**, which is usually supplied by the right coronary artery. Inferior wall MIs can involve only the left ventricle, both the left and right ventricles, or only the right ventricle. This patient's **severe hypotension**, prominent jugular venous distension, and clear lungs (ie, **absence of pulmonary edema**) suggest **primarily right ventricular** involvement.

In acute right ventricular MI, contractile dysfunction causes pressure to back up into the right atrium and vena cava, creating elevated central venous pressure **(↑ CVP) (Choice C)**. RV output drops, decreasing blood flow through the lungs to the left atrium and creating low pulmonary capillary wedge pressure **(↓ PCWP)**. With less blood reaching the left atrium, **left ventricular filling decreases** and systemic cardiac output drops, causing hypotension and a reflexive increase in systemic vascular resistance (SVR) in an effort to maintain blood pressure.

These hemodynamic findings of right ventricular MI **mimic** those seen with obstructive shock involving blockage of blood flow through the pulmonary circulation (eg, pulmonary embolism), and contrast with classic cardiogenic shock resulting from left ventricular contractile dysfunction. In classic cardiogenic shock, blood backs up at the left ventricle, causing increased left atrial pressure (ie, increased PCWP) and pulmonary edema.

(Choice A) In hypovolemic shock, low blood volume causes low CVP, which leads to low PCWP and cardiac output.

(Choice D) In septic shock, peripheral vasodilation reduces SVR, and there is a compensatory increase in cardiac output. CVP and PCWP are low due to vascular leakage and low circulating blood volume.

(Choice E) In high-output heart failure, low SVR triggers an increase in cardiac output in an effort to maintain blood pressure. Despite high output, the heart cannot keep up with the resulting increase in venous return, and elevated PCWP and CVP develop.

Educational objective:
Inferior wall myocardial infarction (MI) commonly involves the right ventricle. MI primarily involving the right ventricle is marked by elevated central venous pressure, low pulmonary capillary wedge pressure (due to reduced blood flow through the lungs to the left atrium), and low cardiac output. This contrasts with classic cardiogenic shock, which results from left ventricular failure and leads to elevated pulmonary capillary wedge pressure with

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References

- Right ventricular myocardial infarction: from pathophysiology to prognosis.

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PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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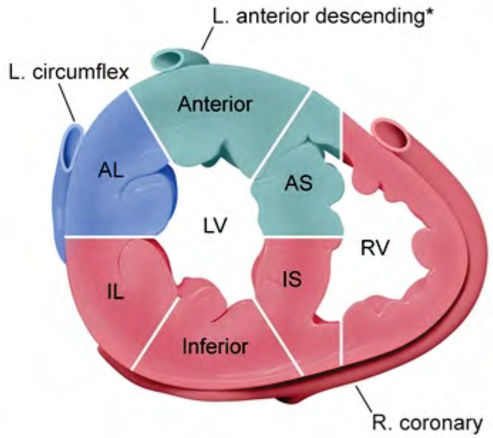
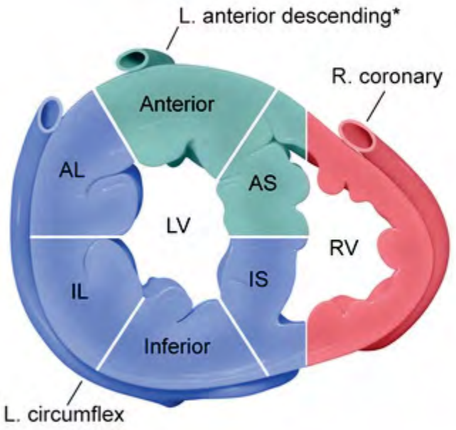
Coronary artery dominance

Right dominant circulation
~85% of patients



Left dominant circulation
~10% of patients



*The anterior wall is substantially larger than the inferior wall in 3 dimensions, such that the left anterior descending artery supplies ~50% of the total left ventricular myocardium.
AL = anterolateral; **AS** = anteroseptal; **IL** = inferolateral; **IS** = inferoseptal; **LV** = left ventricle; **RV** = right ventricle. ©UWorld

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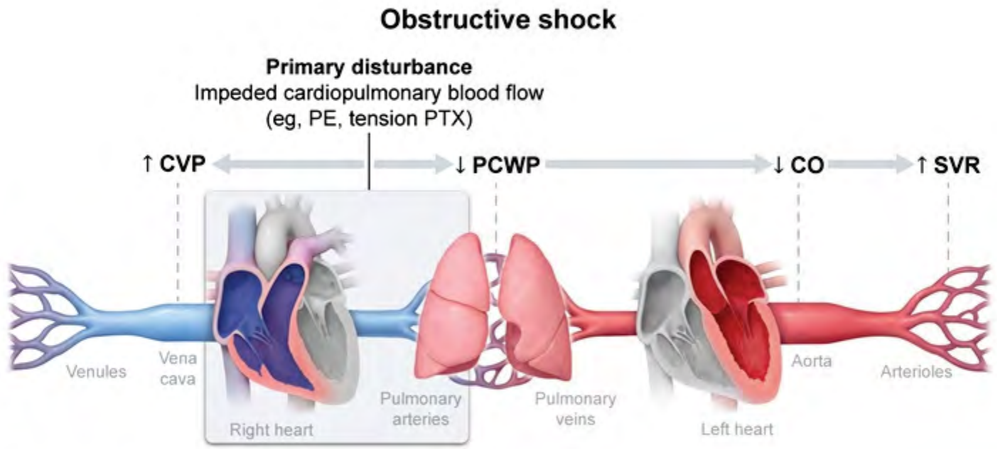

CO = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **PE** = pulmonary embolism; **PTX** = pneumothorax; **SVR** = systemic vascular resistance.

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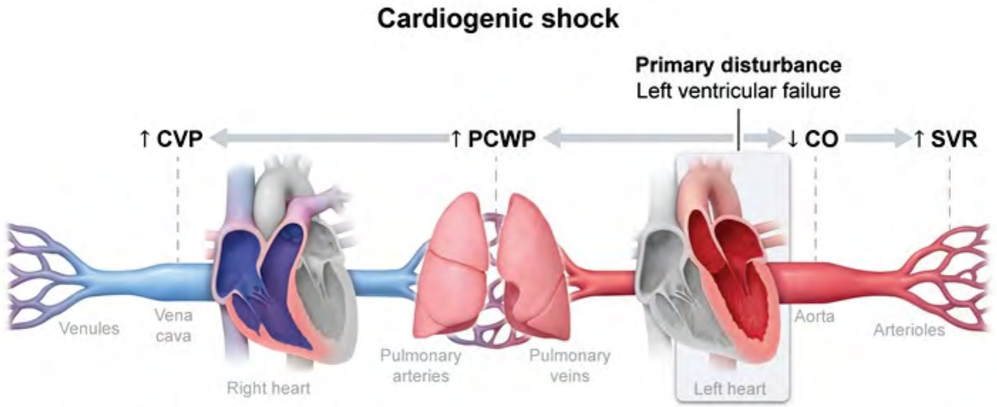

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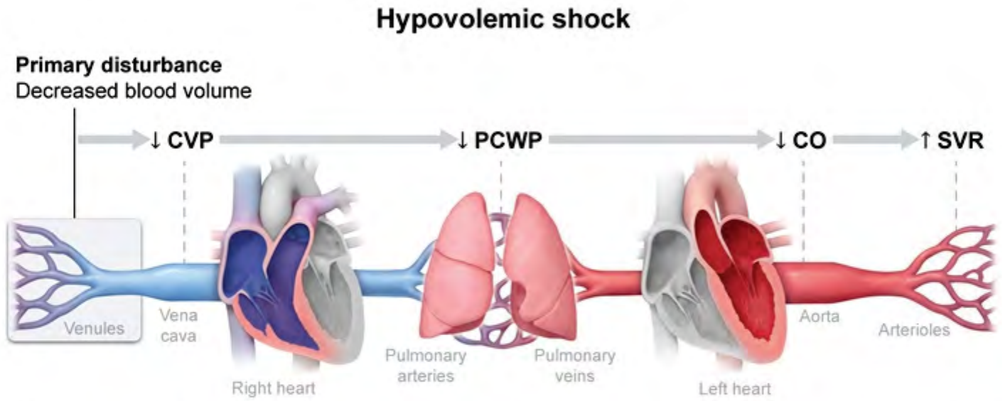

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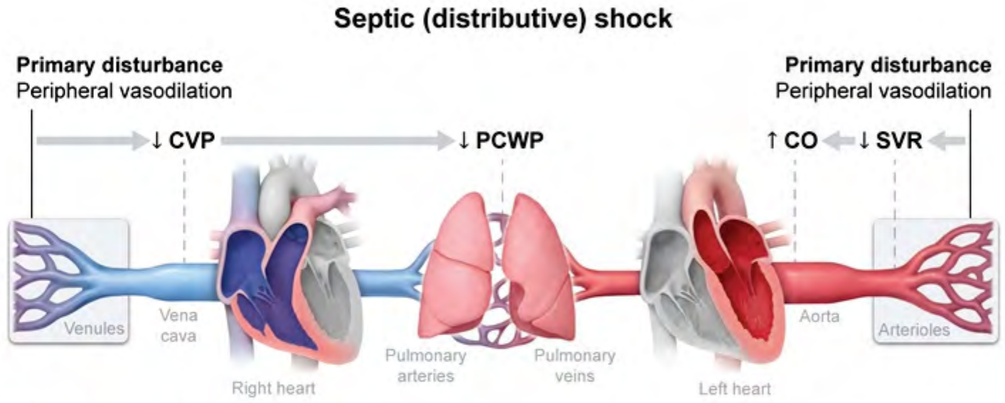

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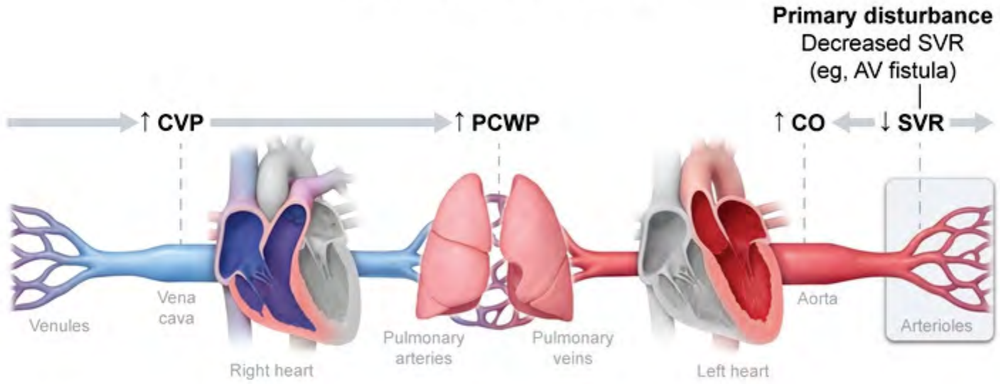
High-output heart failure



AV = arteriovenous; **CO** = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.
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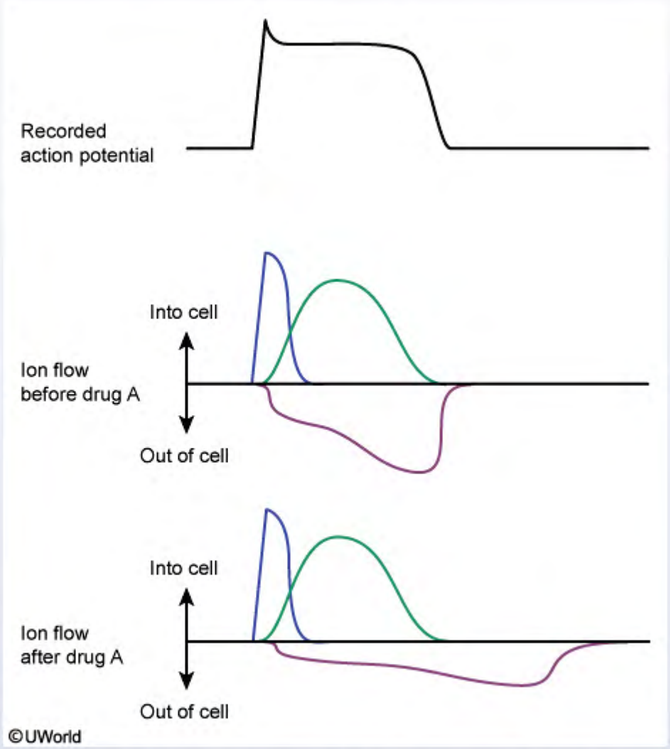
A new antiarrhythmic medication (drug A) is tested in a series of experiments that measure the flow of ions responsible for producing an action potential in cardiac muscle cells. Ion flow is measured before and after the drug is administered, and the results are shown in the chart below (each colored line represents a different type of ion).



Drug A has an antiarrhythmic effect most similar to which of the following drugs?

- A. Adenosine
- B. Digoxin
- C. Dofetilide

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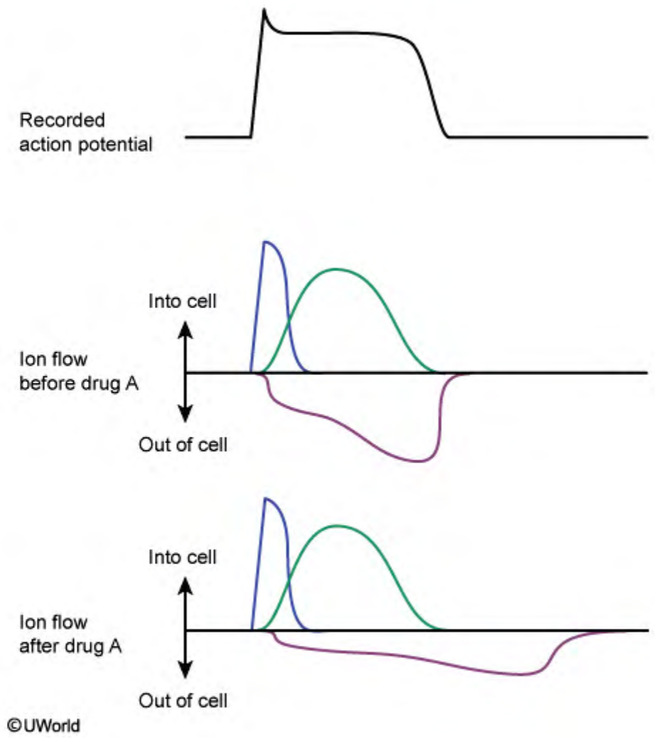

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Drug A

C. Dofetilide

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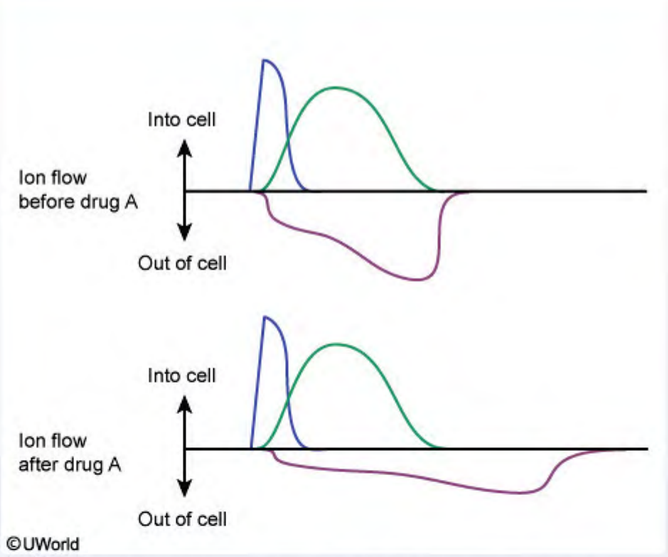

Drug A has an antiarrhythmic effect most similar to which of the following drugs?

- A. Adenosine
- B. Digoxin
- C. Dofetilide
- D. Esmolol
- E. Flecainide
- F. Lidocaine
- G. Quinidine
- H. Verapamil

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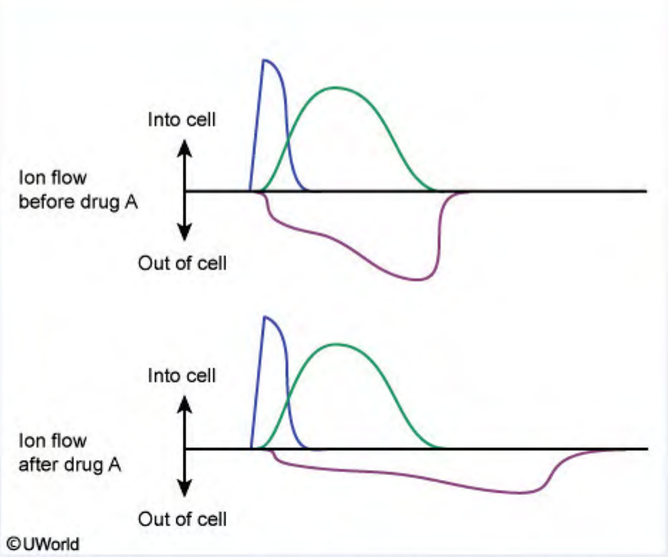

Drug A has an antiarrhythmic effect most similar to which of the following drugs?

- A. Adenosine (8%)
- ✗ B. Digoxin (5%)
- ✓ C. Dofetilide (59%)
- D. Esmolol (4%)
- E. Flecainide (5%)
- F. Lidocaine (3%)
- G. Quinidine (7%)
- H. Verapamil (5%)

Incorrect
Correct answer
C

59%
Answered correctly

10 secs
Time Spent

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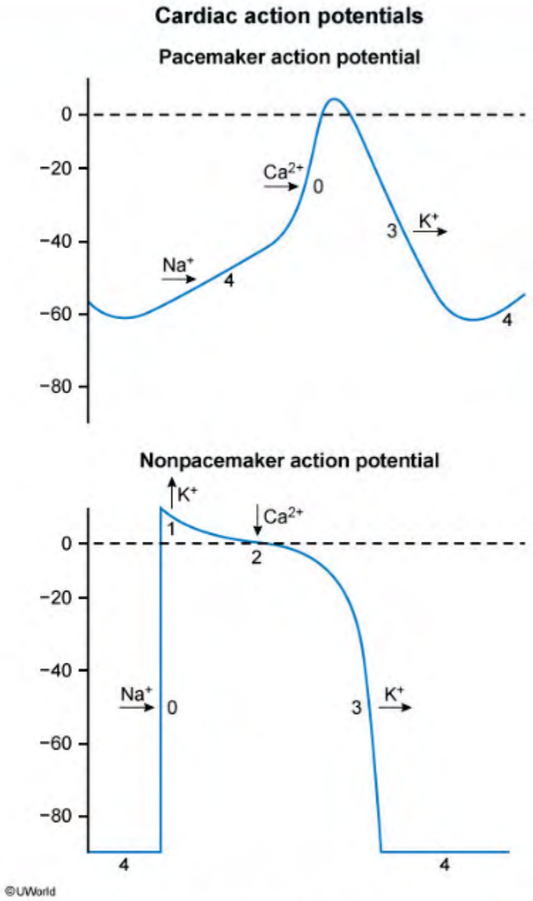
Explanation

Exhibit Display



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The recorded action potential shows the typical profile of **non-pacemaker** cardiac cells, which includes the following phases:

1. **Phase 0 (rapid depolarization)** – occurs when the resting membrane potential reaches threshold; is characterized by rapid sodium entry into the cell due to activation and opening of voltage-dependent sodium channels.
2. **Phase 1 (early repolarization)** – caused primarily by activation of transient outward potassium currents along with a rapid decline in inward sodium current.
3. **Phase 2 (plateau phase)** – stable membrane potential due to a balance of late inward calcium current (L-type channels) and outward potassium current.
4. **Phase 3 (late repolarization)** – characterized by the opening of additional potassium channels (delayed-rectifier potassium currents) and continued efflux of potassium ions from the cell; this is in conjunction with the decay of inward calcium current.
5. **Phase 4 (resting)** – normal resting membrane potential is between −80 mV and −95 mV, and it is generated by outward potassium flow through potassium leak channels open at rest. The Na^+-K^+-ATPase pump allows the potassium and sodium gradients to be maintained across the membrane.

The ion flow graph after drug A administration shows a **decrease in outward potassium flow** from the myocyte during phase 3 repolarization of the myocardial action potential. Class III antiarrhythmic drugs (eg, amiodarone, sotalol, **dofetilide**) predominantly block potassium channels and inhibit the outward potassium currents during phase 3 of the cardiac action potential, thereby prolonging repolarization and total action potential duration.

(Choice A) Adenosine stimulates A1 receptors on the surface of cardiac cells, activating potassium channels and increasing potassium conductance, which causes the membrane potential to remain negative for a longer period. This results in transient slowing of the sinus rate and increased atrioventricular (AV) nodal conduction delay.

(Choice B) Digoxin inhibits the Na^+-K^+-ATPase pump in myocardial cells, resulting in an increase in intracellular sodium, which leads in turn to a rise in intracellular calcium concentration. Digoxin does not affect potassium channels and has minimal effect on potassium efflux from myocardial cells during the action potential.

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(Choice B) Digoxin inhibits the Na^+-K^+-ATPase pump in myocardial cells, resulting in an increase in intracellular sodium, which leads in turn to a rise in intracellular calcium concentration. Digoxin does not affect potassium channels and has minimal effect on potassium efflux from myocardial cells during the action potential.

(Choice D) Esmolol is a rapid-acting, short-duration beta blocker (class II antiarrhythmic) that slows the rate of discharge of sinus or ectopic pacemakers and increases the refractory period of the AV node. It has no effect on potassium efflux from myocardial cells.

(Choices E, F, and G) Class IA (procainamide, quinidine), IB (lidocaine), and IC (flecainide) antiarrhythmic drugs inhibit sodium-dependent (phase 0) depolarization and slow conduction. Class IA and IC agents also have some potassium-blocking activity and can cause prolongation of the action potential (seen mostly with class IA agents). However, all class I drugs have prominent sodium channel-blocking activity, which would blunt the blue tracing in the above graph.

(Choice H) Calcium channel blockers (eg, verapamil, diltiazem) inhibit the L-type calcium channels during phase 2 of the myocardial action potential and phase 0 of the pacemaker action potential. They slow the sinus rate, prolong conduction through the AV node, and depress myocardial contractility.

Educational objective:
Class III antiarrhythmic drugs (eg, amiodarone, sotalol, dofetilide) predominantly block potassium channels and inhibit the outward potassium currents during phase 3 of the cardiac action potential, thereby prolonging repolarization and total action potential duration.

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2. **Phase 1 (early repolarization)** – caused primarily by activation of transient outward potassium currents along

Exhibit Display

Antiarrhythmic drugs – Vaughan Williams classification

Class	Drugs	Predominant actions
IA	Sodium channel blockers • Disopyramide • Procainamide • Quinidine	• Slows AP conduction velocity • Prolongs APD
IB	• Lidocaine • Mexiletine • Phenytoin	• No effect on AP conduction velocity • Shortens APD
IC	• Flecainide • Propafenone	• Slows AP conduction velocity • Minimal effect on APD
II	Beta receptor blockers • Atenolol • Metoprolol • Esmolol • Carvedilol	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness
III	Potassium channel blockers • Amiodarone • Dronedarone • Dofetilide • Sotalol (also class II)	• No effect on AP conduction velocity • Prolongs APD
IV	Non-dihydropyridine calcium channel blockers • Verapamil • Diltiazem	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness

AP = action potential; **APD** = action potential duration; **AV** = atrioventricular.

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potassium-blocking activity and can cause prolongation of the action potential (seen mostly with class IA agents).

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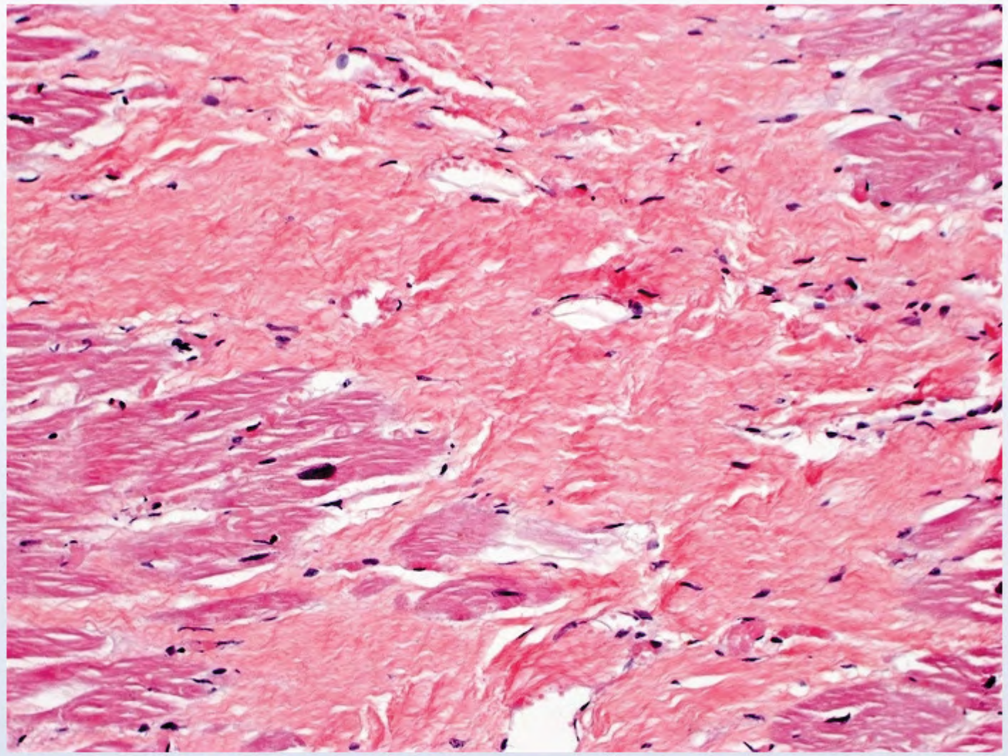
A 67-year-old man is found dead in his home. The cause of death is not apparent; he had a long history of hypertension and he had a myocardial infarction a year ago. An autopsy is performed. Gross examination of the heart shows white scarring and enlargement of the left ventricle. Histologic findings from a section of the left ventricle are shown below.



The type of collagen seen in the autopsy sample is most likely to be extensively found in which of the following normal body tissues?

- A. Basement membrane
- B. Granulation tissue

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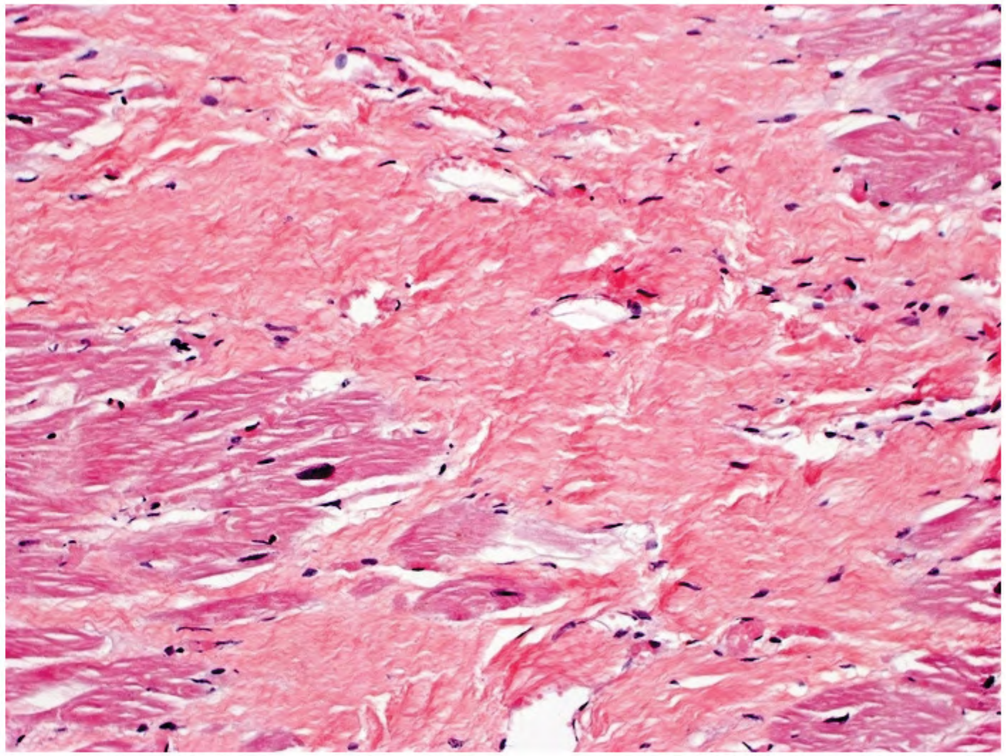

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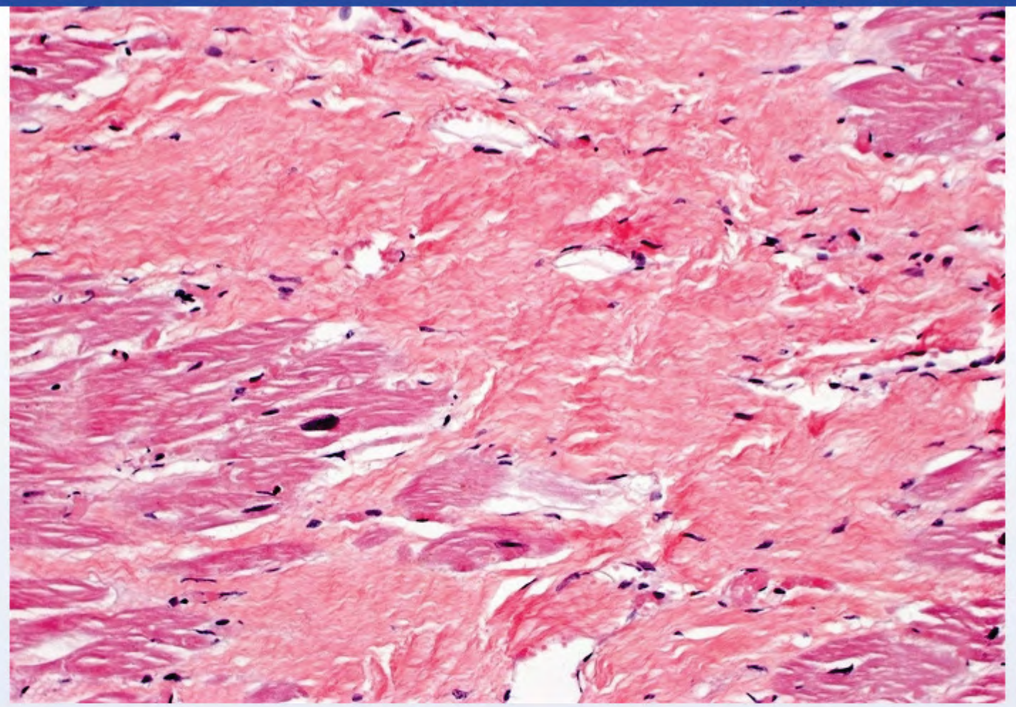

The type of collagen seen in the autopsy sample is most likely to be extensively found in which of the following normal body tissues?

- A. Basement membrane (7%)
- ✕ B. Granulation tissue (34%)
- C. Hyaline cartilage (15%)
- D. Nucleus pulposus (3%)
- ✓ E. Tendon (39%)

Incorrect
Correct answer
E

39%
Answered correctly

35 secs
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Explanation

Collagen subtypes		
Type	**Locations**	**Associated diseases**
I	Skin, bone, tendons, ligaments, dentin, cornea, blood vessels & scar tissue	Osteogenesis imperfecta
II	Cartilage, vitreous humor & nucleus pulposus	Skeletal dysplasias
III	Skin, lungs, intestines, blood vessels, bone marrow, lymphatics & granulation tissue	Vascular Ehlers-Danlos syndrome (Type IV)
IV	Basement membrane	Alport syndrome

Evolution of a myocardial infarction occurs in several stages. The final stage of the healing process begins approximately 2 weeks after infarction and involves increased deposition of **type I collagen**. Fibrosis continues until about 2 months after infarction, resulting in a dense collagenous scar composed primarily of type I collagen. The scar can be identified grossly as a firm, gray-white lesion or microscopically as collagenous tissue replacing myocardium (as in this patient).

In addition to its role in **wound healing**, type I collagen (the most abundant type) provides **strength** and support for a variety of cellular tissues and organs (eg, **tendons, ligaments, skin**). It is also the major organic component in **bones** and significantly contributes to overall bone strength. Defects in type I collagen can occur in osteogenesis imperfecta, an autosomal dominant disorder that may present with fragile bones and ligament laxity.

(Choices A, C, and D) Basement membrane contains type IV collagen; hyaline cartilage (eg, articular surfaces) and nucleus pulposus contain type II collagen.

(Choice B) Granulation tissue is a capillary-rich fibroblastic proliferation composed primarily of type III collagen. Although granulation tissue is seen approximately 7 days after an acute myocardial infarction, it is eventually replaced by type I collagen as the infarct heals. Type III collagen is involved in the structural maintenance of extensible connective tissues and is often found alongside type I collagen.

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III	Skin, lungs, intestines, blood vessels, bone marrow, lymphatics & granulation tissue	Vascular Ehlers-Danlos syndrome (Type IV)
IV	Basement membrane	Alport syndrome

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Educational objective:
Type I collagen is the primary collagen in mature scars (eg, remote myocardial infarction). It is the most prevalent type of collagen and provides strength and support throughout the human body, particularly in bones, tendons, ligaments, and skin.

Histology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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	Skin, lungs, intestines, blood vessels, bone marrow, lymphatics	Vascular Ehlers-Danlos syndrome

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Time after myocardial infarction	Predominant light microscopic changes
0-4 hours	• No visible change
4-12 hours	• Wavy fibers with narrow, elongated myocytes
12-24 hours	• Myocyte hypereosinophilia with pyknotic (shrunken) nuclei
1-3 days	• Coagulation necrosis (loss of nuclei & striations) • Prominent neutrophilic infiltrate
3-7 days	• Disintegration of dead neutrophils & myofibers • Macrophage infiltration at border areas
7-10 days	• Robust phagocytosis of dead cells by macrophages • Beginning formation of granulation tissue at margins
10-14 days	• Well-developed granulation tissue with neovascularization
2-8 weeks	• Progressive collagen deposition & scar formation

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II	Skin, lungs, intestines, blood vessels, bone marrow, lymphatics	Vascular Ehlers-Danlos syndrome
IV		

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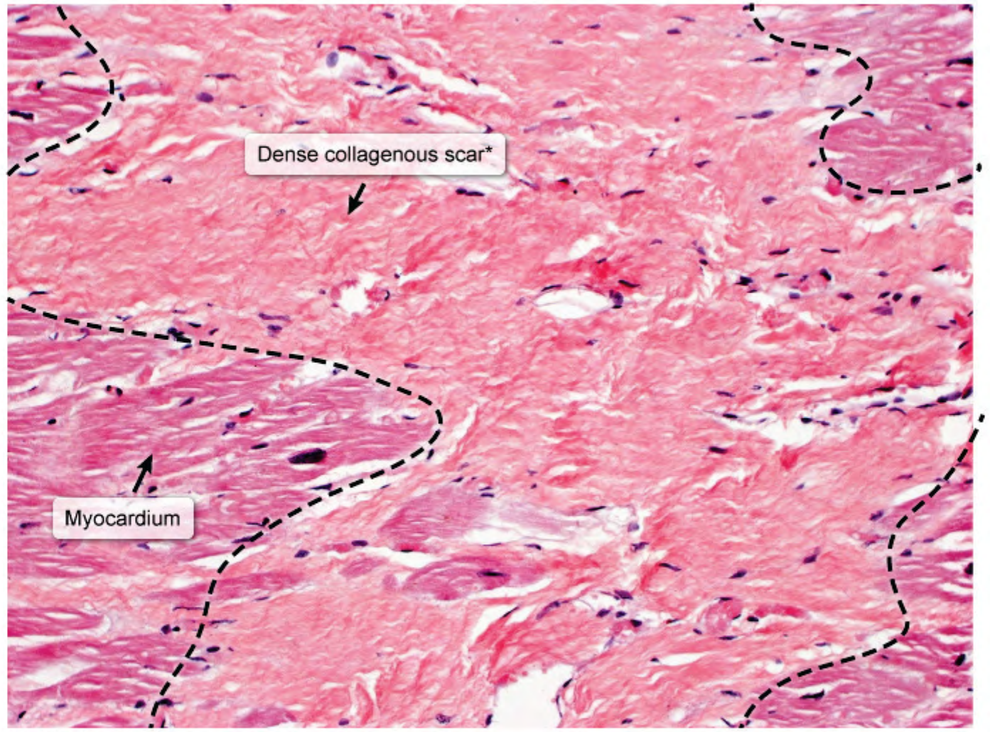
Remote myocardial infarction



*Type I collagen

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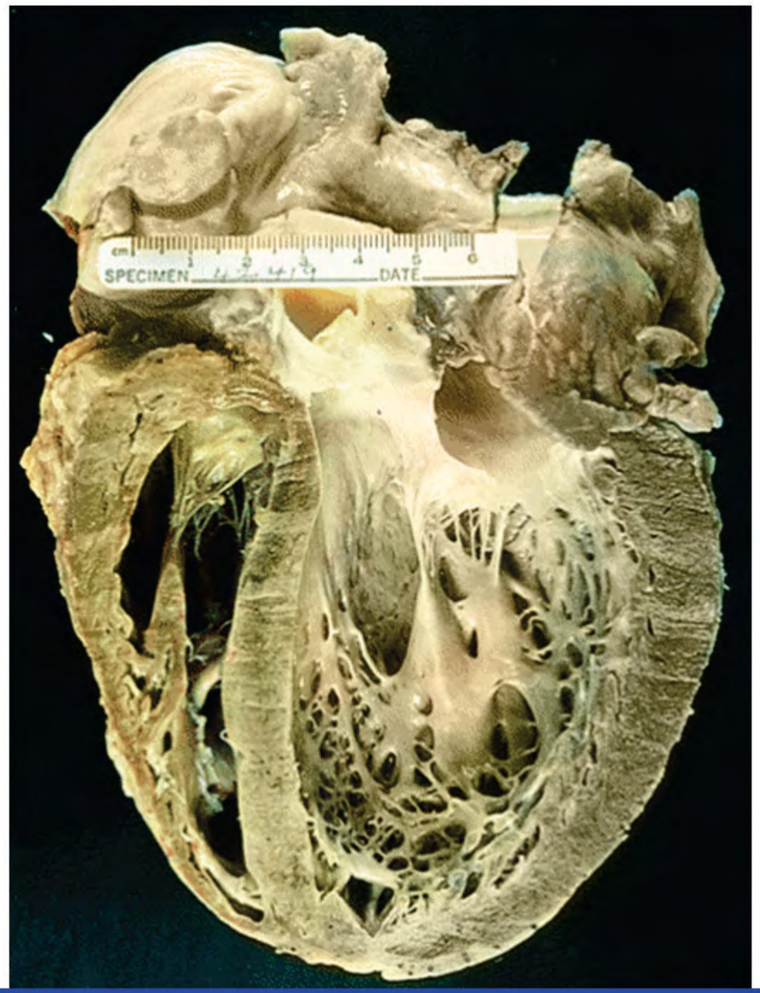
A 42-year-old man comes to the emergency department due to fatigue and exertional dyspnea. Over the last 2 weeks his symptoms have progressively worsened to the point that he can no longer walk across his living room without becoming short of breath. He is admitted to the hospital but dies suddenly despite aggressive treatment. An autopsy is performed, and a cross-section of his heart is shown in the image below.



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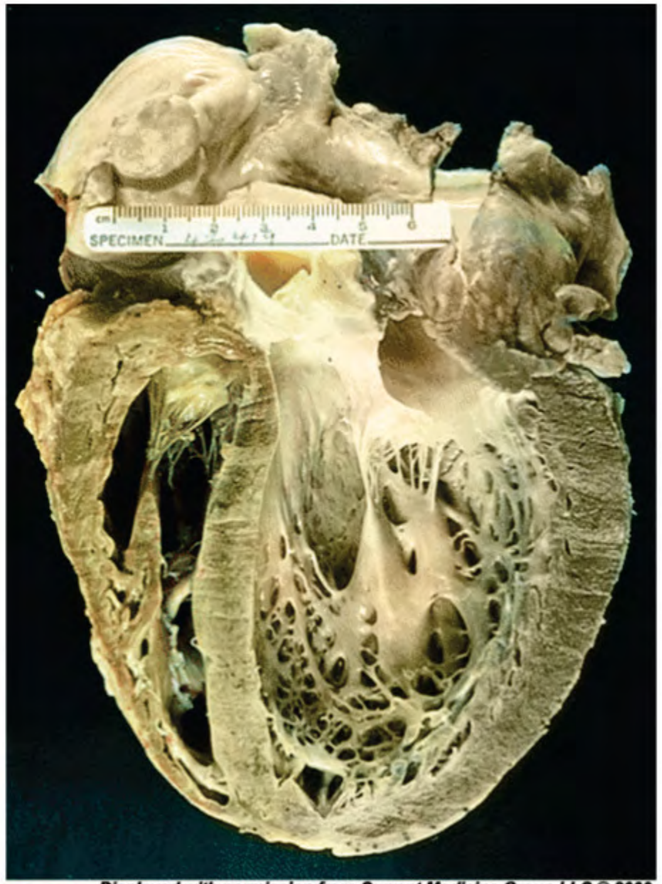

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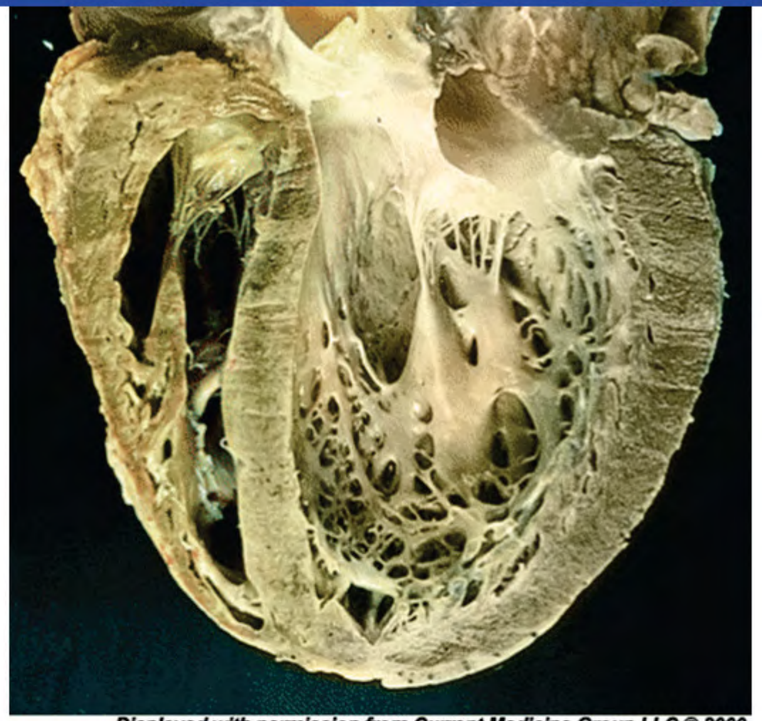

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This patient's symptoms were most likely caused by which of the following mechanisms?

- A. Decreased ventricular contraction force
- B. Dynamic left ventricular outflow obstruction
- C. Impaired left ventricular filling
- D. Increased pulmonary vascular resistance
- E. Poor ventricular wall compliance

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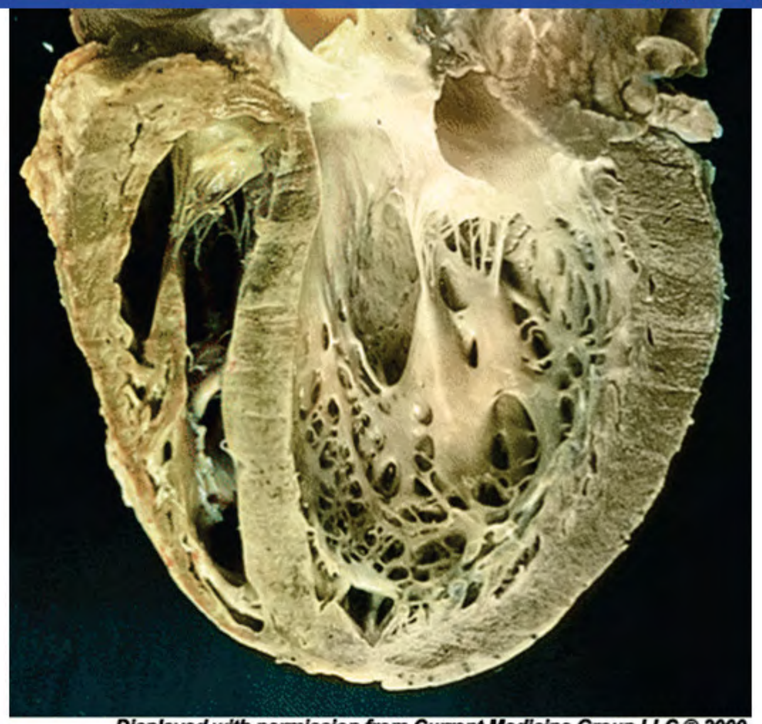

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This patient's symptoms were most likely caused by which of the following mechanisms?

- ✓ A. Decreased ventricular contraction force (64%)
- B. Dynamic left ventricular outflow obstruction (12%)
- C. Impaired left ventricular filling (6%)
- D. Increased pulmonary vascular resistance (6%)
- E. Poor ventricular wall compliance (9%)

Correct

64% Answered correctly

19 secs Time Spent

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Explanation

Causes of nonvalvular heart failure				
	Ventricular wall thickness	**Ventricular cavity size**	**Contractile function**	**Diastolic function**
Ischemic heart disease	↓ or normal	↑	↓	↓ or normal
Dilated cardiomyopathy*				
Hypertensive heart disease	↑	↓	normal	↓
Hypertrophic cardiomyopathy*	↑ septal thickening	↓	normal	↓
Restrictive cardiomyopathy*	↑ or normal	↓ or normal	normal	↓
*Cardiomyopathies are characterized by primary myocardial dysfunction (eg, viral myocarditis, genetic disorder, infiltrative disease). Myocardial disease caused by secondary factors (eg, hypertension, coronary artery disease, valvular dysfunction) is not classified as a cardiomyopathy.				

The gross specimen of this patient's heart demonstrates **left ventricular (LV) cavity enlargement** consistent with eccentric hypertrophy, which is initially an adaptive mechanism that allows the LV to maintain cardiac output in the setting of increasing **volume overload**. Multiple disease processes can lead to eccentric hypertrophy, including **ischemic heart disease**, certain types of valvular disease (ie, aortic regurgitation, mitral regurgitation), and primary myocardial dysfunction (eg, due to viral infection, chronic alcohol abuse, inherited disease) manifesting as **dilated cardiomyopathy**.

Eventually, progressive eccentric enlargement of the LV becomes maladaptive with overwhelming wall stress leading to **decreased ventricular contractility**. The reduced cardiac output leads to **decompensated heart failure** manifesting with fatigue, dyspnea, orthopnea, and peripheral edema. The structural changes to the LV also

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primary myocardial dysfunction (eg, due to viral infection, chronic alcohol abuse, inherited disease) manifesting as **dilated cardiomyopathy**.

Eventually, progressive eccentric enlargement of the LV becomes maladaptive with overwhelming wall stress leading to **decreased ventricular contractility**. The reduced cardiac output leads to **decompensated heart failure** manifesting with fatigue, dyspnea, orthopnea, and peripheral edema. The structural changes to the LV also increase the risk of ventricular arrhythmia (ie, ventricular tachycardia, ventricular fibrillation), a potential cause of sudden cardiac death in these patients.

(Choice B) Dynamic LV outflow tract (LVOT) obstruction can be seen in hypertrophic cardiomyopathy as the degree of LVOT obstruction varies with changes in LV blood volume. Maneuvers or clinical conditions that decrease LV blood volume (eg, Valsalva strain phase, dehydration) exacerbate the obstruction, whereas those that increase LV blood volume (eg, squatting) lessen or may resolve the obstruction.

(Choices C and E) Poor ventricular wall compliance leads to impaired LV filling (diastolic dysfunction). This most commonly occurs as the initial manifestation of hypertensive heart disease with pressure overload leading to concentric LV hypertrophy with thickened LV walls and a small LV cavity. Poor LV wall compliance is also typically present in restrictive cardiomyopathy, severe aortic stenosis, and hypertrophic cardiomyopathy.

(Choice D) Increased pulmonary vascular resistance can cause right heart failure due to pulmonary hypertension. There is initially right ventricular wall thickening eventually leading to right ventricular cavity enlargement as contractile function begins to fail. This patient's right ventricular wall thickness and cavity size appear normal.

Educational objective:
Dilation of the left ventricular cavity commonly occurs in response to systolic dysfunction (eg, ischemic heart disease, dilated cardiomyopathy) or certain types of valvular disease (ie, aortic regurgitation, mitral regurgitation). Chronic volume overload causes progressive eccentric hypertrophy that eventually leads to reduced ventricular contractility and decompensated heart failure.

Pathology	Cardiovascular System	Dilated cardiomyopathy
Subject	System	Topic

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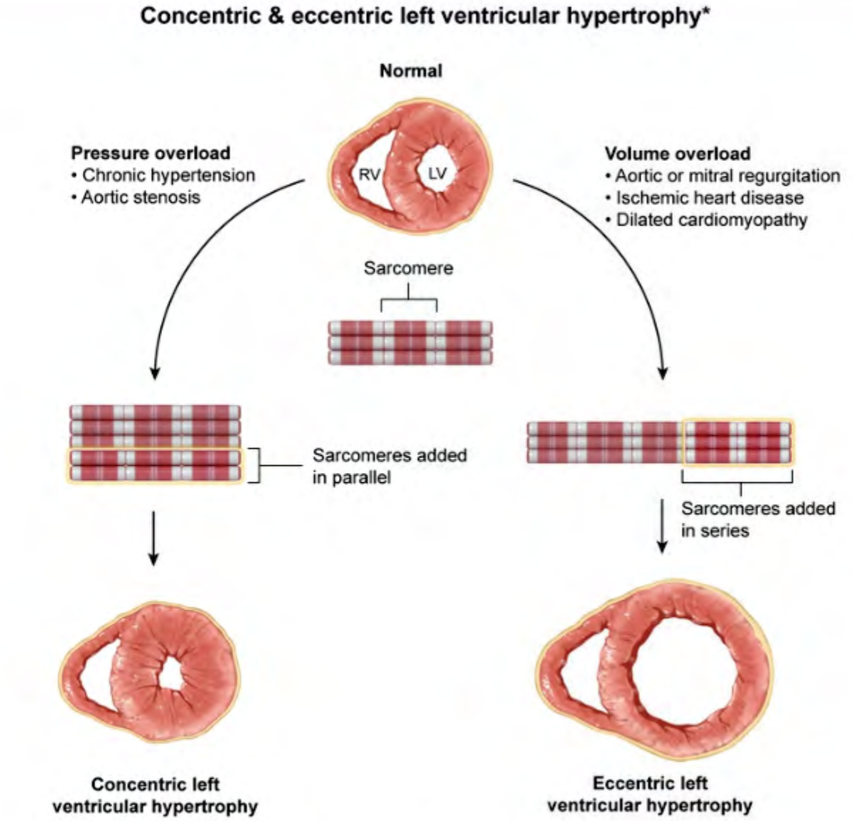

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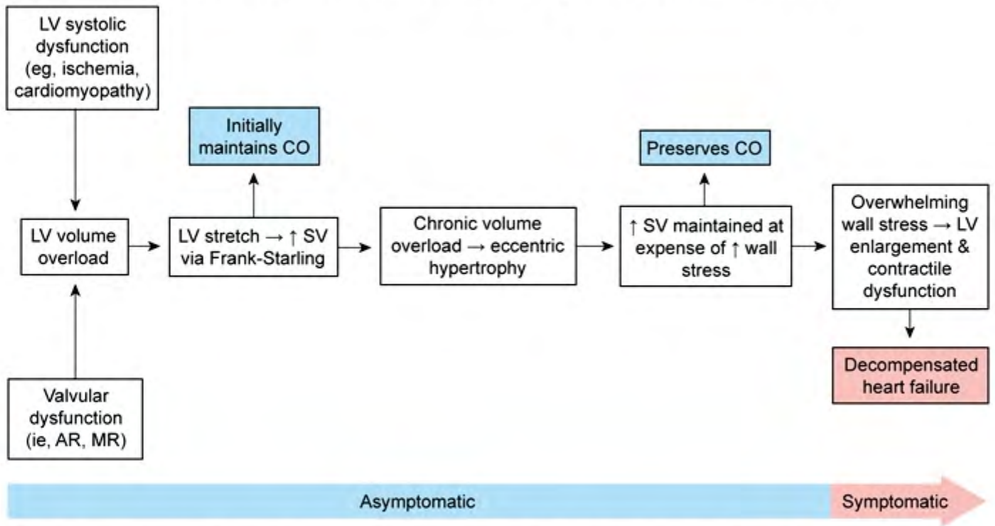
(Choice D) Increased pulmonary vascular resistance can cause right heart failure due to pulmonary hypertension. There is initially right ventricular wall thickening eventually leading to right ventricular cavity

Exhibit Display

Eccentric left ventricular enlargement & heart failure



AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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A 21-year-old man comes to the emergency department following an episode of syncope. The syncopal episode was not provoked by any activity or circumstance, nor was it preceded by lightheadedness. The patient has no significant past medical history and he is not taking any medications. An ECG obtained in the ER reveals QT-interval prolongation but is otherwise unremarkable. Assuming this is an inherited condition, the relevant mutation most likely affects which of the following structures?

- A. Cardiac cell cytoskeleton proteins
- B. Cardiac cell sarcomere proteins
- C. Mitochondrial enzymes of oxidative phosphorylation
- D. Calcium-binding sarcoplasmic reticulum protein
- E. Membrane potassium channel proteins

Submit

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- A. Cardiac cell cytoskeleton proteins (4%)
- ✗ B. Cardiac cell sarcomere proteins (13%)
- C. Mitochondrial enzymes of oxidative phosphorylation (3%)
- D. Calcium-binding sarcoplasmic reticulum protein (12%)
- ✓ E. Membrane potassium channel proteins (66%)

Incorrect
Correct answer
E

66%
Answered correctly

03 secs
Time Spent

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Explanation

This patient's sudden-onset syncopal episode suggests a sudden cardiac arrhythmia. **QT prolongation** in an otherwise healthy young individual is usually congenital. The mutation most likely to cause QT-interval prolongation can be determined based on an understanding of cardiac electrophysiology. On an ECG tracing, the QT interval begins at the start of the QRS complex and ends at the end of the T wave. Therefore, the QT-interval reflects the **cardiac myocyte action potential** duration, which is determined in part by **potassium (K^+) currents** through channel proteins. The mutations listed in the other answer choices would be less likely to directly affect the cardiac cell action potential duration.

There are two important congenital syndromes that cause QT prolongation:

- Jervell and Lange-Nielsen syndrome (autosomal recessive, with neurosensory deafness)

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- Jervell and Lange-Nielsen syndrome (autosomal recessive, with neurosensory deafness)
- Romano-Ward syndrome (more common, autosomal dominant, no deafness)

Both may predispose to *torsades de pointes* (a ventricular tachyarrhythmia) at a young age, causing syncopal episodes and possible sudden cardiac death.

(Choices A and C) Mutations affecting cardiac cell cytoskeletal proteins or the mitochondrial enzymes of oxidative phosphorylation are thought to cause the genetic form of dilated cardiomyopathy (DCM). DCM usually presents with gradual onset of left-sided heart failure (with symptoms like dyspnea on exertion initially), as opposed to the sudden syncopal episode in a previously asymptomatic patient described above.

(Choice B) Mutations in cardiac cell sarcomere proteins (e.g. beta-myosin heavy chain) underlie hypertrophic cardiomyopathy (HCM). Although HCM may present as syncope in a previously asymptomatic young person, the syncope of HCM is typically provoked by exertion. Additionally, QT prolongation is not generally found in HCM.

(Choice D) Mutations of a calcium-binding sarcoplasmic reticulum protein might underlie some cases of arrhythmogenic right ventricular cardiomyopathy (ARVC), a progressive fibrofatty replacement of the right ventricular myocardium of uncertain pathogenesis. QT prolongation is not generally seen with ARVC.

Educational objective:
Unprovoked syncope in a previously asymptomatic young person may result from a congenital QT prolongation

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Educational objective:
Unprovoked syncope in a previously asymptomatic young person may result from a congenital QT prolongation syndrome. The two most important congenital syndromes with QT prolongation, Romano-Ward syndrome and Jervell and Lange-Nielsen syndrome, are thought to result from mutations in a K^+ channel protein that contributes to the delayed rectifier current (I_K) of the cardiac action potential.

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A 76-year-old man is brought to the emergency department of a rural hospital 30 minutes after developing severe midsternal chest pain and diaphoresis. Medical history is significant for type 2 diabetes mellitus and asymptomatic right carotid artery stenosis. Blood pressure is 120/70 mm Hg and pulse is 75/min. The lungs are clear on auscultation. ECG shows ST segment elevations >1 mm in leads I, aVL, and V5-V6. The patient receives aspirin and sublingual nitroglycerin, which improves his chest pain. Timely percutaneous coronary intervention is not available and the patient is treated with fibrinolytic therapy. Several hours later, the patient is found to be comatose. Repeat physical examination shows asymmetric pupils and an irregular breathing pattern. Which of the following is the most likely cause of this patient's current condition?

- A. Carotid artery thrombosis
- B. Dissection of the ascending aorta
- C. Interventricular septum perforation
- D. Intracerebral hemorrhage
- E. Myocardial reperfusion injury
- F. Pulmonary embolism

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A 76-year-old man is brought to the emergency department of a rural hospital 30 minutes after developing severe midsternal chest pain and diaphoresis. Medical history is significant for type 2 diabetes mellitus and asymptomatic right carotid artery stenosis. Blood pressure is 120/70 mm Hg and pulse is 75/min. The lungs are clear on auscultation. ECG shows ST segment elevations >1 mm in leads I, aVL, and V5-V6. The patient receives aspirin and sublingual nitroglycerin, which improves his chest pain. Timely percutaneous coronary intervention is not available and the patient is treated with fibrinolytic therapy. Several hours later, the patient is found to be comatose. Repeat physical examination shows asymmetric pupils and an irregular breathing pattern. Which of the following is the most likely cause of this patient's current condition?

- A. Carotid artery thrombosis
- ✗ B. Dissection of the ascending aorta
- C. Interventricular septum perforation
- ✓ D. Intracerebral hemorrhage
- E. Myocardial reperfusion injury
- F. Pulmonary embolism

Incorrect
Correct answer
D

Collecting Statistics

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Explanation

This patient with chest pain and diaphoresis has ECG changes consistent with a lateral ST elevation myocardial infarction **(STEMI)**. Emergency **reperfusion** by percutaneous coronary intervention (PCI) or fibrinolysis in STEMI patients can reduce mortality and salvage ischemic myocardium. PCI is preferred due to lower rates of hemorrhage and recurrent MI; however, it may not be available at all institutions. In such cases, early treatment with **fibrinolytics**, namely recombinant forms of tissue plasminogen activator (eg, tenecteplase, alteplase),

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Fibrinolytics bind fibrin and convert plasminogen to plasmin, which then hydrolyzes key bonds in the fibrin matrix, causing clot lysis and restoration of coronary arterial blood flow. **Intracerebral hemorrhage** (ICH) is the most devastating **adverse effect** of fibrinolytic therapy; contraindications include prior stroke (especially hemorrhagic), intracranial neoplasm/vascular malformation, recent head trauma, and severe uncontrolled hypertension. A sudden **change in neurologic status** following treatment with fibrinolytics should raise suspicion for ICH.

This patient's loss of consciousness, asymmetric pupils, and irregular breathing are likely due to hemorrhage-induced mass effect and increased intracranial pressure on the midbrain, oculomotor nerve (CN III), and brainstem respiratory center, respectively. These findings are consistent with impending brain herniation, a life-threatening emergency requiring neurosurgical intervention.

(**Choice A)** Carotid artery thrombosis can cause cerebral ischemia resulting in neurologic deficits. However, focal deficits (eg, hemiparesis) rather than global deficits (eg, comatose state) are typically present. In addition, a hemorrhagic complication is more likely than a thrombotic event following fibrinolytic administration.

(Choice B) Aortic dissection can present with chest pain and focal neurologic deficits (due to extension of dissection into the carotid/intercostal arteries). However, the chest pain classically radiates to the back and other risk factors (eg, hypertension, connective tissue disorder) are often present. In addition, this patient's constellation of neurologic findings is more characteristic of brain herniation.

(Choices C and E) Interventricular septum perforation is a mechanical complication of MI that presents with heart failure (eg, cardiogenic shock). Myocardial reperfusion injury (causing cardiomyocyte dysfunction from reperfusion of ischemic tissue) is a potential complication of fibrinolysis that may manifest with heart failure or ventricular arrhythmia. Neither of these conditions would explain this patient's asymmetric pupils.

(Choice F) Pulmonary embolism (PE) presents with acute-onset dyspnea, tachycardia, and pleuritic chest pain

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(Choices C and E) Interventricular septum perforation is a mechanical complication of MI that presents with heart failure (eg, cardiogenic shock). Myocardial reperfusion injury (causing cardiomyocyte dysfunction from reperfusion of ischemic tissue) is a potential complication of fibrinolysis that may manifest with heart failure or ventricular arrhythmia. Neither of these conditions would explain this patient's asymmetric pupils.

(Choice F) Pulmonary embolism (PE) presents with acute-onset dyspnea, tachycardia, and pleuritic chest pain. Hypotension and loss of consciousness can occur with a large PE, but asymmetric pupils would not be expected.

Educational objective:
Fibrinolytic therapy for acute ST-segment elevation myocardial infarction improves clinical outcomes when percutaneous coronary intervention is unavailable. A life-threatening complication of fibrinolytic therapy is intracerebral hemorrhage, which should be suspected in a patient with acute neurologic deterioration.

References

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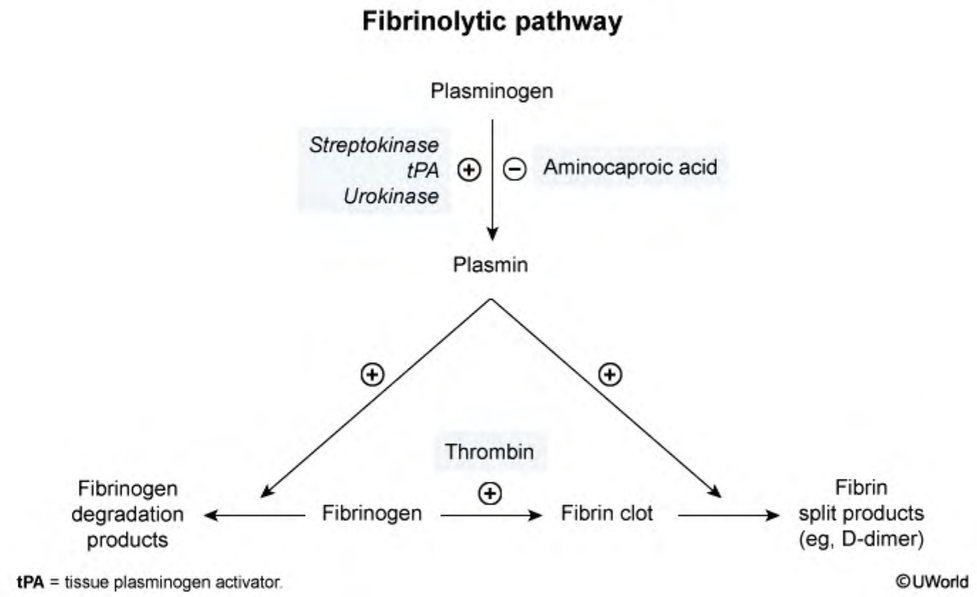

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(Choices C and E) Interventricular septum perforation is a mechanical complication of MI that presents with heart failure (eg, cardiogenic shock). Myocardial reperfusion injury (causing cardiomyocyte dysfunction from reperfusion

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(Choice E) Pulmonary embolism (PE) presents with acute-onset dyspnea, tachycardia, and pleuritic chest pain

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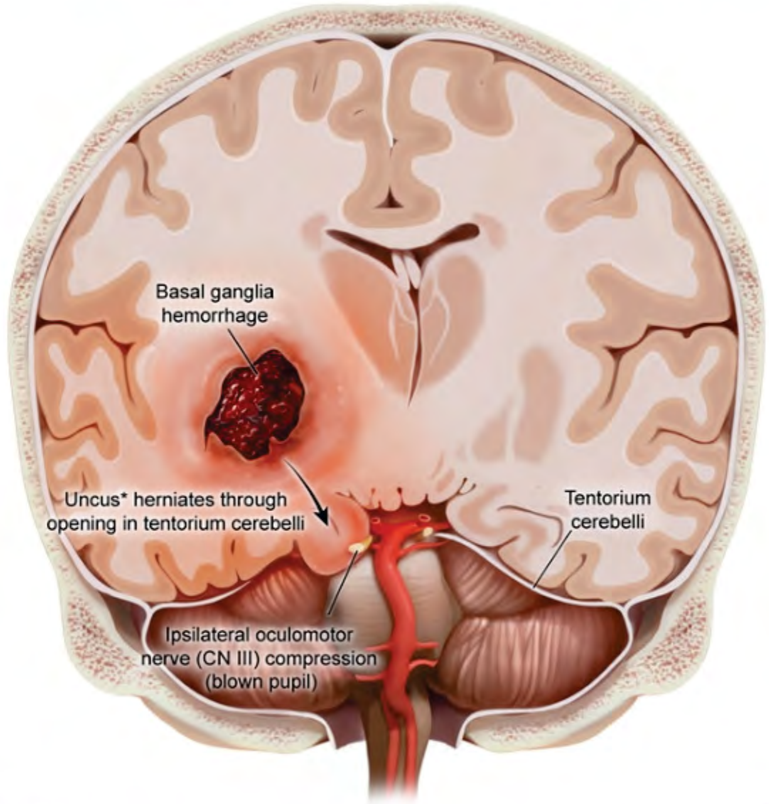
Uncal herniation due to basal ganglia hemorrhage



*Medial temporal lobe.

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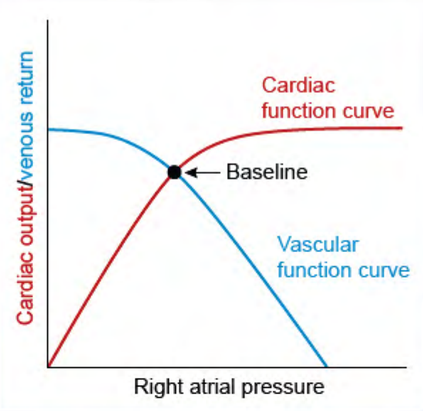
A 24-year-old healthy woman participates in a pharmacologic clinical study. Various cardiovascular parameters are measured while she is resting in an upright position. These baseline values are plotted on the cardiac and vascular function graph shown below.



She receives a moderate dose of intravenous nitroglycerin by continuous infusion, and the measurements are repeated. Which of the following changes will most likely be observed?

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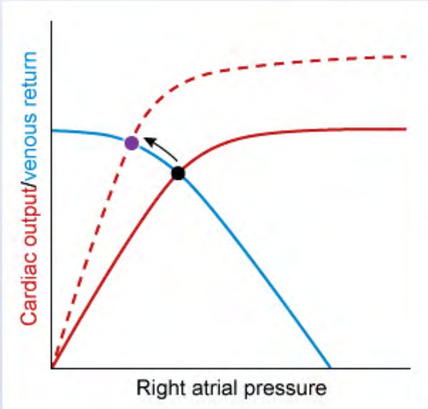

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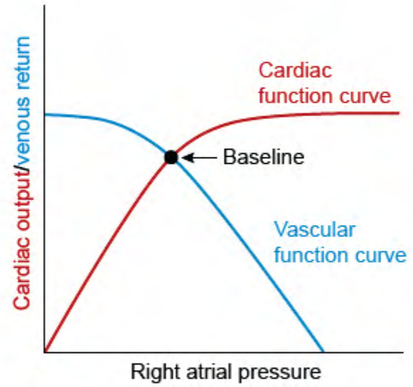

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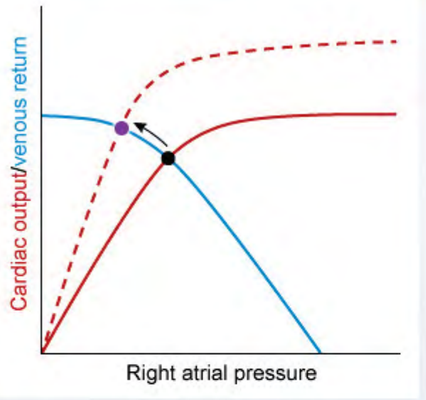

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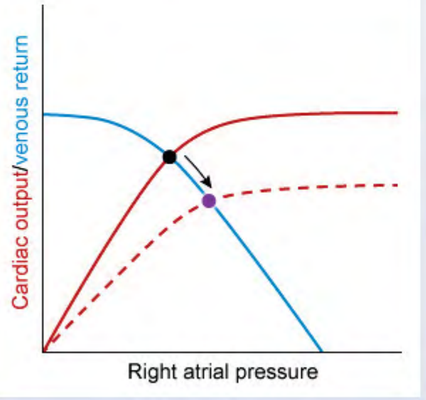

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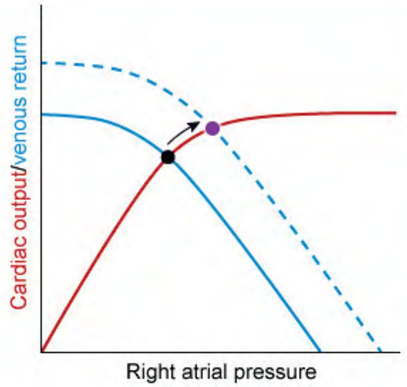

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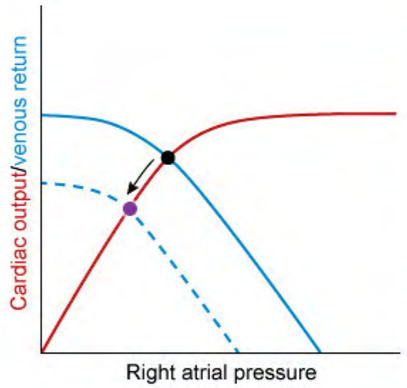

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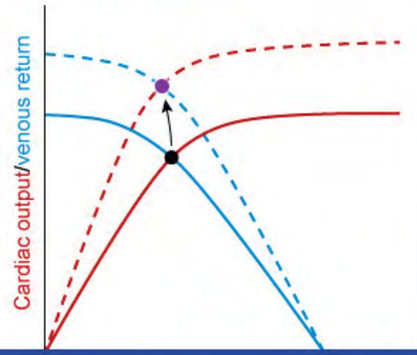

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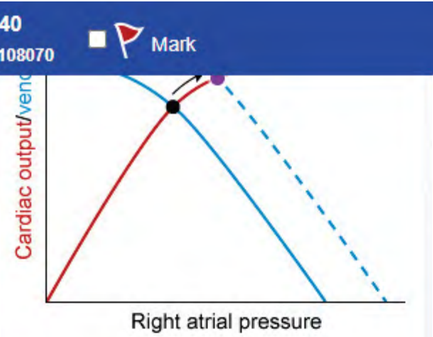

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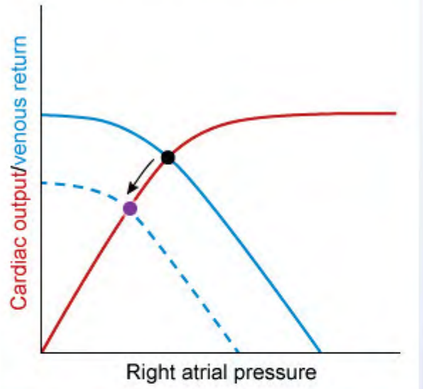

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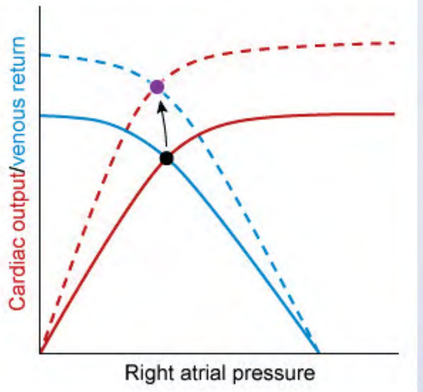

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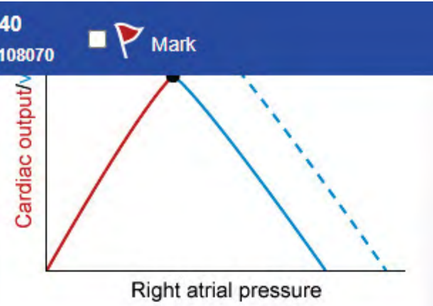

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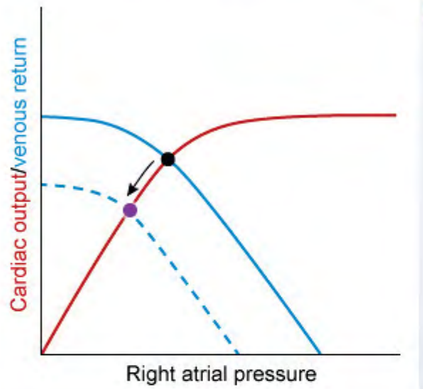

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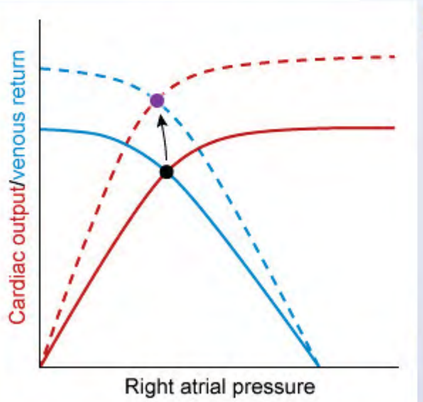

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Explanation

The cardiovascular system is a closed circuit: Blood ejected from the left ventricle (cardiac output) returns to the right atrium (venous return) to pass through the lungs and be ejected once again. **Cardiac function–vascular function curves** demonstrate the interdependent relationship between cardiac output and venous return. These curves can be described as follows:

- The **cardiac function curve** is essentially a **Frank-Starling** curve, representing constant left ventricular contractility (inotropy) along the curve. As right atrial pressure (or preload) increases, cardiac output increases due to myocardial fiber stretching and a corresponding increase in stroke volume. Changes in contractility shift the curve to the left or the right.
- The **vascular function curve** represents **venous return** to the right atrium. As right atrial pressure increases, venous return decreases as there is a reduced pressure gradient driving blood return to the right atrium. Changes in blood volume, venous capacitance, or systemic vascular resistance (SVR) shift the curve to the left or the right.
- The **intersection point** of the curves represents the **steady-state** right atrial pressure and cardiac output for a given set of cardiac and vascular parameters.

This study participant was given nitroglycerin, which primarily causes venodilation and **increased venous capacitance**. The result is additional blood pooling in the capacitance veins and decreased venous return, represented by a shift in the venous return curve to the left. The cardiac function curve is unchanged as contractility is not affected. A new steady-state point develops at a lower right atrial pressure and cardiac output.

(Choices A and B) A shift in the cardiac function curve to the left represents an increase in contractility. This change, with no change in the vascular function curve, is expected with administration of a positive inotrope that has no effect on the vasculature (eg, digoxin). A shift in the cardiac function curve to the right represents a decrease in contractility, as is expected with administration of a negative inotrope (eg, verapamil).

(Choice C) A shift in the venous return curve to the right represents an increase in venous return. This change, with no change in the cardiac function curve, is expected with the increase in blood volume that occurs with infusion of normal saline.

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(Choice C) A shift in the venous return curve to the right represents an increase in venous return. This change, with no change in the cardiac function curve, is expected with the increase in blood volume that occurs with infusion of normal saline.

(Choice E) A leftward shift in the cardiac function curve combined with a rightward shift in the vascular function curve can be seen with the increase in contractility and decrease in SVR that occur with exercise.

Educational objective:
Cardiac function–vascular function curves demonstrate the interdependent relationship between cardiac output and venous return. Nitroglycerin is a venodilator that increases venous capacitance to reduce venous return and shift the vascular function curve to the left.

References

- Venous return and mean systemic filling pressure: physiology and clinical applications.

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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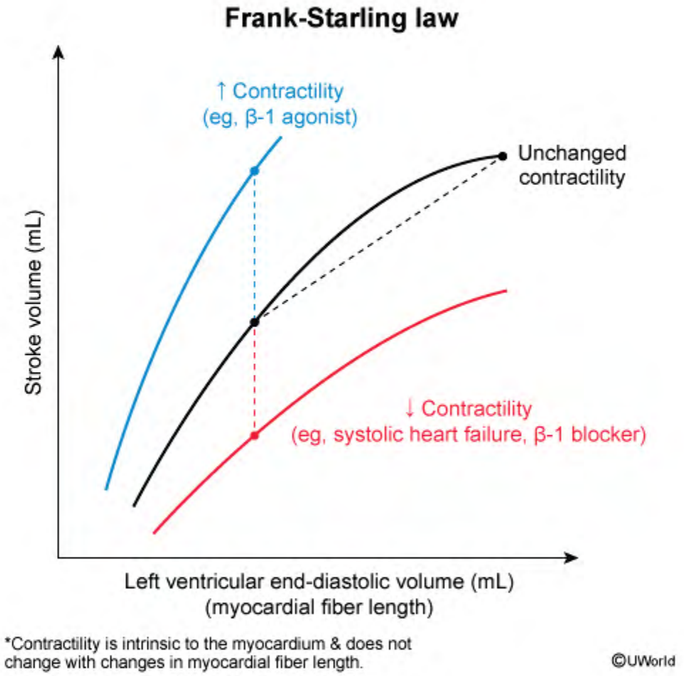

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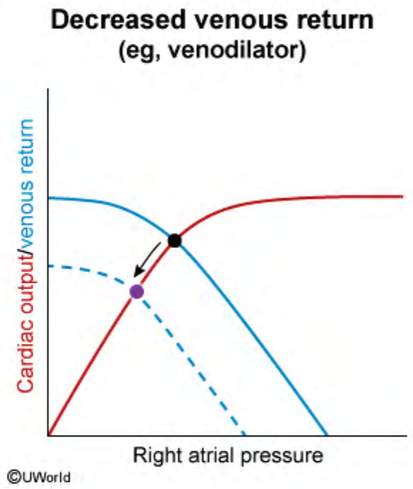

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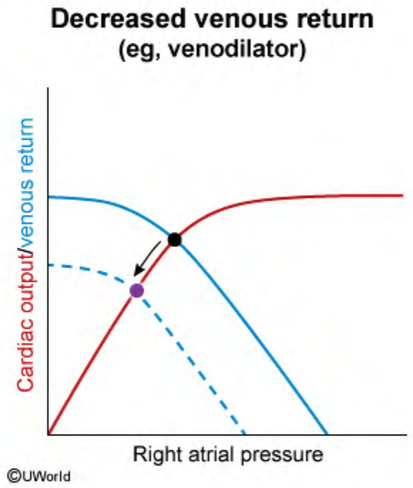

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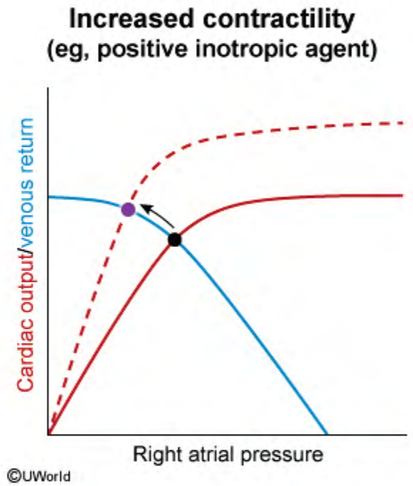

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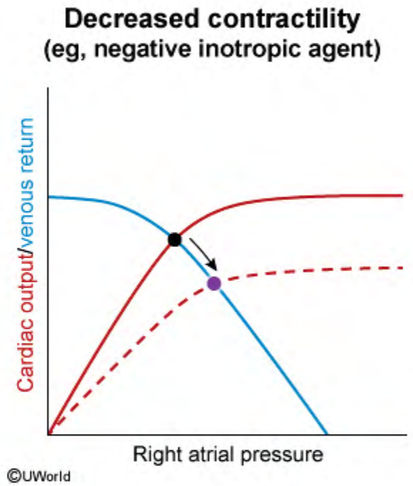

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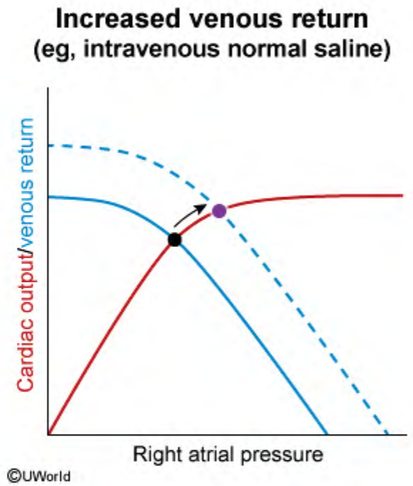

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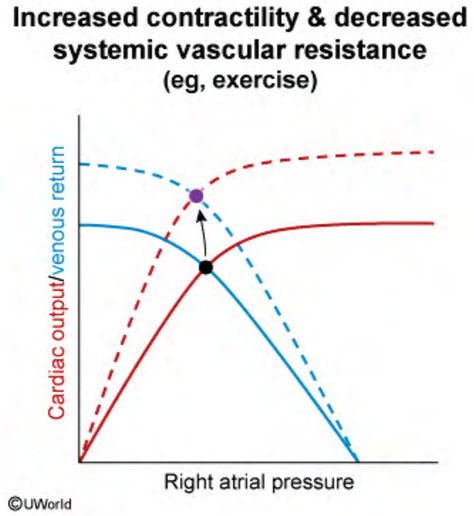

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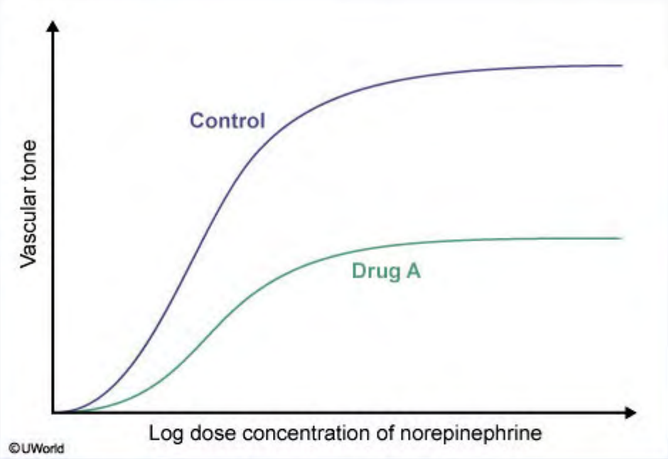
In an experiment investigating vasoconstriction of the arterial wall, two samples of isolated porcine arterial vessels are studied. Vascular tone is measured in the control vessel during infusion of increasing doses of norepinephrine. The other vessel is pretreated with experimental drug A prior to infusion of norepinephrine. A graph of the study results is depicted below.



Drug A is most similar to which of the following agents?

- A. Atropine
- B. Labetalol
- C. Phenoxybenzamine
- D. Phentolamine
- E. Propranolol

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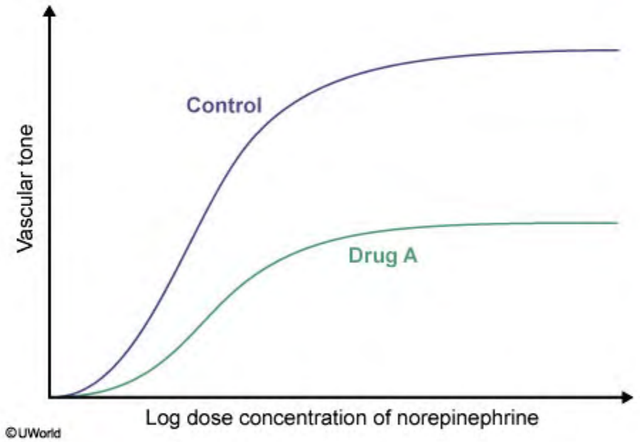

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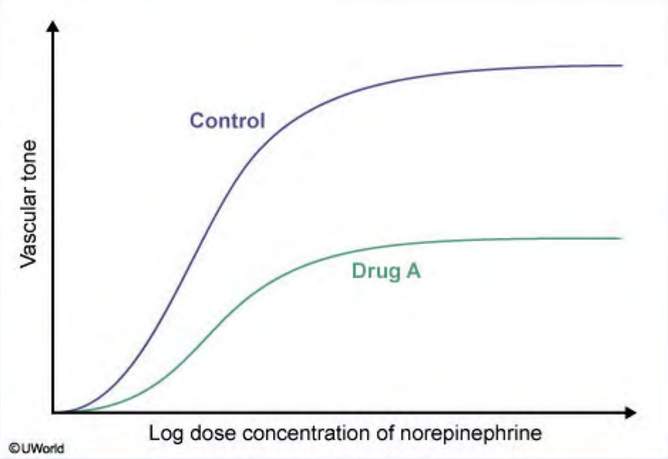
In an experiment investigating vasoconstriction of the arterial wall, two samples of isolated porcine arterial vessels are studied. Vascular tone is measured in the control vessel during infusion of increasing doses of norepinephrine. The other vessel is pretreated with experimental drug A prior to infusion of norepinephrine. A graph of the study results is depicted below.



Drug A is most similar to which of the following agents?

- A. Atropine (5%)
- ✗ B. Labetalol (11%)
- ✓ C. Phenoxybenzamine (48%)
- D. Phentolamine (24%)
- E. Propranolol (9%)

Incorrect
Correct answer
C

48%
Answered correctly

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Maximum effect

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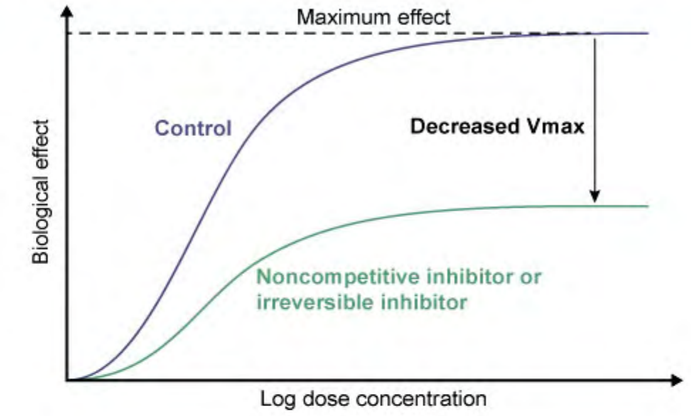

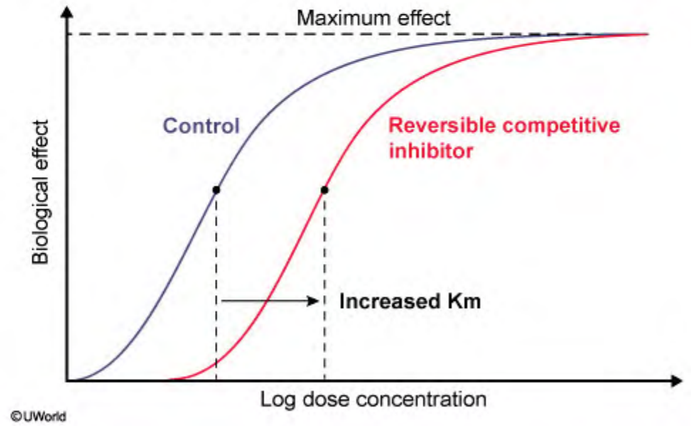

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without a significant change in the affinity (K_m) of norepinephrine for α1 receptors. This suggests that drug A is acting as either a **noncompetitive** antagonist or an **irreversible antagonist** of α1 adrenergic receptors.

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Alpha-1 adrenergic receptors mediate arterial vasoconstriction whereas β2 receptors mediate vasodilation. **Norepinephrine** is an agonist at α1 and β1 receptors but has significantly less action at β2 receptors; it is therefore a potent vasoconstrictor. This experiment is testing the efficacy of drug A at blocking the **α1-mediated vasoconstriction** caused by norepinephrine administration.

The results show that pretreatment with drug A results in **decreased maximal effect** (V_{max}) of norepinephrine without a significant change in the affinity (K_m) of norepinephrine for α1 receptors. This suggests that drug A is acting as either a **noncompetitive** antagonist or an **irreversible antagonist** of α1 adrenergic receptors. **Phenoxybenzamine** is an irreversible α1 and α2 receptor antagonist that would produce results similar to those observed. It is primarily used in the treatment of pheochromocytoma, in which a tumor of the adrenal medulla overproduces norepinephrine, causing systemic arterial vasoconstriction.

(Choice A) Atropine is a competitive muscarinic acetylcholine receptor antagonist. Pretreatment with atropine would not alter norepinephrine's effects on vascular tone.

(Choices B and D) Labetalol is a reversible, competitive antagonist of α1 and β adrenergic receptors with partial β2 agonist activity that is used to treat hypertension. Phentolamine is a reversible, competitive α-adrenergic antagonist used in the management of catecholamine-induced hypertensive crises (eg, pheochromocytoma, monoamine oxidase inhibitor toxicity, cocaine intoxication). High doses of norepinephrine can overcome the α-adrenergic inhibition of these drugs.

(Choice E) Propranolol is a nonspecific β-adrenergic antagonist. Pretreatment with a β antagonist would potentiate the vasoconstrictive effects of norepinephrine due to unopposed α-adrenergic stimulation.

Educational objective:
Phenoxybenzamine is an irreversible α1 and α2 adrenergic antagonist that effectively reduces the arterial vasoconstriction induced by norepinephrine. Because phenoxybenzamine is an irreversible antagonist, even very high concentrations of norepinephrine, such as those seen in pheochromocytoma, cannot overcome its effects.

Pharmacology	Cardiovascular System	Dose response curves
Subject	System	Topic

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A 54-year-old man comes to the office for evaluation of hypertension. He was diagnosed with hypertension 2 years ago but has had inconsistent follow-up and has not seen a physician in the last 6 months. The patient is not currently taking any medications but feels well and has good exercise tolerance. He has a family history of hypertension and ischemic stroke. His BMI is 26 kg/m^2. Physical examination is unremarkable. Serum creatinine level is normal. The patient is started on valsartan and hydrochlorothiazide, and the following values were measured at baseline (before starting treatment) and after 3 weeks of therapy:

	Blood pressure	Plasma renin activity, morning
Baseline	165/95 mm Hg	2.5 ng/mL per hour (normal: 1-4 ng/mL per hour)
After 3 weeks	162/96 mm Hg	2.4 ng/mL per hour

Which of the following best explains the observed findings in this patient?

- A. Low sodium diet
- B. Medication noncompliance
- C. Obstructive sleep apnea
- D. Primary hyperaldosteronism
- E. Renal artery stenosis

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	Blood pressure	Plasma renin activity, morning
Baseline	165/95 mm Hg	2.5 ng/mL per hour (normal: 1-4 ng/mL per hour)
After 3 weeks	162/96 mm Hg	2.4 ng/mL per hour

Which of the following best explains the observed findings in this patient?

- ✗ A. Low sodium diet (1%)
- ✓ B. Medication noncompliance (67%)
- C. Obstructive sleep apnea (1%)
- D. Primary hyperaldosteronism (10%)
- E. Renal artery stenosis (19%)

Incorrect
Correct answer
B

67%
Answered correctly

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Explanation

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Explanation

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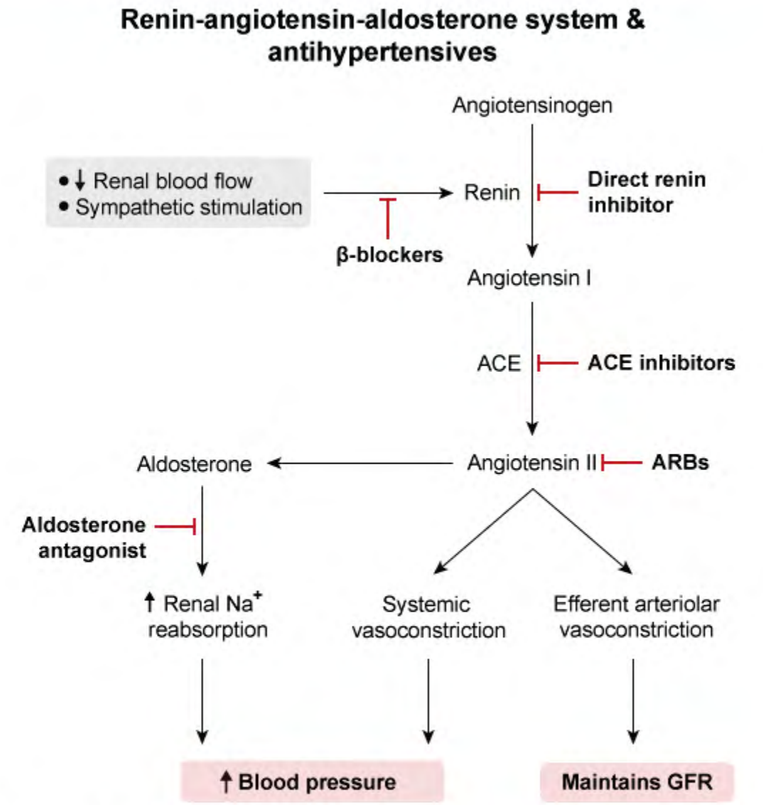

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ACE inhibitors and angiotensin II receptor antagonists (eg, valsartan) block the RAAS pathway, causing a

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Renin converts angiotensinogen to angiotensin I, which is then converted in the lung to angiotensin II, a potent vasoconstrictor that stimulates aldosterone secretion. **Plasma renin activity** (PRA) is measured as the amount of angiotensin I generated per unit of time and is a useful tool for assessing the function of the renin-angiotensin-aldosterone (RAAS) axis.

ACE inhibitors and angiotensin II receptor antagonists (eg, valsartan) block the RAAS pathway, causing a feedback increase in serum renin levels. **Diuretic-induced hypovolemia** leads to decreased renal blood flow, which also **increases renin** levels and activates RAAS. By contrast, drugs that block sympathetic stimulation (eg, beta blockers and clonidine) reduce renin levels. Because this patient's PRA did not increase after being prescribed valsartan and hydrochlorothiazide, this suggests likely medication noncompliance.

(Choice A) Sodium intake increases intravascular volume, which suppresses renin production by inducing pressure natriuresis and increased distal tubule sodium delivery. In contrast, a low sodium diet usually leads to increased serum renin and aldosterone levels. However, this patient's normal PRA makes a low sodium diet less likely.

(Choice C) Obstructive sleep apnea (OSA) is a secondary cause of hypertension but usually does not directly affect PRA. In addition, this patient does not have the typical findings of OSA (eg, excessive daytime sleepiness, snoring at night, obesity).

(Choice D) Primary hyperaldosteronism causes inappropriately elevated serum aldosterone, which suppresses renin production and would cause low serum renin levels.

(Choice E) Renal artery stenosis decreases the effective arterial volume to the kidney, which can lead to increased PRA, increased aldosterone, and elevated blood pressure. This patient has a normal PRA. In addition, initiation of angiotensin II receptor antagonists often cause a precipitous drop in blood pressure, along with acute kidney injury, in patients with underlying renal artery stenosis.

Educational objective:
Plasma renin activity (PRA) is a measure of the amount of angiotensin I generated per unit of time. It provides a useful assessment of the renin-angiotensin-aldosterone axis. Factors that increase PRA include low sodium intake and antihypertensive medications such as diuretics (eg, hydrochlorothiazide), ACE inhibitors, and angiotensin II receptor blockers (eg, valsartan).

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A 63-year-old man comes to the office for a follow-up visit. Two months ago, he was hospitalized for chest pain and was found to have a blockage in the left anterior descending artery. He had percutaneous coronary intervention with placement of a drug-eluting stent, and was discharged on appropriate medical therapy. His other medical problems include hypertension, type 2 diabetes mellitus, and degenerative joint disease. The patient has been taking all his medications as prescribed. He has no chest pain but does have diffuse muscle aches and cramps, especially after exercise. Physical examination is unremarkable. Laboratory testing reveals elevated serum creatine kinase. The medication most likely responsible for this patient's current findings also causes which of the following effects?

- A. Decreased gastric mucosal prostaglandin synthesis
- B. Elevated plasma bradykinin level
- C. Impaired potassium entry into cells
- D. Increased hepatocyte LDL receptor recycling
- E. Increased renal calcium reabsorption
- F. Inhibition of hepatic gluconeogenesis

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A 63-year-old man comes to the office for a follow-up visit. Two months ago, he was hospitalized for chest pain and was found to have a blockage in the left anterior descending artery. He had percutaneous coronary intervention with placement of a drug-eluting stent, and was discharged on appropriate medical therapy. His other medical problems include hypertension, type 2 diabetes mellitus, and degenerative joint disease. The patient has been taking all his medications as prescribed. He has no chest pain but does have diffuse muscle aches and cramps, especially after exercise. Physical examination is unremarkable. Laboratory testing reveals elevated serum creatine kinase. The medication most likely responsible for this patient's current findings also causes which of the following effects?

- ✗ A. Decreased gastric mucosal prostaglandin synthesis (3%)
- B. Elevated plasma bradykinin level (6%)
- C. Impaired potassium entry into cells (7%)
- ✓ D. Increased hepatocyte LDL receptor recycling (64%)
- E. Increased renal calcium reabsorption (4%)
- F. Inhibition of hepatic gluconeogenesis (13%)

Incorrect
Correct answer
D

64%
Answered correctly

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Explanation

This patient's symptoms are most likely due to **statin-induced myopathy**. Statins are an effective treatment for hypercholesteremia and are now routinely prescribed for all patients with symptomatic coronary artery disease, regardless of baseline cholesterol levels. Myopathy is the most common complication of statin use, and symptoms can range from myalgia or myopathy, with or without myonecrosis (elevated serum creatine kinase), to frank rhabdomyolysis.

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Explanation

This patient's symptoms are most likely due to **statin-induced myopathy**. Statins are an effective treatment for hypercholesteremia and are now routinely prescribed for all patients with symptomatic coronary artery disease, regardless of baseline cholesterol levels. Myopathy is the most common complication of statin use, and symptoms can range from myalgia or myopathy, with or without myonecrosis (elevated serum creatine kinase), to frank rhabdomyolysis.

Statins act through competitive **inhibition** of **HMG-CoA reductase**, preventing conversion of HMG-CoA to mevalonic acid (the rate-limiting step in cholesterol biosynthesis). Decreased liver cholesterol synthesis leads to increased hepatic clearance of LDL from the circulation by LDL receptors. After mediating endocytosis of LDL particles, the LDL receptors are returned to the cell surface for reuse (receptor recycling); LDL is digested and used for metabolic purposes. This **increase in LDL receptor recycling** allows intrahepatic cholesterol levels to remain at normal levels while blood levels are kept low.

(Choice A) Nonsteroidal anti-inflammatory drugs inhibit cyclooxygenase (prostaglandin synthase), leading to decreased synthesis of gastric mucosal prostaglandin.

(Choice B) Angiotensin-converting enzyme (ACE) inhibitors inhibit the conversion of angiotensin I to angiotensin II. ACE also acts as a kininase, and ACE inhibition also leads to increased plasma bradykinin levels.

(Choice C) Stimulation of beta-2 receptors by catecholamines increases extracellular potassium movement into cells. This effect is blocked by beta-blockers (primarily non-selective agents such as propranolol or labetalol), which impair potassium entry into cells and cause minor elevations in plasma potassium.

(Choice E) Thiazide diuretics increase calcium reabsorption in the distal tubule of the nephron and can lead to hypercalcemia. In contrast, decreased calcium excretion is useful in patients with renal stones due to hypercalciuria.

(Choice F) Metformin inhibits hepatic gluconeogenesis via inhibition of the mitochondrial isoform of glycerophosphate dehydrogenase in the liver. Typical side effects of metformin include gastrointestinal upset and

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mevalonic acid (the rate-limiting step in cholesterol biosynthesis). Decreased liver cholesterol synthesis leads to increased hepatic clearance of LDL from the circulation by LDL receptors. After mediating endocytosis of LDL particles, the LDL receptors are returned to the cell surface for reuse (receptor recycling); LDL is digested and used for metabolic purposes. This **increase in LDL receptor recycling** allows intrahepatic cholesterol levels to remain at normal levels while blood levels are kept low.

(Choice A) Nonsteroidal anti-inflammatory drugs inhibit cyclooxygenase (prostaglandin synthase), leading to decreased synthesis of gastric mucosal prostaglandin.

(Choice B) Angiotensin-converting enzyme (ACE) inhibitors inhibit the conversion of angiotensin I to angiotensin II. ACE also acts as a kininase, and ACE inhibition also leads to increased plasma bradykinin levels.

(Choice C) Stimulation of beta-2 receptors by catecholamines increases extracellular potassium movement into cells. This effect is blocked by beta-blockers (primarily non-selective agents such as propranolol or labetalol), which impair potassium entry into cells and cause minor elevations in plasma potassium.

(Choice E) Thiazide diuretics increase calcium reabsorption in the distal tubule of the nephron and can lead to hypercalcemia. In contrast, decreased calcium excretion is useful in patients with renal stones due to hypercalciuria.

(Choice F) Metformin inhibits hepatic gluconeogenesis via inhibition of the mitochondrial isoform of glycerophosphate dehydrogenase in the liver. Typical side effects of metformin include gastrointestinal upset and diarrhea. Myopathy is not a common effect.

Educational objective:
Statins inhibit the intracellular rate-limiting step of cholesterol biosynthesis via competitive inhibition of HMG-CoA reductase. This leads to enhanced hepatic LDL receptor recycling and increased LDL clearance from the circulation. Statin-induced myopathy (eg, myalgia, elevated creatine kinase) is the most common complication of statin use.

References

- Inhibitors of cholesterol biosynthesis increase hepatic low-density lipoprotein receptor protein degradation.
- The LDL receptor.

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A 7-year-old girl is brought to the clinic by her parents, who are concerned about her loss of interest in playing sports. During a recent competition, she walked off the field in the middle of the game because of pain in her legs. The patient takes no medications, and vaccinations are up to date. Height is at the 2nd percentile, and weight is at the 10th percentile. Temperature is 36.7 C (98 F), blood pressure is 130/80 mm Hg, pulse is 80/min, and respirations are 16/min. On physical examination, a systolic click and 2/6 systolic ejection murmur are heard. Femoral pulses are diminished relative to brachial pulses. Back examination is significant for an asymmetric curvature of the thoracic spine. This patient's underlying condition is most likely associated with which of the following additional findings?

- A. Blue sclerae
- B. Brushfield spots
- C. Gonadal dysgenesis
- D. Neurofibromas
- E. Skin elasticity
- F. Thymic hypoplasia

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- ✗ A. Blue sclerae (12%)
- B. Brushfield spots (5%)
- ✓ C. Gonadal dysgenesis (46%)
- D. Neurofibromas (4%)
- E. Skin elasticity (22%)
- F. Thymic hypoplasia (7%)

Incorrect
Correct answer
C

46%
Answered correctly

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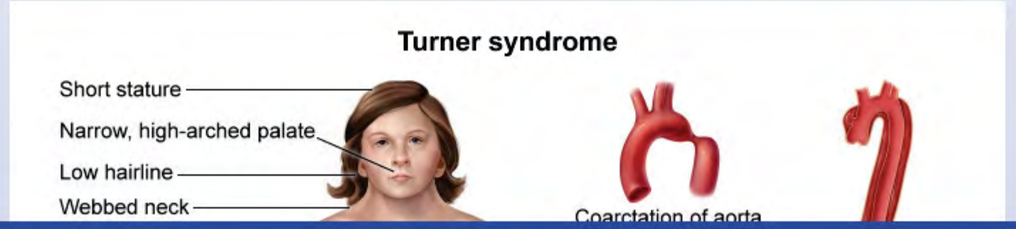
Explanation



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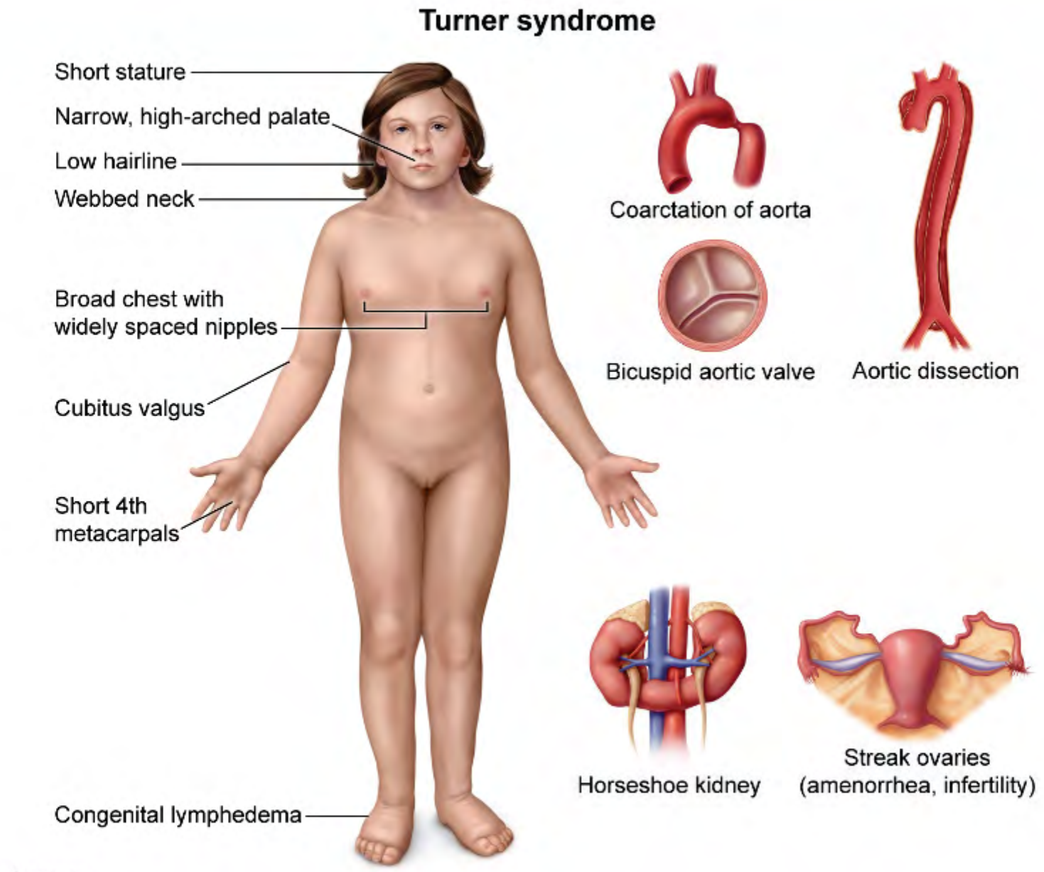

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Low hairline
Webbed neck
Coarctation of aorta

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This patient with hypertension and diminished femoral pulses most likely has **aortic coarctation**. Although severe coarctation presents in infancy with differential cyanosis or heart failure and shock, milder stenosis can present in childhood with lower extremity **claudication** (pain/cramping with exercise), blood pressure discrepancy between upper and lower extremities, and delayed/**diminished femoral pulses**. Findings on cardiac auscultation vary: patients may have no murmur, systolic murmur from flow across the narrowed aorta, continuous murmur due to collateral vessels, or murmur from coexisting cardiac anomalies. In this case, the patient's systolic click and ejection murmur are consistent with a **bicuspid aortic valve**.

Aortic coarctation is often an isolated finding, but this patient's other characteristics are suggestive of **Turner syndrome** (TS), which is caused by loss of an X chromosome. Left-sided cardiac lesions (eg, aortic coarctation, bicuspid aortic valve) are common comorbidities in TS, and almost all patients have **short stature** (height ≤2nd percentile) due to loss of a *SHOX* (short-stature homeobox) gene on chromosome X required for appropriate linear growth. Dysmorphic features and **scoliosis** are also common.

Absence of an X chromosome in TS results in **gonadal dysgenesis**, in which ovarian follicles are fibrotic and unable to produce estrogen. Primary ovarian insufficiency (low estrogen, elevated FSH/LH) leads to absent/minimal breast development, primary or secondary amenorrhea, and infertility.

(Choice A) Blue sclerae are characteristic of osteogenesis imperfecta, a collagen disorder that causes spontaneous fractures. Patients can have short stature and scoliosis, but aortic coarctation is not typical.

(Choice B) Brushfield spots (hypopigmented spots in the iris) are seen in Down syndrome, which is associated with endocardial cushion defects (eg, atrial septal defects, regurgitant atrioventricular valves), not aortic coarctation.

(Choice D) Patients with neurofibromatosis type I have a predilection for neurofibromas, and scoliosis is common. However, short stature and heart defects are not typical.

(Choice E) Skin elasticity and joint hypermobility are classic for Ehlers-Danlos syndrome. Risk of aortic dissection and mitral valve prolapse, not aortic coarctation, is increased.

(Choice F) Thymic hypoplasia occurs in DiGeorge syndrome, which is associated with conotruncal cardiac defects (eg, truncus arteriosus, interrupted aortic arch) but typically presents in infancy with dysmorphic facies, recurrent

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ejection murmur are consistent with a **bicuspid aortic valve**.

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(Choice F) Thymic hypoplasia occurs in DiGeorge syndrome, which is associated with conotruncal cardiac defects (eg, truncus arteriosus, interrupted aortic arch) but typically presents in infancy with dysmorphic facies, recurrent infections (T-cell deficiency), cleft palate, and hypocalcemia (hypoparathyroidism).

Educational objective:
Aortic coarctation can present with lower extremity claudication, upper and lower extremity blood pressure discrepancy, and diminished femoral pulses. It is commonly associated with Turner syndrome, a genetic condition characterized by short stature and gonadal dysgenesis.

References

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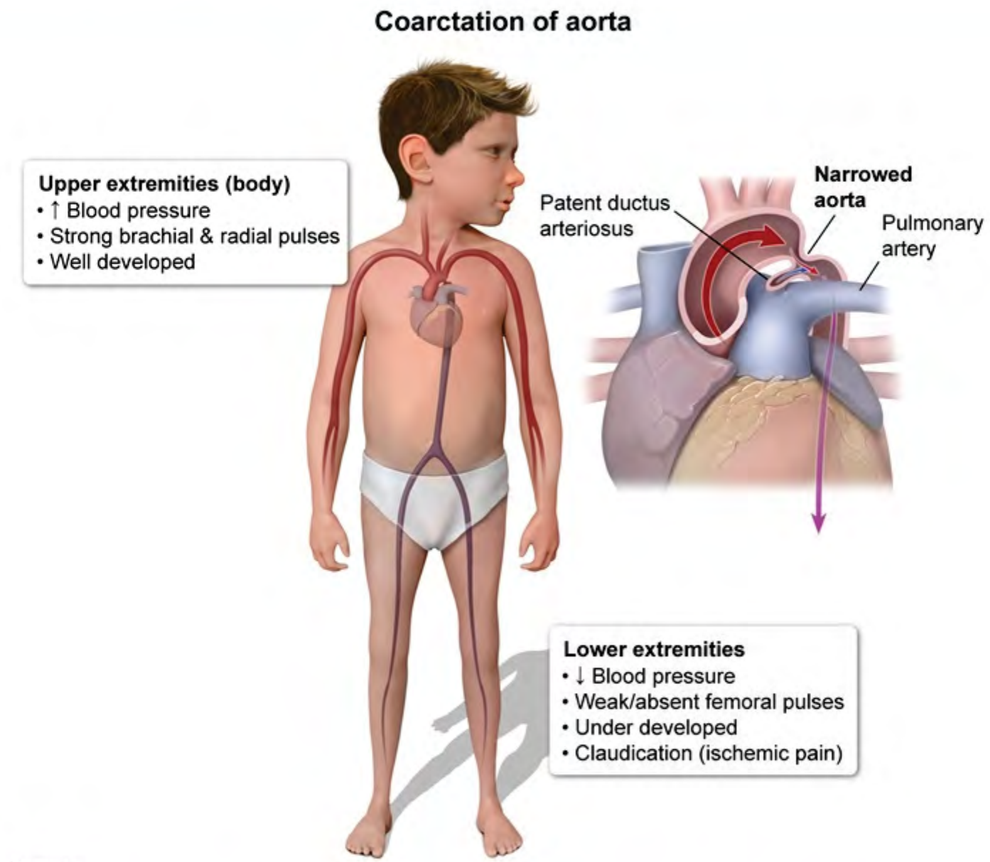

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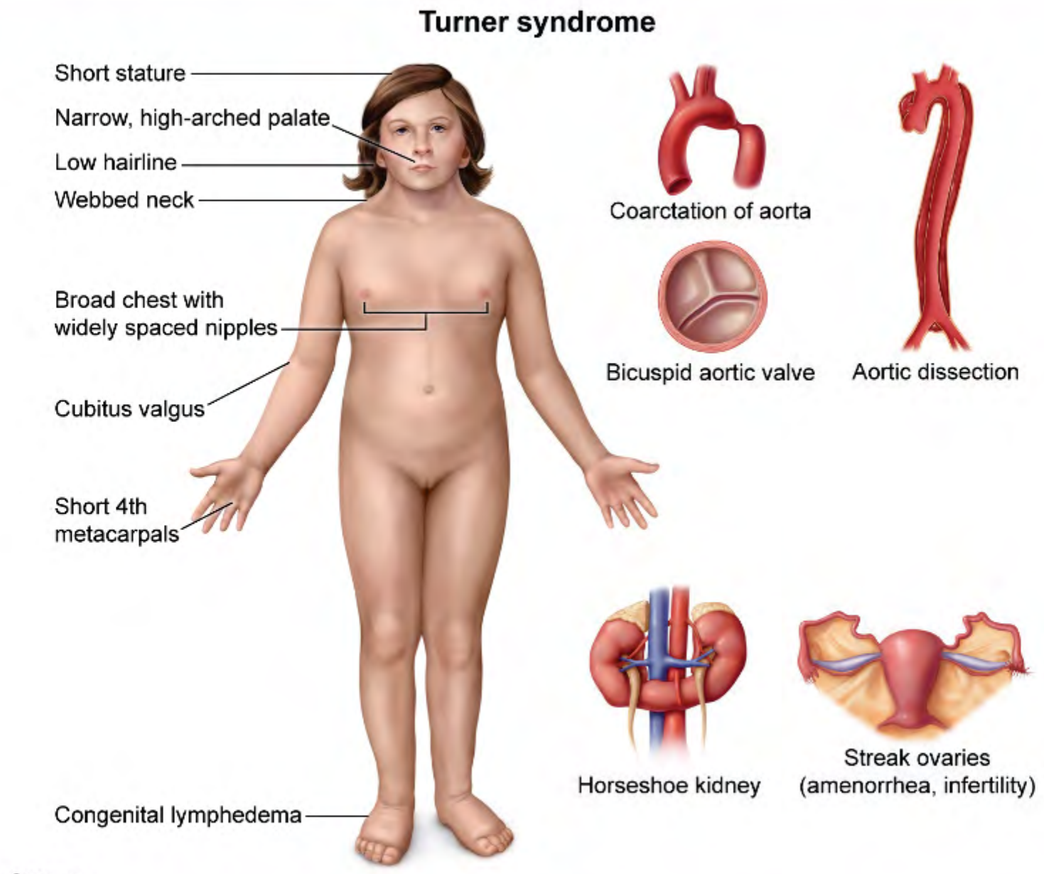

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Horseshoe kidney

Streak ovaries
(amenorrhea, infertility)

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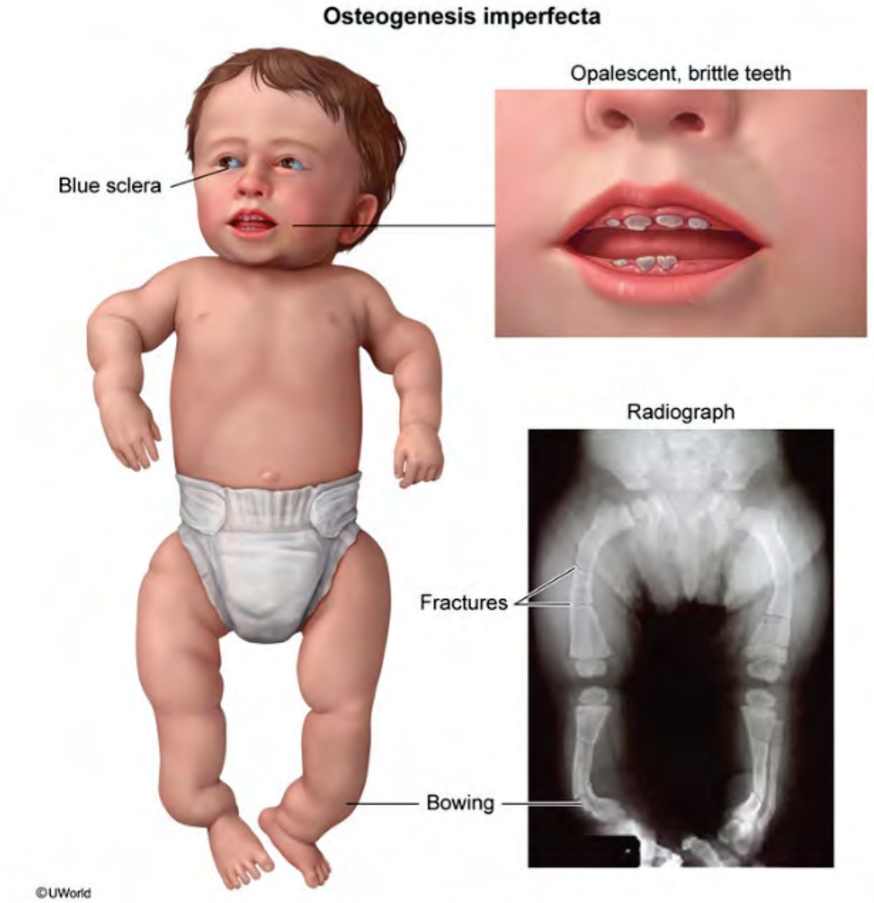

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(Choice E) Skin elasticity and joint hypermobility are classic for Ehlers-Danlos syndrome. Risk of aortic dissection

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This patient with hypertension and diminished femoral pulses most likely has **aortic coarctation**. Although severe

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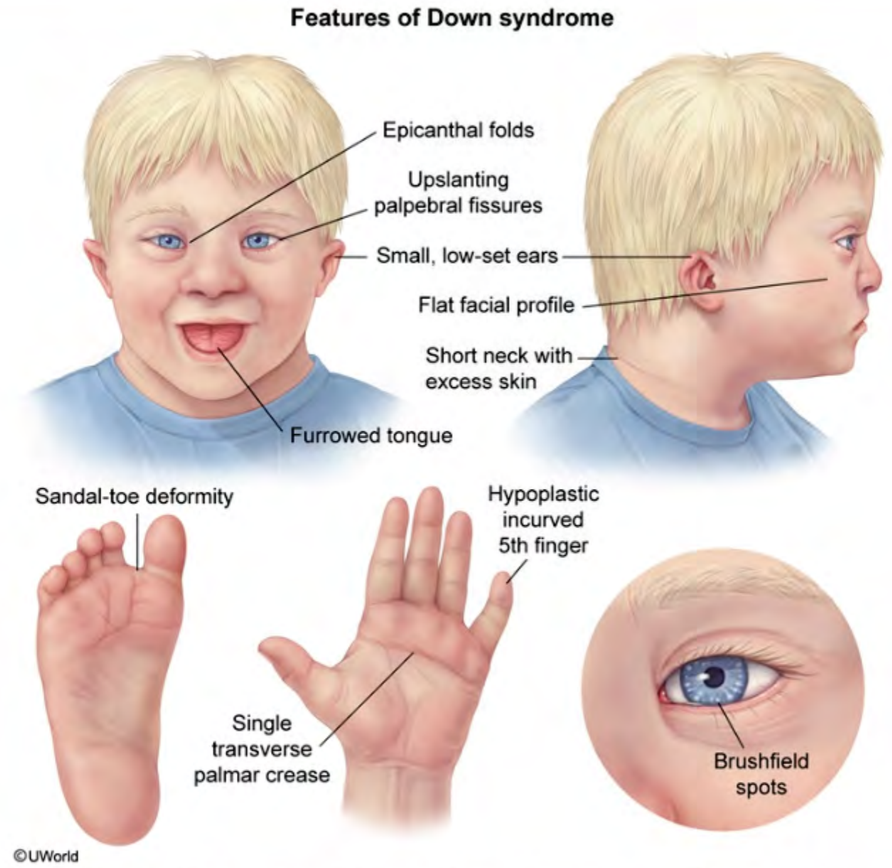

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(Choice F) Thymic hypoplasia occurs in DiGeorge syndrome, which is associated with conotruncal cardiac defects (eg, truncus arteriosus, interrupted aortic arch) but typically presents in infancy with dysmorphic facies, recurrent

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upper and lower extremities, and delayed/**diminished femoral pulses**. Findings on cardiac auscultation vary:

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Educational objective:

Aortic coarctation can present with lower extremity claudication, upper and lower extremity blood pressure

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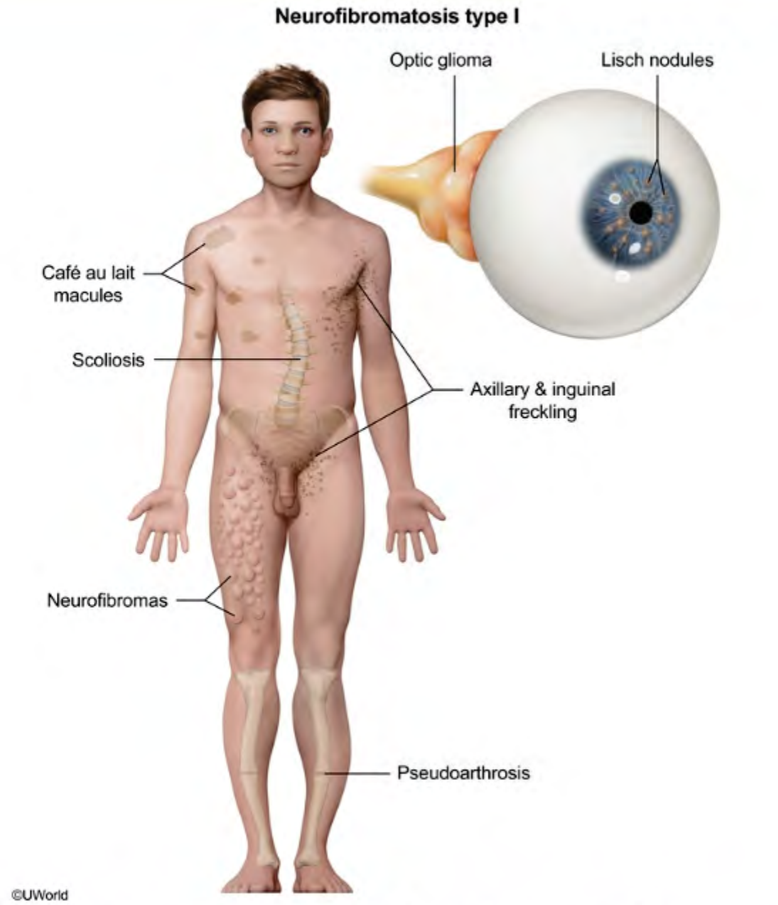

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A 58-year-old man comes to the emergency department with abrupt-onset, severe chest pain that radiates to his back. His blood pressure is 220/130 mm Hg in the left arm and 180/100 mm Hg in the right. His heart rate is 100/min. Initial laboratory studies show normal serum troponin levels. Electrocardiogram is negative for ST-segment changes. This patient's acute condition was most likely triggered by which of the following events?

- A. Intimal fatty streak formation
- B. Intimal tearing
- C. Medial calcification
- D. Medial inflammation
- E. Vasa vasorum obliteration

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- ✗ A. Intimal fatty streak formation (3%)
- ✓ B. Intimal tearing (85%)
- C. Medial calcification (2%)
- D. Medial inflammation (2%)
- E. Vasa vasorum obliteration (5%)

Incorrect
Correct answer
B

85%
Answered correctly

04 secs
Time Spent

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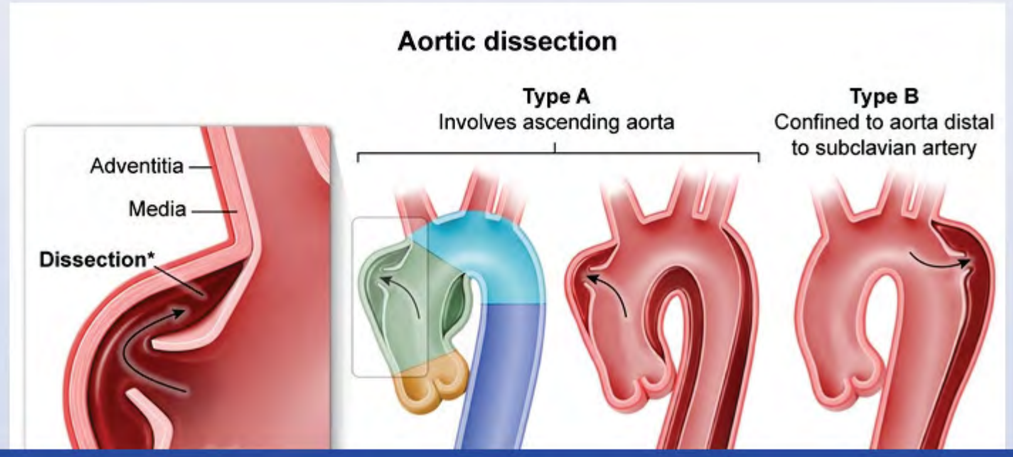
Explanation



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A 58-year-old man comes to the emergency department with abrupt-onset, severe chest pain that radiates to his back.
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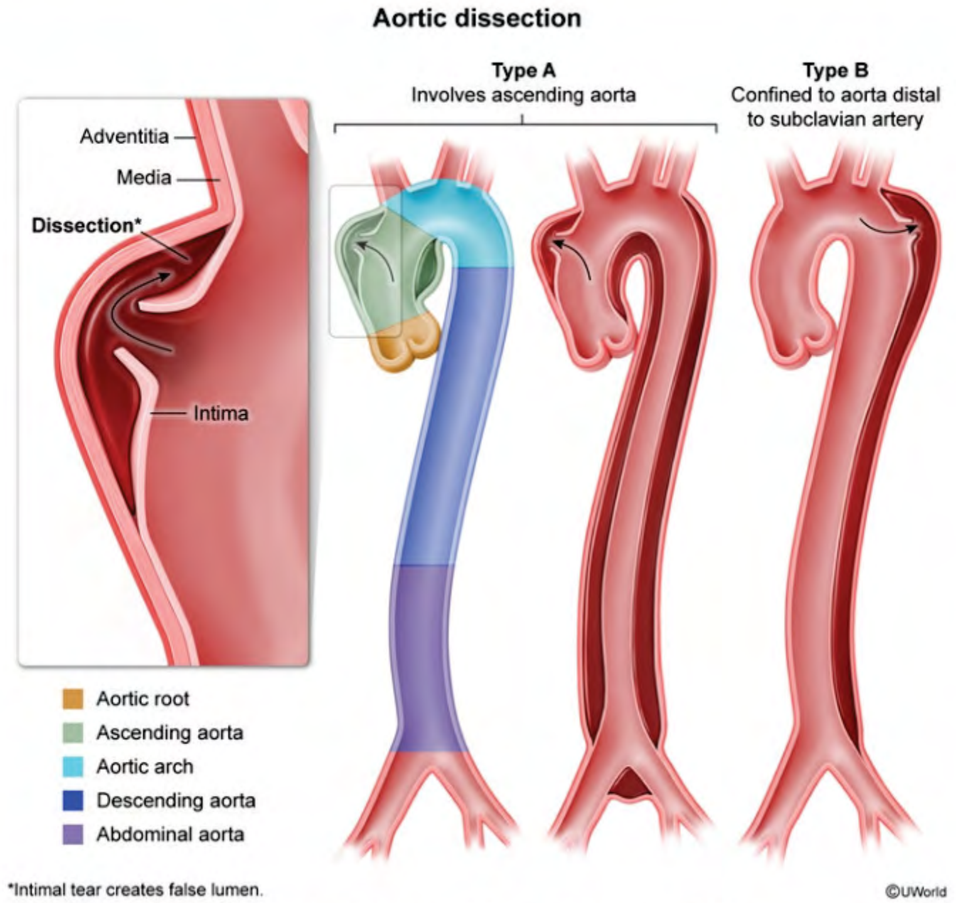

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*Intimal tear creates false lumen.

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This patient's presentation is most consistent with aortic dissection, which is characterized by severe retrosternal chest pain radiating to the mid-to-upper back that can move downward as the dissection progresses. It is initiated by a **tear in the aortic intima** that typically extends for about 1-5 cm in a transverse or oblique direction. The dissection can progress both proximally and distally as blood is forced through the tear, bisecting through the aortic media. Dissections involving any portion of the ascending aorta are classified as type A; those confined to the descending aorta are classified as type B. As the dissecting intramural hematoma spreads along the aortic wall, it can compress major arterial branches. This patient's brachial **blood pressure discrepancy** suggests compromise of the brachiocephalic trunk servicing his right arm.

Hypertension is the primary risk factor for aortic dissection. In many patients with longstanding hypertension, there is medial hypertrophy of the aortic vasa vasorum and, consequently, reduced blood flow to the aortic media. This can cause medial degeneration with a loss of smooth muscle cells, leading to aortic enlargement and increased wall stiffness. Both of these changes exacerbate aortic wall stress, which is already increased due to the hypertension itself. This synergistic increase in aortic wall stress greatly increases the risk of intimal tearing and subsequent development of aortic dissection.

(Choice A) An intimal fatty streak is the initial lesion of atherosclerosis. Some fatty streaks progress to frank atheromas, which can progressively weaken the underlying media of the aortic wall. However, atherosclerosis predisposes more to aortic aneurysm formation than to aortic dissection.

(Choice C) Monckeberg sclerosis (medial calcific sclerosis) is a form of arteriosclerosis characterized by calcific deposits in the medial layer of muscular arteries. These lesions generally occur in patients age >50 and are thought to cause isolated systolic hypertension due to arterial hardening.

(Choices D and E) Aortitis with medial inflammation can be caused by autoimmune conditions such as Takayasu and giant cell arteritis. **Tertiary syphilis** can also cause a form of aortitis characterized by **obliterative endarteritis** of the vasa vasorum. These conditions weaken the aortic wall and can predispose to aortic aneurysms and, rarely, dissections. However, hypertension is a more important risk factor for the development of intimal tearing, the primary triggering event of an aortic dissection.

Educational objective:

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descending aorta are classified as type B. As the dissecting intramural hematoma spreads along the aortic wall, it can compress major arterial branches. This patient's brachial **blood pressure discrepancy** suggests compromise of the brachiocephalic trunk servicing his right arm.

Hypertension is the primary risk factor for aortic dissection. In many patients with longstanding hypertension, there is medial hypertrophy of the aortic vasa vasorum and, consequently, reduced blood flow to the aortic media. This can cause medial degeneration with a loss of smooth muscle cells, leading to aortic enlargement and increased wall stiffness. Both of these changes exacerbate aortic wall stress, which is already increased due to the hypertension itself. This synergistic increase in aortic wall stress greatly increases the risk of intimal tearing and subsequent development of aortic dissection.

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Educational objective:
Aortic dissection classically presents with severe retrosternal pain that radiates to the back. This condition develops when overwhelming hemodynamic stress leads to tearing of the aortic intima with blood subsequently dissecting through the aortic media. The resulting intramural hematoma can extend both proximally and distally and can compress major arterial branches and impair blood flow.

References

- Pathogenesis in acute aortic syndromes: aortic dissection, intramural hematoma, and penetrating

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- Pathogenesis in acute aortic syndromes: aortic dissection, intramural hematoma, and penetrating

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Aortic calcifications

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A 78-year-old man comes to the office due to a one-month history of progressive dyspnea, generalized weakness, fatigue, and palpitations. He also reports tingling and numbness in both lower limbs. His daughter, who is visiting from another state, adds that since his wife's death a year ago, the patient has not been taking care of himself. Blood pressure is 105/50 mm Hg and pulse is 104/min. Cardiovascular examination shows a displaced apical impulse at the sixth intercostal space, a third heart sound, and high-volume, collapsing carotid pulses. Bilateral basal crackles, 2+ bilateral pedal edema, and mild hepatomegaly are also present. Neurologic examination shows decreased light touch and vibration sense in the feet, with decreased knee and ankle reflexes bilaterally. Laboratory evaluation shows normal blood counts. Deficiency of which of the following nutrients is most likely responsible for this patient's symptoms?

- A. Ascorbic acid
- B. Cobalamin
- C. Niacin
- D. Pyridoxine
- E. Retinol
- F. Riboflavin
- G. Thiamine

Submit

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- A. Ascorbic acid (1%)
- B. Cobalamin (35%)
- C. Niacin (3%)
- ✗ D. Pyridoxine (11%)
- E. Retinol (0%)
- F. Riboflavin (1%)
- ✓ G. Thiamine (45%)

Incorrect
Correct answer
G

45%
Answered correctly

27 secs
Time Spent

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Explanation

Water-soluble vitamins		
Vitamin	**Primary function**	**Deficiency**

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Explanation

Water-soluble vitamins		
Vitamin	**Primary function**	**Deficiency**
Thiamine (B_1)	Decarboxylation of α-keto acids (carbohydrate metabolism)	• Beriberi (peripheral neuropathy, heart failure) • Wernicke-Korsakoff syndrome
Riboflavin (B_2)	Mitochondrial redox reactions (FMN, FAD)	• Cheilosis, stomatitis, glossitis • Normocytic anemia
Niacin (B_3)	Redox reactions (NAD/NADP)	• Pellagra (dermatitis, dementia, diarrhea)
Pantothenic acid (B_5)	Acetylation reactions (CoA)	• Distal paresthesia (rare)
Pyridoxine (B_6)	Transamination of amino acids (amino acid synthesis)	• Peripheral neuropathy • Cheilosis, stomatitis, glossitis
Biotin (B_7)	Carboxylase reactions (CO_2 carrier)	• Dermatitis, conjunctivitis, alopecia, neurologic changes
Folate (B_9)	Hydroxymethyl/formyl carrier (purine & thymine synthesis)	• Megaloblastic anemia • Neural tube defects (fetus)
Cobalamin (B_{12})	Isomerase & methyltransferase cofactor (DNA & methionine synthesis)	• Megaloblastic anemia • Neurologic deficits
Ascorbic acid (C)	Hydroxylation of proline & lysine (collagen synthesis)	• Scurvy (perifollicular hemorrhage, gingivitis, muscle pain)

Thiamine deficiency causes infantile and adult beriberi as well as **Wernicke-Korsakoff syndrome** (confusion

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	(collagen synthesis)	gingivitis, muscle pain)

Thiamine deficiency causes infantile and adult beriberi as well as **Wernicke-Korsakoff syndrome** (confusion, ataxia, oculomotor abnormalities, and permanent memory deficits). Manifestations of infantile beriberi appear at age 2-3 months and include a fulminant cardiac syndrome with cardiomegaly, tachycardia, cyanosis, dyspnea, and vomiting. Adult **beriberi** is categorized as dry or wet depending on cardiac involvement. Dry beriberi is characterized by symmetrical **peripheral neuropathy** of the distal extremities, with resulting sensory and motor impairments. Wet beriberi includes the addition of **cardiac involvement** (cardiomyopathy, high-output congestive heart failure, peripheral edema, and tachycardia). Central nervous system involvement (ie, Wernicke-Korsakoff syndrome) occurs primarily with heavy alcohol use.

(Choice A) Ascorbic acid (vitamin C) deficiency causes scurvy (petechial hemorrhages, gingival swelling, impaired wound healing, and weakened immune response).

(Choice B) Vitamin B_{12} (cobalamin) deficiency typically presents as macrocytic anemia. Neurologic involvement can cause upper and lower motor neuron dysfunction and sensory peripheral neuropathy.

(Choice C) Niacin deficiency causes pellagra (dementia, dermatitis, and diarrhea).

(Choice D) Pyridoxine (vitamin B_6) deficiency is characterized by cheilosis, glossitis, dermatitis, and affective symptoms.

(Choice E) Vitamin A (retinol) deficiency is characterized by night blindness, xerophthalmia, and vulnerability to infection (especially measles).

(Choice F) Common manifestations of vitamin B_2 (riboflavin) deficiency include cheilosis, stomatitis, glossitis, anemia, and seborrheic dermatitis.

Educational objective:
Thiamine deficiency causes beriberi and Wernicke-Korsakoff syndrome. Dry beriberi is characterized by symmetrical peripheral neuropathy; wet beriberi includes the addition of high-output congestive heart failure.

Biochemistry	Cardiovascular System	Dilated cardiomyopathy
Subject	System	Topic

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A 24-year-old man comes to the office for an exercise evaluation. He is an avid long-distance runner. One of his team members recently died suddenly in his sleep, which made the patient worried about his health. He has no medical history, takes no medications, and does not use illicit drugs. His maternal grandfather had a heart attack at age 40 and underwent coronary artery bypass surgery. Blood pressure is 122/70 mm Hg and pulse is 49/min. Transthoracic echocardiogram shows the following:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	increased
LV wall thickness	borderline increased uniformly
LV mass	increased
LV ejection fraction	64%

The right ventricular cavity is slightly enlarged and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- A. Anabolic steroid abuse
- B. Cardiac adaptation
- C. Hypertrophic cardiomyopathy
- D. Premature coronary artery disease
- E. Subclinical viral myocarditis

Submit

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LV wall thickness	borderline increased uniformly
LV mass	increased
LV ejection fraction	64%

The right ventricular cavity is slightly enlarged and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- A. Anabolic steroid abuse (2%)
- ✓ B. Cardiac adaptation (81%)
- C. Hypertrophic cardiomyopathy (14%)
- D. Premature coronary artery disease (0%)
- E. Subclinical viral myocarditis (1%)

Correct | 81% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

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Explanation

This patient's echocardiogram findings are consistent with **athlete's heart** (ie, the physiologic cardiac adaptations that occur in highly trained athletes). **Endurance exercise** (eg, long-distance running) mostly increases the volume load on the heart (increased cardiac output) with a small component of increased pressure load (increased afterload). As a result, the left ventricle predominantly undergoes eccentric hypertrophy (**cavity enlargement** improves diastolic function) with a small amount of concentric hypertrophy (wall thickening increases systolic function). The right ventricular cavity also slightly enlarges to support an increase in cardiac output, and there is **enhanced coronary capillary development** and a **decreased resting heart rate** due to improved efficiency of cardiac pumping.

Because these **physiologic changes improve cardiac function**, athlete's heart is not associated with an increased risk of cardiovascular morbidity or mortality. However, it is imperative that the changes of athlete's heart are differentiated from similar cardiac anomalies that may reflect serious underlying pathology that could increase the risk of sudden cardiac death (eg, hypertrophic cardiomyopathy). Findings that favor the presence of hypertrophic cardiomyopathy **(Choice C)** include left ventricular (LV) hypertrophy predominantly affecting the septum, LV hypertrophy with reduced LV cavity size, impaired diastolic function, and a poorly developed coronary capillary network.

(Choice A) Anabolic steroid abuse can cause pathologic concentric LV hypertrophy, characterized by reduced diastolic function and poor coronary vascularization.

(Choice D) Severe coronary artery disease can lead to ischemic cardiomyopathy, which typically manifests as dilated cardiomyopathy with an enlarged LV cavity but thin LV walls with impaired systolic function.

(Choice E) Viral myocarditis may sometimes lead to dilated cardiomyopathy with eccentric hypertrophy and impaired LV systolic function. Subclinical disease is typically quite mild and resolves without sequelae.

Educational objective:
Athlete's heart refers to physiologic cardiac adaptations that improve cardiac function in response to high-level endurance training. There is predominant left ventricular (LV) eccentric hypertrophy with a small component of concentric hypertrophy, leading to an overall increase in LV mass, enlarged LV cavity size, slightly increased LV

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volume load on the heart (increased cardiac output) with a small component of increased pressure load (increased afterload). As a result, the left ventricle predominantly undergoes eccentric hypertrophy (**cavity enlargement** improves diastolic function) with a small amount of concentric hypertrophy (wall thickening increases systolic function). The right ventricular cavity also slightly enlarges to support an increase in cardiac output, and there is **enhanced coronary capillary development** and a **decreased resting heart rate** due to improved efficiency of cardiac pumping.

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(Choice A) Anabolic steroid abuse can cause pathologic concentric LV hypertrophy, characterized by reduced diastolic function and poor coronary vascularization.

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Educational objective:
Athlete's heart refers to physiologic cardiac adaptations that improve cardiac function in response to high-level endurance training. There is predominant left ventricular (LV) eccentric hypertrophy with a small component of concentric hypertrophy, leading to an overall increase in LV mass, enlarged LV cavity size, slightly increased LV wall thickness, and decreased resting heart rate.

References

- Athlete's heart.

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concentric hypertrophy, leading to an overall increase in LV mass, enlarged LV cavity size, slightly increased LV

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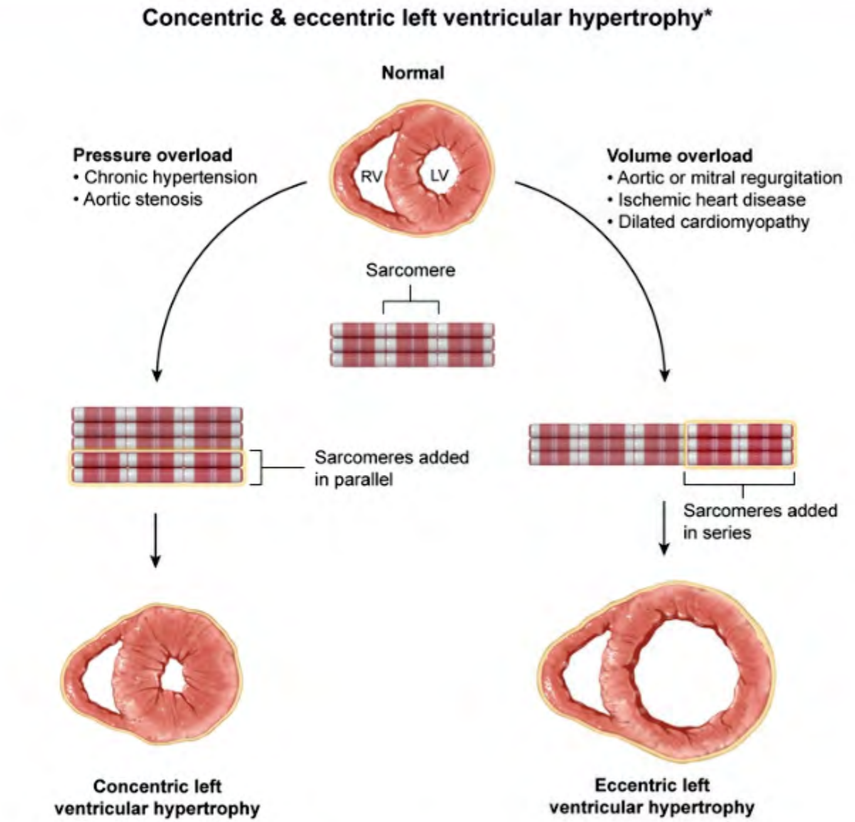

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A 14-year-old girl is brought to the office for a routine physical examination. The patient will be starting her freshman year of high school soon. She plays the clarinet in the school band but does not play any sports. The patient says that she is not sexually active and does not use tobacco, alcohol, or illicit drugs. Height is at the 5th percentile, and weight is at the 25th percentile. Her temperature is 36.7 C (98 F), blood pressure is 120/80 mm Hg, pulse is 88/min, and respirations are 16/min. Physical examination shows a short and thick neck, a broad chest, and shortened fourth metacarpals bilaterally. A murmur is heard on cardiac auscultation. Which of the following would most likely be seen in an echocardiogram?

- A. Atrial septal defect
- B. Bicuspid aortic valve
- C. Mitral stenosis
- D. Mitral valve prolapse
- E. Patent ductus arteriosus
- F. Ventricular septal defect

Submit

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- ✗ A. Atrial septal defect (5%)
- ✓ B. Bicuspid aortic valve (76%)
- C. Mitral stenosis (1%)
- D. Mitral valve prolapse (4%)
- E. Patent ductus arteriosus (7%)
- F. Ventricular septal defect (5%)

Incorrect
Correct answer
B

76%
Answered correctly

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Explanation

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E. Patent ductus arteriosus (7%)

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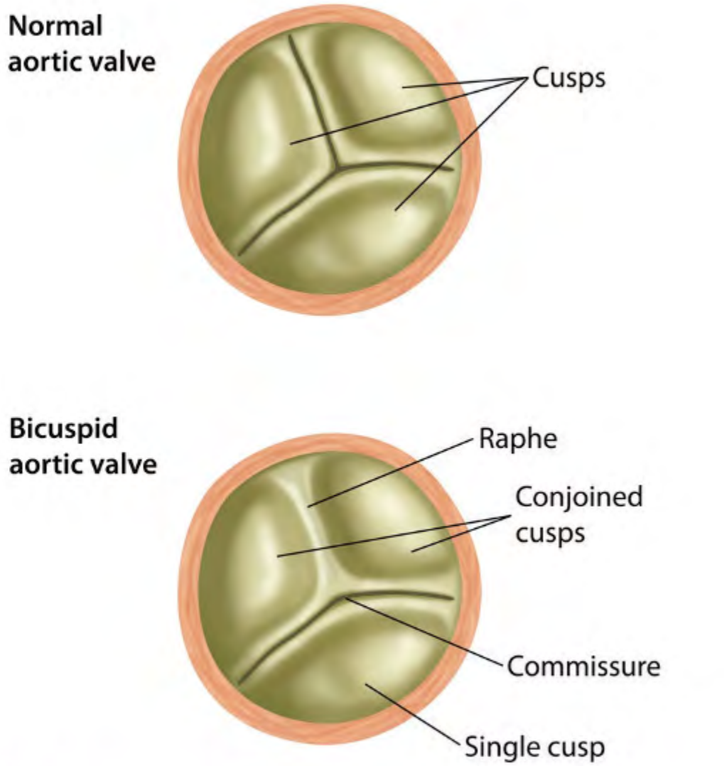
Bicuspid aortic valve disease



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Bicuspid

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This patient's short stature, short and thick neck, broad chest, and shortened fourth metacarpals are characteristic of Turner syndrome (TS). **Bicuspid aortic valve** is the most common congenital cardiac malformation in patients with TS. It is usually an isolated abnormality; however, it may occur in combination with other anomalies, particularly aortic coarctation. A nonstenotic bicuspid aortic valve manifests with an aortic ejection sound, which is an **early systolic, high-frequency click** heard over the **right second interspace**. As the valve calcifies, it may result in progressive valvular dysfunction, manifesting with aortic stenosis or regurgitation and associated murmurs. Patients with a bicuspid aortic valve are susceptible to infectious endocarditis due to abnormal leaflet structure and turbulent flow.

(Choices A and F) Complete atrioventricular canal, atrial septal defect (ASD), and ventricular septal defect (VSD) are the most common cardiac anomalies in Down syndrome. Fixed splitting of the second heart sound is characteristically heard with an ASD, whereas VSDs produce a holosystolic murmur at the left lower sternal border.

(Choice C) Mitral stenosis is seen in rheumatic heart disease. It produces a mid-diastolic, low-pitched, rumbling murmur that may begin with an opening snap.

(Choice D) A mid-systolic click, often accompanied by a late systolic murmur, is the hallmark of mitral valve prolapse (MVP). MVP is more common in connective tissue disorders such as Marfan and Ehlers-Danlos syndromes.

(Choice E) Patent ductus arteriosus (PDA) produces a continuous, machinelike murmur during both systole and diastole. PDA is common in premature infants, especially those with respiratory distress syndrome. A PDA may also be seen in an infant with coarctation of the aorta but is less likely in older patients.

Educational objective:
Turner syndrome is associated with congenital anomalies of the aorta, and the most common defect is a bicuspid aortic valve. A nonstenotic bicuspid aortic valve can manifest as an early systolic, high-frequency click over the right second interspace. Bicuspid aortic valves are at risk for stenosis, insufficiency, and infection.

References

- Turner syndrome: diagnosis and management.
- Aortic valve disease in Turner syndrome.

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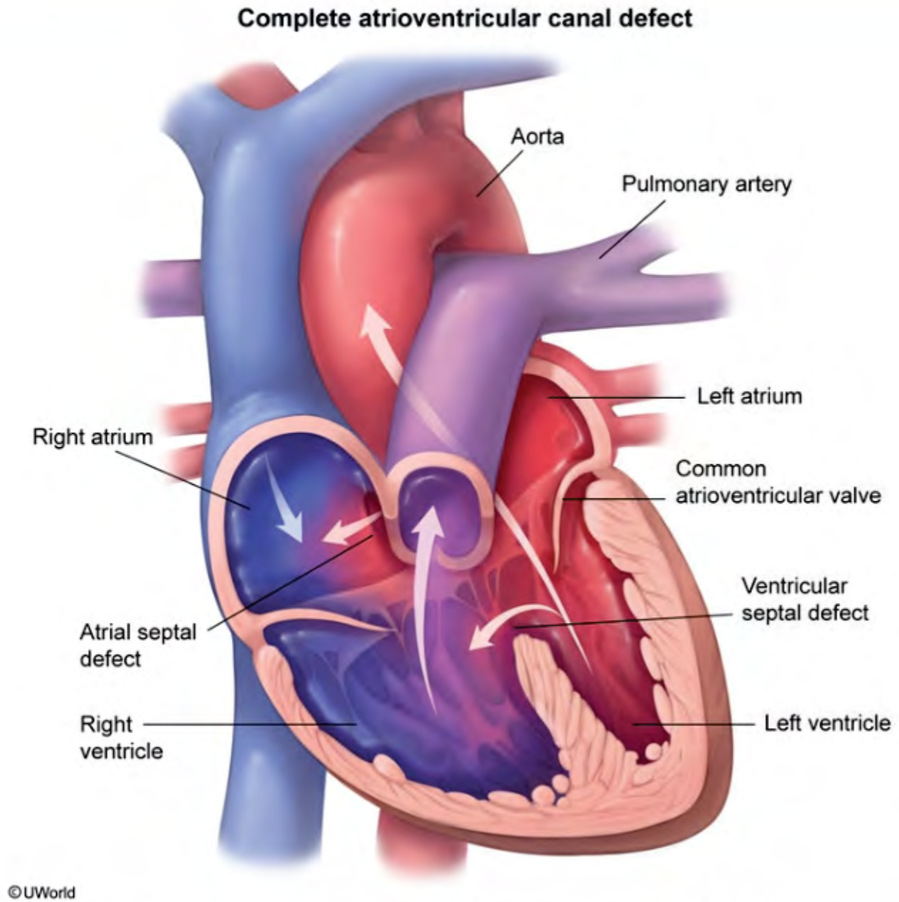

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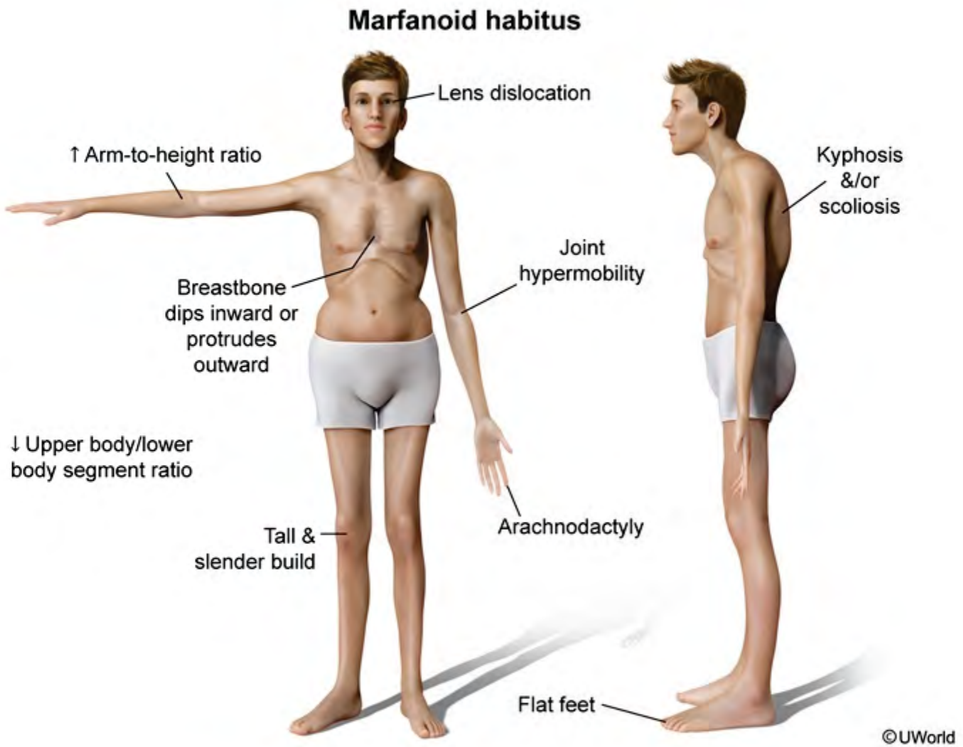

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A 43-year-old woman comes to the emergency department due to nausea and vomiting with left-sided chest pain. She states that the pain began 30 minutes ago while playing with her children. She has a history of dyslipidemia and hypertension. The patient's mother died from a myocardial infarction at age 51. Physical examination reveals mild tenderness to palpation over the left chest. An ECG and cardiac biomarkers are normal. She is scheduled for an exercise stress test. During the test, which of the following parameters is likely to be the most similar between the systemic and pulmonary circulation?

- A. Arterial oxygen content
- B. Arterial resistance
- C. Blood flow per minute
- D. Diastolic arterial pressure
- E. Driving pressure for blood flow
- F. Mean arterial pressure

Submit

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- A. Arterial oxygen content (9%)
- ✗ B. Arterial resistance (5%)
- ✓ C. Blood flow per minute (64%)
- D. Diastolic arterial pressure (6%)
- E. Driving pressure for blood flow (5%)
- F. Mean arterial pressure (8%)

Incorrect
Correct answer
C

64%
Answered correctly

03 secs
Time Spent

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Explanation

In order to maintain blood flow through the body, the blood flow (mL/min) in the **pulmonary circulation** must closely **match** the blood flow in the **systemic circulation**. This is true for conditions of both exercise and rest as the circulatory system is a continuous circuit. If the flow of blood through the pulmonary circulation is less than the flow of blood through the systemic circulation, the left ventricle would soon empty completely. Alternately, if the flow of blood is significantly greater in the pulmonary circulation than it is in the systemic circulation, the left ventricle would soon be overloaded.

The major exception to this is the bronchial circuit, which supplies oxygen and nutrients to the pulmonary

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The major exception to this is the bronchial circuit, which supplies oxygen and nutrients to the pulmonary parenchyma from the systemic circulation but drains mostly to the left atrium as opposed to the right atrium (creating a right to left shunt that acts as a partially independent circuit). However, this typically accounts for <5% of the systemic cardiac output.

(Choice A) The arterial oxygen contents of the pulmonary and systemic circulations are dramatically different due to deoxygenated blood in the pulmonary arterial circulation and oxygenated blood in the systemic arterial circulation.

(Choice B) Although the resistance of the pulmonary vasculature may be increased in some circumstances (eg, high altitudes, idiopathic pulmonary hypertension), the arterial resistance in the systemic circulation is considerably higher in all scenarios.

(Choices D, E, and F) The mean arterial pressure, diastolic arterial pressure, and driving pressure for blood flow (difference between mean arterial and venous pressures) are considerably different in the pulmonary and systemic circulations both at rest and during exercise. The normal mean systemic arterial pressure ranges between 70-100 mm Hg and the normal mean pulmonary arterial pressure is approximately 14 mm Hg, whereas venous pressures in both circuits approach 0 mm Hg. Due to the low resistance of the pulmonary circuit, only a small difference between arterial and venous pressures is needed to maintain blood flow compared to the relatively large difference needed between systemic arterial and venous pressures.

Educational objective:
The circulatory system is a continuous circuit, and therefore the volume output of the left ventricle must closely match the output of the right ventricle. This balance is necessary to maintain continuous blood flow through the

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(Choice A) The arterial oxygen contents of the pulmonary and systemic circulations are dramatically different due to deoxygenated blood in the pulmonary arterial circulation and oxygenated blood in the systemic arterial circulation.

(Choice B) Although the resistance of the pulmonary vasculature may be increased in some circumstances (eg, high altitudes, idiopathic pulmonary hypertension), the arterial resistance in the systemic circulation is considerably higher in all scenarios.

(Choices D, E, and F) The mean arterial pressure, diastolic arterial pressure, and driving pressure for blood flow (difference between mean arterial and venous pressures) are considerably different in the pulmonary and systemic circulations both at rest and during exercise. The normal mean systemic arterial pressure ranges between 70-100 mm Hg and the normal mean pulmonary arterial pressure is approximately 14 mm Hg, whereas venous pressures in both circuits approach 0 mm Hg. Due to the low resistance of the pulmonary circuit, only a small difference between arterial and venous pressures is needed to maintain blood flow compared to the relatively large difference needed between systemic arterial and venous pressures.

Educational objective:
The circulatory system is a continuous circuit, and therefore the volume output of the left ventricle must closely match the output of the right ventricle. This balance is necessary to maintain continuous blood flow through the body and exists both at rest and during exercise.

References

- Organ-specific physiological responses to acute physical exercise and long-term training in humans

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Correct answer
C

64%
Answered correctly

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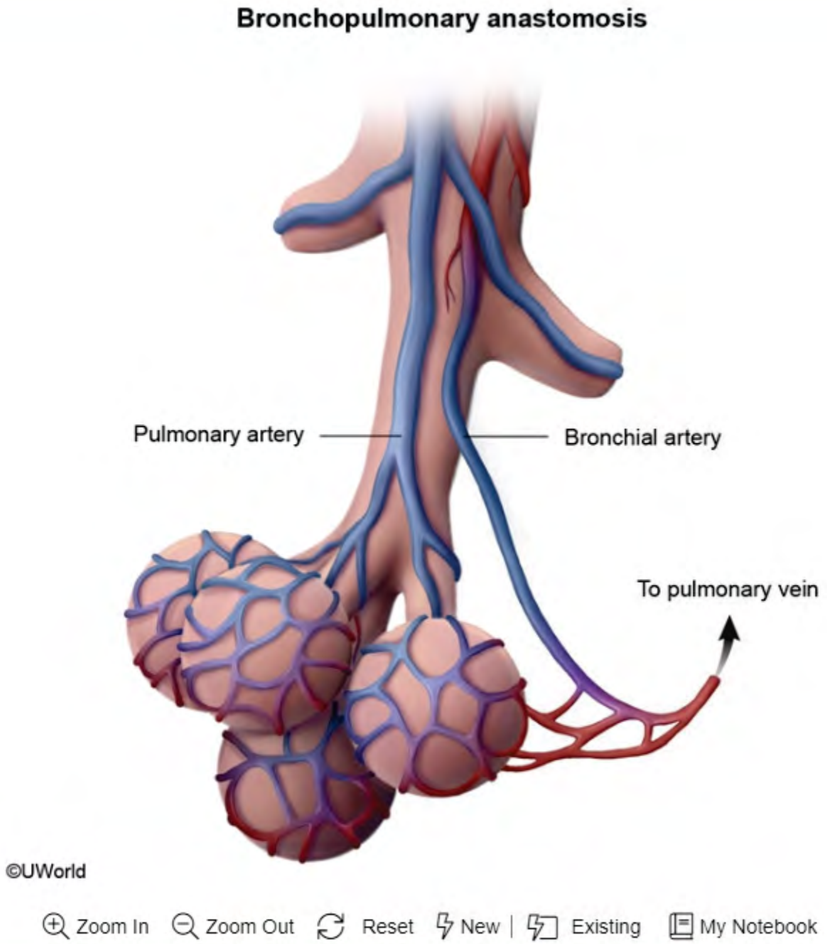

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A 55-year-old man is brought to the emergency department due to severe substernal chest pain that is accompanied by sweating and mild dyspnea. The pain started as midline chest discomfort several hours ago but then progressively worsened such that the patient could not fall asleep. He received sublingual nitroglycerin in the ambulance without significant pain relief. His medical history includes hypertension, type 2 diabetes mellitus, and hyperlipidemia. ECG demonstrates normal sinus rhythm and ST-segment elevation in leads I, aVL, and V1-V4, with deep Q wave development over the next several hours. Cardiac catheterization in this patient would most likely show which of the following?

- A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus
- B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus
- C. Ruptured atherosclerotic plaque with fully obstructive thrombus
- D. Significant coronary artery vasospasm causing flow obstruction
- E. Ulcerated atherosclerotic plaque with partially obstructive thrombus

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- A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus (1%)
- ✗ B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus (13%)
- ✓ C. Ruptured atherosclerotic plaque with fully obstructive thrombus (75%)
- D. Significant coronary artery vasospasm causing flow obstruction (5%)
- E. Ulcerated atherosclerotic plaque with partially obstructive thrombus (4%)

Incorrect
Correct answer
C

75%
Answered correctly

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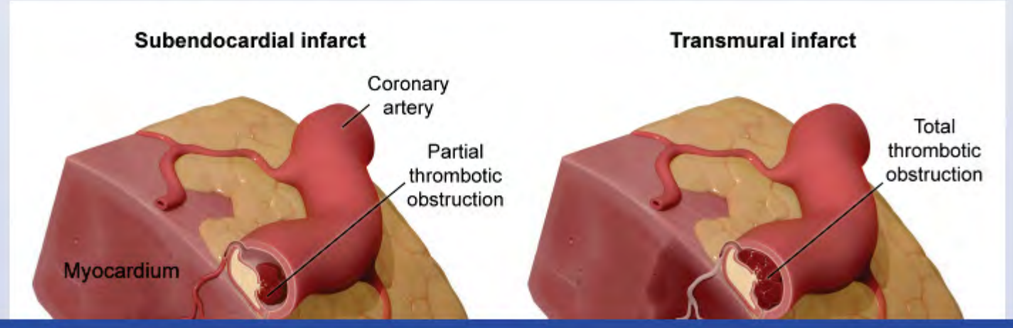
Explanation



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likely show which of the following?

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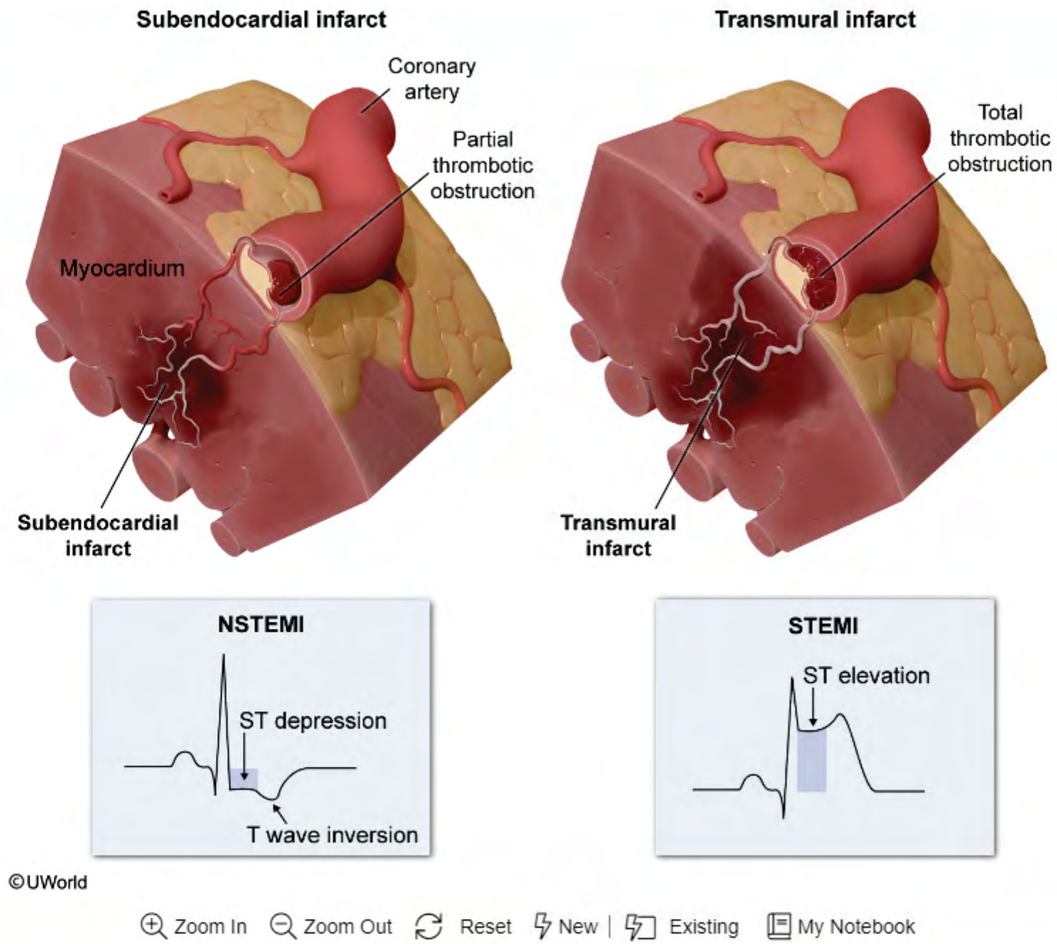

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This patient with persistent substernal chest pain and ECG demonstrating ST elevation in the anterolateral leads has an **ST-elevation myocardial infarction** (STEMI). In the evolution of STEMI on ECG, peaked T waves in the affected leads are typically the first manifestation, followed by the appearance of ST elevation within several minutes, then Q waves within hours to days. Patients with STEMI typically have sudden-onset substernal chest pain that may be accompanied by nausea, diaphoresis, or dyspnea; the pain does not improve with rest or the administration of short-acting nitrates (eg, nitroglycerin).

Myocardial infarction is usually caused by acute **atherosclerotic plaque rupture** with release of lipids, collagen, and tissue factor into the blood stream; because these contents are highly thrombogenic, there is rapid formation of **overlying thrombus**. In STEMI, the overlying thrombus is extensive enough to fully occlude the coronary artery lumen, completely blocking blood flow to a portion of the heart and leading to transmural (full-thickness) infarction of the myocardial wall. Partially obstructive thrombus **(Choice E)** is likely to produce non-STEMI or unstable angina. Non-STEMI is associated with impaired blood flow to only the subendocardial region; the symptomatic presentation is similar to STEMI, but ECG reveals ST segment and T wave abnormalities with no ST elevation.

(Choice A) Atherosclerotic plaque obstructing 50% of the coronary artery lumen is likely to be asymptomatic. Symptoms of stable angina do not typically become prominent until >70% of the cross-sectional area of the coronary artery lumen is occluded.

(Choice B) Atherosclerotic plaque obstructing 80% of the coronary artery lumen is likely to cause stable angina, characterized by substernal or left-sided chest pressure, tightness, or pain that is precipitated by exertion and relieved by rest or short-acting nitrates.

(Choice D) Coronary artery vasospasm (vasospastic or Prinzmetal angina) results from hyperactivity of intimal smooth muscle, usually in the absence of significant atherosclerotic coronary artery disease. Transient anginal chest pain with corresponding ST elevation on ECG can occur; however, the chest pain should rapidly resolve with nitroglycerin.

Educational objective:
ST-elevation myocardial infarction involves transmural (full-thickness) infarction of the myocardial wall, and usually results from acute atherosclerotic plaque rupture with the development of overlying thrombus that fully occludes

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affected leads are typically the first manifestation, followed by the appearance of ST elevation within several minutes, then Q waves within hours to days. Patients with STEMI typically have sudden-onset substernal chest pain that may be accompanied by nausea, diaphoresis, or dyspnea; the pain does not improve with rest or the administration of short-acting nitrates (eg, nitroglycerin).

Myocardial infarction is usually caused by acute **atherosclerotic plaque rupture** with release of lipids, collagen, and tissue factor into the blood stream; because these contents are highly thrombogenic, there is rapid formation of **overlying thrombus**. In STEMI, the overlying thrombus is extensive enough to fully occlude the coronary artery lumen, completely blocking blood flow to a portion of the heart and leading to transmural (full-thickness) infarction of the myocardial wall. Partially obstructive thrombus **(Choice E)** is likely to produce non-STEMI or unstable angina. Non-STEMI is associated with impaired blood flow to only the subendocardial region; the symptomatic presentation is similar to STEMI, but ECG reveals ST segment and T wave abnormalities with no ST elevation.

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Educational objective:
ST-elevation myocardial infarction involves transmural (full-thickness) infarction of the myocardial wall, and usually results from acute atherosclerotic plaque rupture with the development of overlying thrombus that fully occludes the coronary artery lumen. It classically presents with sudden-onset substernal chest pain that is not relieved by rest or short-acting nitrates. ECG demonstrates ST elevation in the affected leads with subsequent development of Q waves.

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Researchers conduct a study in an animal fetus with circulation similar to that of humans. The fetus is delivered close to term by cesarean delivery. Prior to placental separation and the initiation of pulmonary respiration, blood samples are simultaneously collected from several different fetal blood vessels. The blood oxygen saturations measured from each of the samples are 71%, 64%, 62%, 55%, and 43%. The sample with the greatest oxygen saturation was most likely obtained from which of the following blood vessels?

- A. Descending aorta
- B. Ductus arteriosus
- C. Inferior vena cava
- D. Pulmonary trunk
- E. Superior vena cava
- F. Umbilical artery

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- A. Descending aorta (6%)
- B. Ductus arteriosus (13%)
- ✓ C. Inferior vena cava (53%)
- D. Pulmonary trunk (4%)
- E. Superior vena cava (4%)
- F. Umbilical artery (17%)

Correct

53%
Answered correctly

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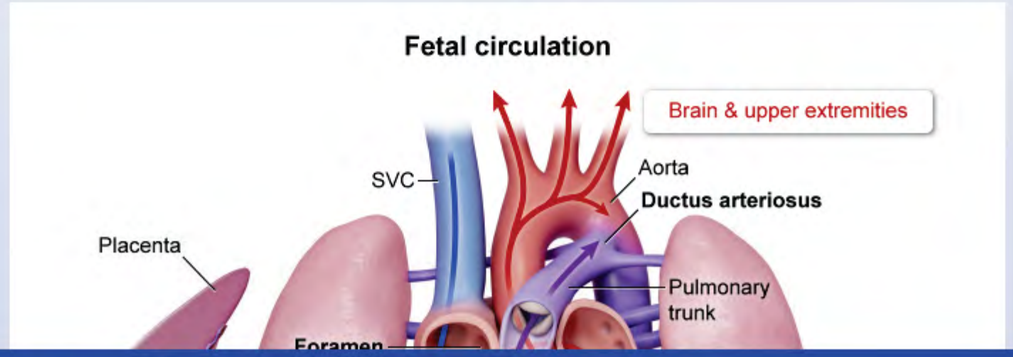
Explanation



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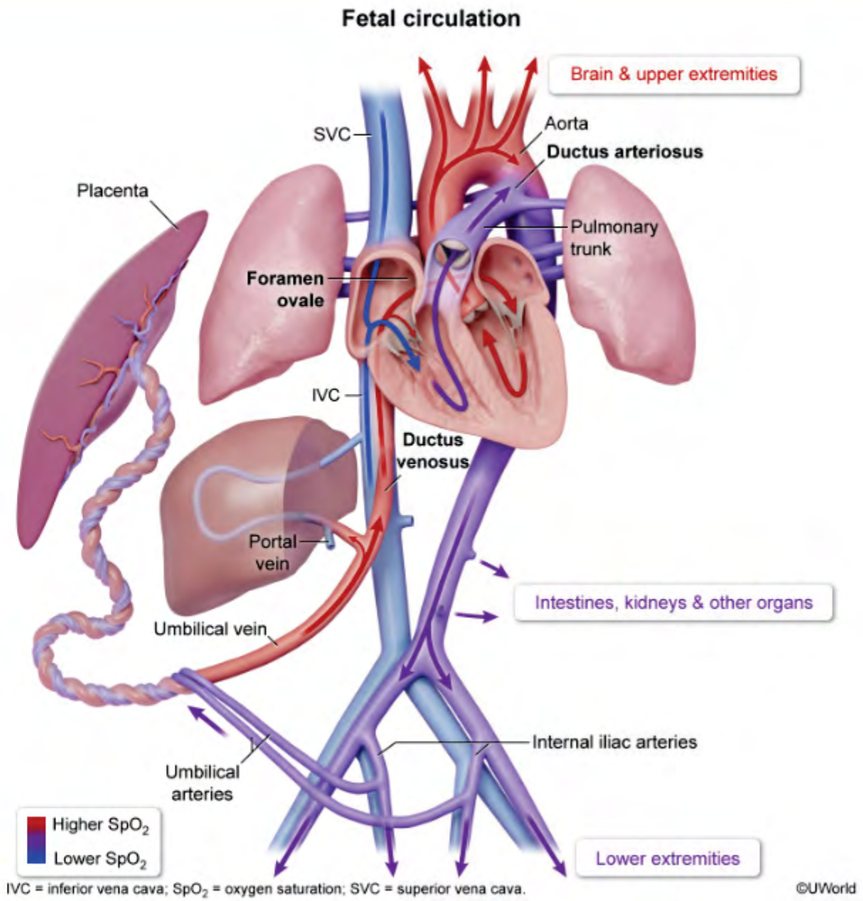

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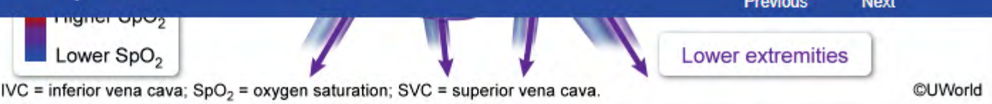

IVC = inferior vena cava; SpO_2 = oxygen saturation; SVC = superior vena cava.

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Fetal circulation differs considerably from adult circulation in that **oxygen** is supplied to the fetus by the mother's **placenta**, no gas exchange occurs in the fetal lungs, and the majority of blood is directed away from the lungs via right-to-left shunting (eg, foramen ovale, ductus arteriosus).

Oxygenated blood from the placenta is delivered to the fetus via the **umbilical vein**. Upon entering the fetal abdomen, a portion of this oxygen-rich blood is directed to the portal vein to supply the liver, while the majority is shunted through the **ductus venosus** to the **inferior vena cava** (IVC). In the IVC, oxygen-rich blood from the ductus venosus runs alongside a stream of deoxygenated blood returning from the lower body.

As blood from the IVC enters the right atrium, a **preferential streaming** effect allows the majority of the oxygenated blood to pass directly through the **foramen ovale** into the left atrium. The oxygen-rich blood delivered to the left atrium proceeds to the left ventricle and out the aorta, where it is supplied to the brain, myocardium (via the coronary arteries), and upper extremities.

Meanwhile, deoxygenated blood from the IVC and superior vena cava fills the right atrium and proceeds to the right ventricle. **High pulmonary vascular resistance** causes most of the blood ejected into the pulmonary trunk to be shunted away from the lungs, with 90% of the deoxygenated blood being shunted through the **ductus arteriosus** into the aorta **(Choices B and D)**. This deoxygenated blood mixes with the oxygen-rich blood in the aorta, sending moderately oxygenated blood down the descending aorta to supply the intestines, kidneys, and lower body.

(Choice A) Oxygen saturation in the descending aorta is significantly lower than in the ductus venosus because the oxygenated blood in the descending aorta is mixed with deoxygenated blood from both the lower and the upper body.

(Choice E) The superior vena cava carries deoxygenated blood returning from the brain and upper extremities.

(Choice F) The umbilical arteries branch off the internal iliac arteries to return moderately oxygenated blood to the placenta, where carbon dioxide and other wastes are unloaded and oxygen is again collected.

Educational objective:

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(Choice F) The umbilical arteries branch off the internal iliac arteries to return moderately oxygenated blood to the placenta, where carbon dioxide and other wastes are unloaded and oxygen is again collected.

Educational objective:
The most highly oxygenated blood in the fetus is carried by the umbilical vein, which empties directly into the inferior vena cava via the ductus venosus.

References

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A 66-year-old woman comes to the office due to worsening chest discomfort and shortness of breath for the past 4 weeks. There is no history of recent upper respiratory illness, fever, cough, palpitations, or syncope. Medical history is significant for hypertension and breast adenocarcinoma treated with surgery and chemotherapy 6 years ago. The patient does not use tobacco or alcohol and has not traveled recently. Temperature is 37 C (98.6 F), blood pressure is 110/70 mm Hg, and pulse is 90/min. Physical examination shows changes consistent with prior right mastectomy. The right axillary lymph nodes are enlarged. The lungs are clear to auscultation. Heart sounds are distant with no murmur. There is no extremity edema. Bedside echocardiography shows a large pericardial effusion. Pericardiocentesis in this patient is most likely to reveal which of the following findings?

- A. Exudative fluid containing acid-fast bacilli
- B. Hemorrhagic fluid with atypical cells
- C. Purulent fluid with Gram-positive cocci
- D. Serosanguinous fluid positive for viral PCR
- E. Turbid fluid containing fungal elements

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- ✗ A. Exudative fluid containing acid-fast bacilli (3%)
- ✓ B. Hemorrhagic fluid with atypical cells (70%)
- C. Purulent fluid with Gram-positive cocci (7%)
- D. Serosanguinous fluid positive for viral PCR (15%)
- E. Turbid fluid containing fungal elements (2%)

Incorrect
Correct answer
B

70%
Answered correctly

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Explanation

Pericardial effusion	
Etiology	• Acute pericarditis (eg, viral, bacterial, autoimmune) • Cardiac injury (eg, MI, cardiac surgery) • Malignancy (eg, metastatic spread of noncardiac tumors) • Renal failure with uremia

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Explanation

Pericardial effusion	
Etiology	• Acute pericarditis (eg, viral, bacterial, autoimmune) • Cardiac injury (eg, MI, cardiac surgery) • Malignancy (eg, metastatic spread of noncardiac tumors) • Renal failure with uremia
Clinical features	• Dyspnea, chest discomfort, fatigue • Possible cardiac tamponade* (eg, hypotension, JVD, ↓ heart sounds) • ECG: ↓ QRS voltage ± electrical alternans • CXR: enlarged cardiac silhouette** & clear lungs
*Acute or large effusions. **With subacute & large effusions. **CXR** = chest x-ray; **JVD** = jugular venous distension; **MI** = myocardial infarction.	

This patient with a history of right **breast cancer**, right axillary lymph node enlargement (ie, likely metastatic disease), and a large pericardial effusion (eg, distant heart sounds) mostly likely has **malignant pericardial effusion**.

Malignancy (eg, breast cancer) is a common cause of pericardial effusion and may be the initial manifestation of cancer recurrence. Pericardial involvement occurs by either direct tumor extension or metastatic spread through the blood or lymphatics; this often results in **subacute accumulation** (eg, over weeks) of pericardial fluid. Subacute (vs rapid) accumulation gives the pericardium time to **progressively stretch**, keeping intrapericardial pressure stable; during this period, patients often have only **nonspecific symptoms** (eg, dyspnea, chest discomfort). However, large volumes can eventually exceed the pericardium's stretch capacity, increase intrapericardial pressure, and compromise cardiac function.

When the cause of pericardial effusion is not clear, **pericardiocentesis** can be used to acquire fluid for laboratory analysis to help determine etiology. In malignant effusion, pericardial fluid is often **hemorrhagic** due to

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pressure stable; during this period, patients often have only **nonspecific symptoms** (eg, dyspnea, chest discomfort). However, large volumes can eventually exceed the pericardium's stretch capacity, increase intrapericardial pressure, and compromise cardiac function.

When the cause of pericardial effusion is not clear, **pericardiocentesis** can be used to acquire fluid for laboratory analysis to help determine etiology. In malignant effusion, pericardial fluid is often **hemorrhagic** due to inflammation associated with metastatic invasion that causes bleeding from irritated capillaries. Cytologic analysis often reveals **atypical malignant cells**.

(Choice A) Some patients with pulmonary tuberculosis (TB) develop tuberculous pericarditis, which can be accompanied by pericardial effusion containing acid-fast bacilli. However, patients with tuberculous pericarditis typically have symptoms of extrapulmonary TB, including fever, night sweats, and weight loss. In addition, most patients are from, or have traveled to, areas where TB is endemic.

(Choices C and E) Bacteria (eg, *Staphylococcus aureus* [gram-positive cocci]) and fungi can cause purulent pericarditis, which may be accompanied by pericardial effusion containing gross pus (ie, purulent fluid) or microscopic purulence (ie, turbid fluid). However, most patients with purulent pericarditis are acutely ill (eg, fever, tachycardia) and have predisposing risk factors such as recent or ongoing infection, immunosuppression, or thoracic surgery.

(Choice D) Viral infection is a common cause of pericarditis, which can result in pericardial effusion. However, viral pericarditis is often preceded by viral respiratory or gastrointestinal symptoms. In this patient with evidence of metastatic breast cancer (eg, enlarged axillary lymph nodes) and no recent illness, malignant effusion is more likely.

Educational objective:
Malignancy is a common cause of pericardial effusion, which is often subacute due to slower accumulation of pericardial fluid with compensatory pericardial stretching. Pericardiocentesis can acquire fluid for cytologic analysis (eg, atypical cells) to help confirm the etiology.

References

- Neoplastic pericardial effusion.

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A 69-year-old man comes to the office due to a 6-month history of chest tightness when he walks uphill or climbs stairs. The discomfort radiates to his throat and lasts 5-10 minutes before slowly subsiding. Medical history is significant for hyperlipidemia and type 2 diabetes mellitus. His father had coronary artery bypass surgery at age 60 and his mother suffered a stroke. Vital signs are normal. Cardiac examination reveals no murmurs or additional heart sounds. The patient is prescribed a medication that is metabolized to S-nitrosothiols in the vascular smooth muscle cells, and he reports rapid and significant symptom relief. Which of the following components of the cardiovascular system is most susceptible to the medication prescribed to this patient?

- A. Cardiac muscle
- B. Large arteries
- C. Large veins
- D. Precapillary sphincters
- E. Small arteries and arterioles

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- ✗ A. Cardiac muscle (2%)
- B. Large arteries (5%)
- ✓ C. Large veins (64%)
- D. Precapillary sphincters (2%)
- E. Small arteries and arterioles (25%)

Incorrect
Correct answer
C

64%
Answered correctly

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Time Spent

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Explanation

This patient has **stable angina pectoris** and experienced rapid symptomatic relief following receipt of a **nitrate**. Pharmacologic nitrates (eg, nitroglycerin, isosorbide mononitrate, isosorbide dinitrate) are metabolized to **nitric oxide and S-nitrosothiols** via mitochondrial aldehyde dehydrogenase within vascular smooth muscle cells. These compounds in turn activate guanylate cyclase and promote the conversion of guanosine triphosphate to cyclic guanosine monophosphate (cGMP), which leads to dephosphorylation of myosin light chains and causes vascular smooth muscle relaxation.

Nitrates predominantly affect the **large veins**, where smooth muscle relaxation leads to **venodilation** and

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This patient has **stable angina pectoris** and experienced rapid symptomatic relief following receipt of a **nitrate**. Pharmacologic nitrates (eg, nitroglycerin, isosorbide mononitrate, isosorbide dinitrate) are metabolized to **nitric oxide and S-nitrosothiols** via mitochondrial aldehyde dehydrogenase within vascular smooth muscle cells. These compounds in turn activate guanylate cyclase and promote the conversion of guanosine triphosphate to cyclic guanosine monophosphate (cGMP), which leads to dephosphorylation of myosin light chains and causes vascular smooth muscle relaxation.

Nitrates predominantly affect the **large veins**, where smooth muscle relaxation leads to **venodilation** and increased venous capacitance. Cardiac venous return **(preload) is reduced**, leading to a reduction in left ventricular end-diastolic volume and pressure and a consequent decrease in left ventricular wall stress. **Decreased myocardial oxygen demand** leads to relief of anginal symptoms.

(Choice A) Both beta blockers and nondihydropyridine calcium channel blockers (eg, verapamil) are negative inotropic and chronotropic agents. They exert antianginal effects via direct actions on cardiac myocytes to reduce myocardial contractility and heart rate, and thereby decrease myocardial oxygen demand. Nitrates have no direct effect on cardiac myocytes.

(Choice B) Sodium nitroprusside generates nitric oxide while in the bloodstream (rather than within vascular smooth muscle cells) and is much less selective for the large veins than nitrates. It causes relatively balanced vasodilation of both the large arteries and veins, and is useful for reducing blood pressure in patients with hypertensive emergency.

(Choice D) Precapillary sphincters are bands of smooth muscle that regulate blood flow from arterioles into mesenteric capillary beds. The effect of nitrates on precapillary sphincters is minimal compared to that on the large veins.

(Choice E) Dihydropyridine calcium channel blockers (eg, nifedipine, amlodipine, nicardipine) block L-type calcium channels in vascular smooth muscle cells to trigger vasodilation. This effect is exerted on small arteries and arterioles in both the peripheral and coronary circulation, resulting in 2 therapeutic effects on stable angina. The peripheral vasodilation reduces afterload leading to a decrease in cardiac work and myocardial oxygen demand, and the coronary vasodilation increases myocardial oxygen supply.

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Nitrates predominantly affect the **large veins**, where smooth muscle relaxation leads to **venodilation** and increased venous capacitance. Cardiac venous return **(preload) is reduced**, leading to a reduction in left ventricular end-diastolic volume and pressure and a consequent decrease in left ventricular wall stress. **Decreased myocardial oxygen demand** leads to relief of anginal symptoms.

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Educational objective:
Pharmacologic nitrates (eg, nitroglycerin, isosorbide mononitrate, isosorbide dinitrate) are metabolized to nitric oxide and S-nitrothiols in vascular smooth muscle cells, leading to an increase in cyclic guanosine monophosphate (cGMP) that stimulates vasodilation. Large veins are predominantly affected, leading to increased venous capacitance and reduced venous return (preload), which decreases left ventricular wall stress and myocardial oxygen demand to relieve anginal symptoms.

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E. Small arteries and arterioles (25%)

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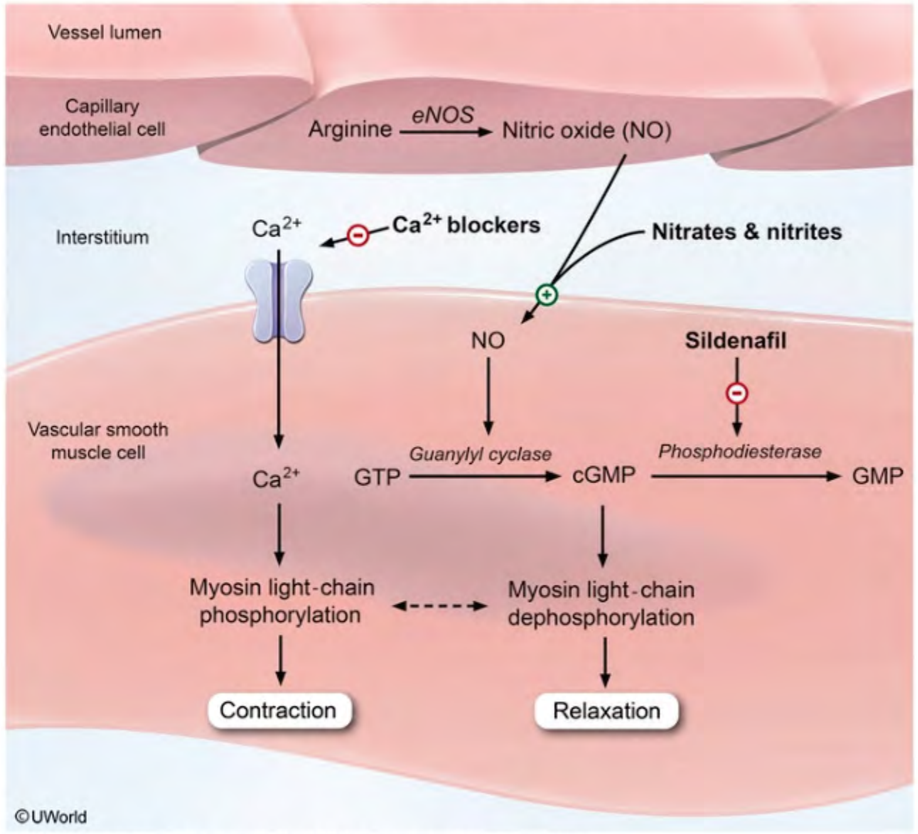
Mechanism of action of vasodilators



cGMP = cyclic guanosine monophosphate; **eNOS** = endothelial nitric oxide synthase; **GMP** = guanosine monophosphate; **GTP** = guanosine 5'-triphosphate.

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(Choice D) Precapillary sphincters are bands of smooth muscle that regulate blood flow from arterioles into mesenteric capillary beds. The effect of nitrates on precapillary sphincters is minimal compared to that on the large

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A 52-year-old man comes to the office due to periodic substernal chest heaviness. The pain is precipitated by fast walking, especially uphill and against the wind, and remits after 5 minutes of rest. The patient has a history of hypertension and smokes a pack of cigarettes a day. Blood pressure is 140/80 mm Hg and pulse is 80/min. The lungs are clear to auscultation, and no heart murmurs are heard. There is no peripheral edema. Which of the following pathologic states is most likely to be present in this patient?

- A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus
- B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus
- C. Ruptured atherosclerotic plaque with fully obstructive thrombus
- D. Significant coronary artery vasospasm causing flow obstruction
- E. Ulcerated atherosclerotic plaque with partially obstructive thrombus

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- A. Atherosclerotic plaque obstructing 50% of the coronary artery lumen, no thrombus (16%)
- ✓ B. Atherosclerotic plaque obstructing 80% of the coronary artery lumen, no thrombus (78%)
- C. ~~Ruptured atherosclerotic plaque with fully obstructive thrombus~~ (0%)
- D. Significant coronary artery vasospasm causing flow obstruction (2%)
- E. Ulcerated atherosclerotic plaque with partially obstructive thrombus (1%)

Correct | 78% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

This patient with multiple risk factors for coronary artery disease (eg, age, hypertension, smoking) has intermittent substernal chest heaviness consistent with **stable angina pectoris**. Stable angina is characterized by substernal or left-sided chest pressure, tightness, or pain that is reliably produced by exertion and relieved by rest. It occurs due to temporary myocardial ischemia resulting from **demand-supply mismatch** of oxygen-rich blood to the myocardium.

Myocardial oxygen demand is determined by heart rate, blood pressure (afterload), left-ventricular end-diastolic volume (preload), and cardiac contractility, whereas myocardial oxygen supply is determined by coronary blood flow. The demand-supply mismatch of stable angina results from **restricted coronary blood flow** due to a fixed **atherosclerotic lesion that obstructs >70%** of the coronary artery lumen. These lesions allow sufficient blood

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Explanation

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(Choice A) An atherosclerotic plaque obstructing 50% of the lumen is unlikely to produce symptomatic myocardial ischemia. Symptoms of stable angina do not become prominent until approximately 70% of the cross-sectional area of the coronary artery lumen is occluded.

(Choice C) Ruptured atherosclerotic plaque with overlying fully obstructive thrombus describes the pathophysiology of ST elevation myocardial infarction. The abrupt cessation of myocardial blood flow leads to transmural infarction and presents with persistent chest pain that is not relieved by rest.

(Choice D) Coronary artery vasospasm (vasospastic angina or Prinzmetal angina) results from hyperactivity of vascular smooth muscle, usually in the absence of significant atherosclerotic coronary artery disease. Transient anginal chest pain is typical; however, the pain is not precipitated by exertion or relieved by rest.

(Choice E) Ulcerated atherosclerotic plaque with overlying partially obstructive thrombus is likely to produce acute coronary syndrome in the form of non-ST elevation (partial-thickness) myocardial infarction or unstable angina. Unstable angina describes anginal pain occurring at rest or with markedly increased frequency, intensity, or duration compared to the patient's baseline stable angina.

Educational objective:

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substernal chest heaviness consistent with **stable angina pectoris**. Stable angina is characterized by substernal or left-sided chest pressure, tightness, or pain that is reliably produced by exertion and relieved by rest. It occurs due to temporary myocardial ischemia resulting from **demand-supply mismatch** of oxygen-rich blood to the myocardium.

Myocardial oxygen demand is determined by heart rate, blood pressure (afterload), left-ventricular end-diastolic volume (preload), and cardiac contractility, whereas myocardial oxygen supply is determined by coronary blood flow. The demand-supply mismatch of stable angina results from **restricted coronary blood flow** due to a fixed **atherosclerotic lesion that obstructs >70%** of the coronary artery lumen. These lesions allow sufficient blood flow to the myocardium during rest but insufficient blood flow to meet myocardial oxygen demands during exertion.

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Educational objective:
Stable angina pectoris results from myocardial oxygen demand-supply mismatch and manifests as chest pressure, tightness, or pain that is reliably produced by exertion and relieved by rest. It most commonly occurs due to a fixed atherosclerotic plaque obstructing >70% of the coronary artery lumen that limits blood flow during exertion.

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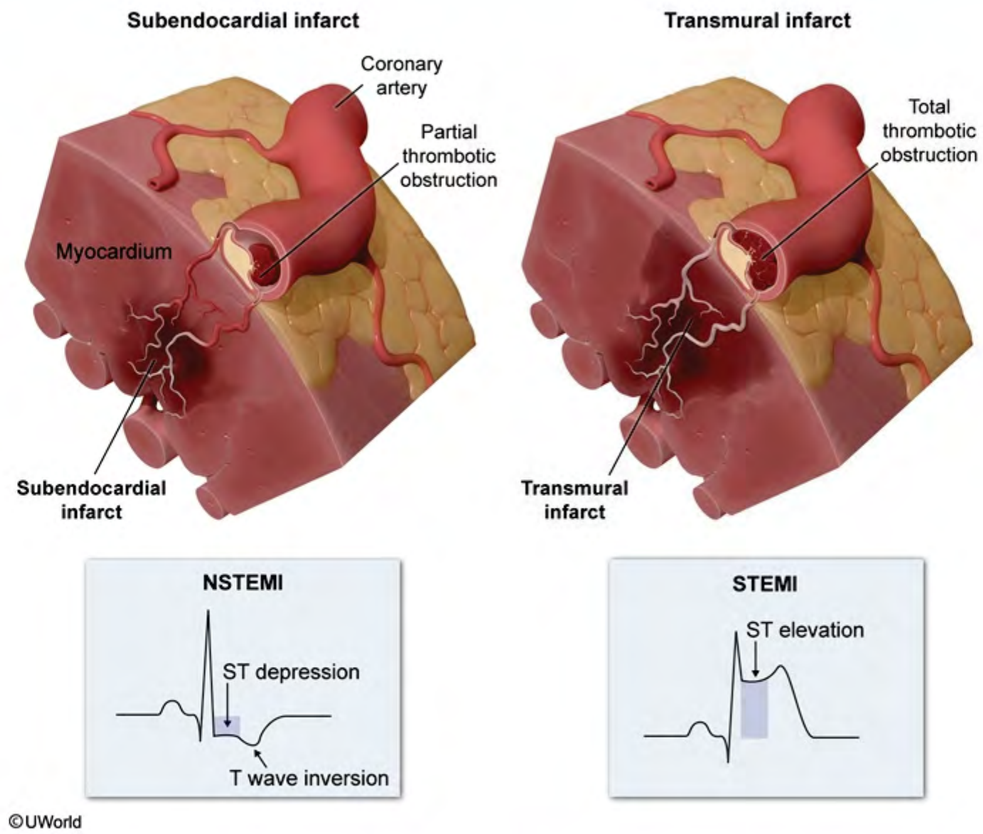

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A 70-year-old man comes to the emergency department due to severe midback pain that started several hours ago. He describes the pain as excruciating and wants immediate relief. The patient also has nausea, diaphoresis, and lightheadedness. He has a history of hypertension and chronic kidney disease. His medication compliance has been poor. He is an active smoker with a 40-pack-year history. His blood pressure on the right arm is 220/105 mm Hg. ECG shows sinus tachycardia and voltage criteria for left ventricular hypertrophy with secondary ST-segment and T wave changes. After initial evaluation, a transesophageal echocardiogram shows a dissection flap in the descending aorta but no evidence of dissection in the ascending aorta. The dissection flap in this patient most likely originates near which of the following points?

- A. Brachiocephalic trunk
- B. Celiac trunk
- C. Intercostal arteries
- D. Left subclavian artery
- E. Renal arteries

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- ✗ A. Brachiocephalic trunk (12%)
- B. Celiac trunk (14%)
- C. Intercostal arteries (5%)
- ✓ D. Left subclavian artery (46%)
- E. Renal arteries (21%)

Incorrect
Correct answer
D

46%
Answered correctly

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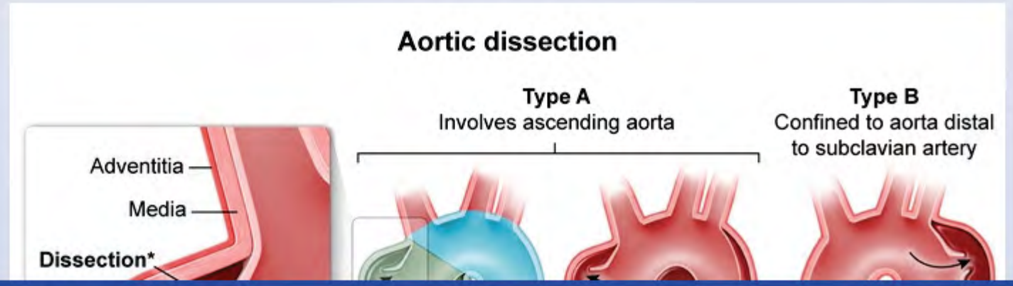
Explanation



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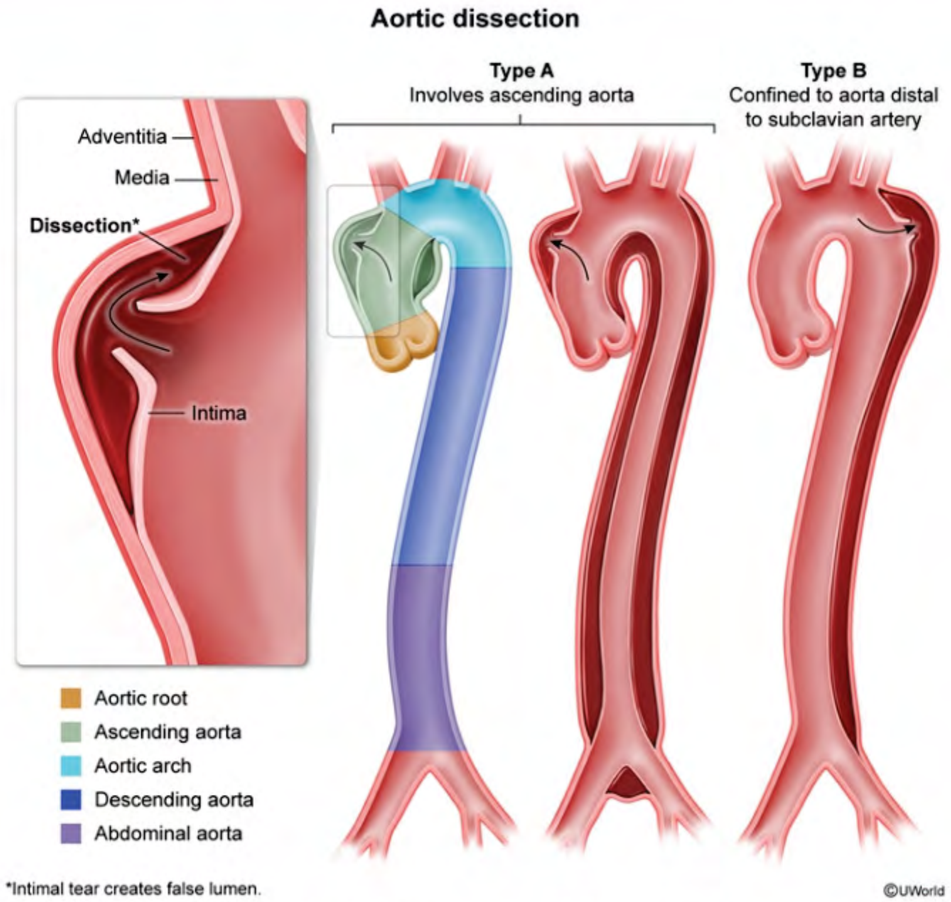

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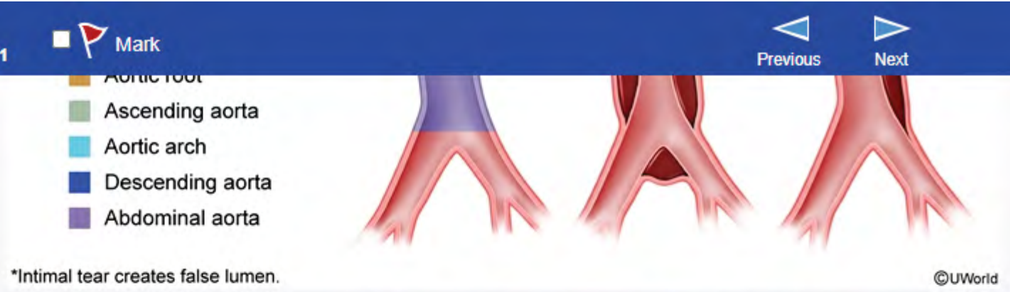

This patient's clinical presentation with severe back pain, hypertension, ECG abnormalities, and transesophageal echocardiogram finding of a dissection flap in the descending aorta is consistent with Stanford type B **aortic dissection**. The Stanford system uses the anatomic localization of the dissection plane for classification of aortic dissections:

- **Stanford type A** refers to aortic dissections that involve any part of the **ascending aorta**.
- **Stanford type B** refers to all other dissections involving the **descending aorta**.

Type B dissections involving the descending aorta typically originate close to the origin of the **left subclavian artery**. Type A aortic dissections usually originate in the sinotubular junction. The areas are thought to be predominantly affected due to increases in the rate of rise of pressure (dP/dT) and in shearing forces at these sites in patients with severe hypertension.

(Choices A, B, C, and E) Distal propagation of a type A dissection or proximal propagation of a type B dissection can affect the aortic arch (eg, brachiocephalic artery, left common carotid artery, left subclavian artery). Either type of dissection can propagate distally into the thoracoabdominal aorta, involving the origins of the celiac trunk, intercostal arteries, and renal arteries.

Educational objective:
The intimal tear in Stanford type A aortic dissection (involving the ascending aorta) usually originates in the sinotubular junction whereas the intimal flap in Stanford type B aortic dissection usually starts near the origin of the left subclavian artery. Dissections can propagate distally to the thoracoabdominal aorta.

Anatomy	Cardiovascular System	Aortic dissection
Subject	System	Topic

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An 8-month-old previously healthy infant is brought to the urgent care clinic due to vomiting and watery diarrhea for the past 3 days. He attends a day care where recently several other children had similar symptoms. His mother states that the infant is less active and has no interest in feeding. Physical examination shows a listless infant with tachycardia, dry mucous membranes, and decreased skin turgor. The abdomen is nontender with hyperactive bowel sounds. Compared to pre-illness levels, which of the following sets of laboratory findings are most likely present in this patient?

	Hematocrit	Serum albumin concentration	Serum uric acid concentration
A.	Decreased	Decreased	Decreased
B.	Decreased	Decreased	Normal
C.	Decreased	Increased	Decreased
D.	Increased	Decreased	Normal
E.	Increased	Increased	Increased
F.	Increased	Normal	Normal

Submit

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		Hematocrit	Serum albumin concentration	Serum uric acid concentration	
✗	A.	Decreased	Decreased	Decreased	(2%)
	B.	Decreased	Decreased	Normal	(9%)
	C.	Decreased	Increased	Decreased	(4%)
	D.	Increased	Decreased	Normal	(13%)
✓	E.	Increased	Increased	Increased	(49%)
	F.	Increased	Normal	Normal	(19%)

Incorrect
Correct answer
E

49%
Answered correctly

03 secs
Time Spent

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Explanation

This patient's history of vomiting and diarrhea and physical examination findings of tachycardia, dry mucous membranes, and decreased skin turgor are consistent with moderate to severe **hypovolemia**. Vomiting and diarrhea cause gastrointestinal losses of both sodium and water from the extracellular space with a consequent **decrease in plasma volume**. Because red blood cells and proteins such as albumin are relatively large and

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Explanation

This patient's history of vomiting and diarrhea and physical examination findings of tachycardia, dry mucous membranes, and decreased skin turgor are consistent with moderate to severe **hypovolemia**. Vomiting and diarrhea cause gastrointestinal losses of both sodium and water from the extracellular space with a consequent **decrease in plasma volume**. Because red blood cells and proteins such as albumin are relatively large and trapped intravascularly, these blood components become concentrated, leading to **increased hematocrit** and **increased serum albumin**. Such hemoconcentration can be a clue to hypovolemia caused by salt and/or water depletion; however, it does not occur with hypovolemia due to acute blood loss because red blood cells and albumin are also depleted.

In the setting of hypovolemia, the kidneys respond by increasing sodium reabsorption to increase circulating blood volume. This increased sodium reabsorption occurs in both the distal renal tubule via increased aldosterone activity and the proximal renal tubule through poorly understood mechanisms. Uric acid absorption is closely tied and directly related to sodium absorption in the proximal renal tubule; therefore, there is typically **increased serum uric acid** in the setting of hypovolemia. Although uric acid levels are not typically collected in the management of hypovolemia, the relationship between hypovolemia and serum uric acid level has clinical application in that hypovolemia increases the risk of acute gout flares.

Educational objective:
Hypovolemia due to loss of sodium and/or water causes increased concentration of red blood cells (ie, hematocrit) and albumin as both of these blood components are trapped within the intravascular space. Hypovolemia also triggers increased absorption of uric acid in the proximal renal tubule, resulting in an increased serum uric acid level.

References

- Renal transport of uric acid: evolving concepts and uncertainties.

Physiology	Cardiovascular System	Hypovolemia
Subject	System	Topic

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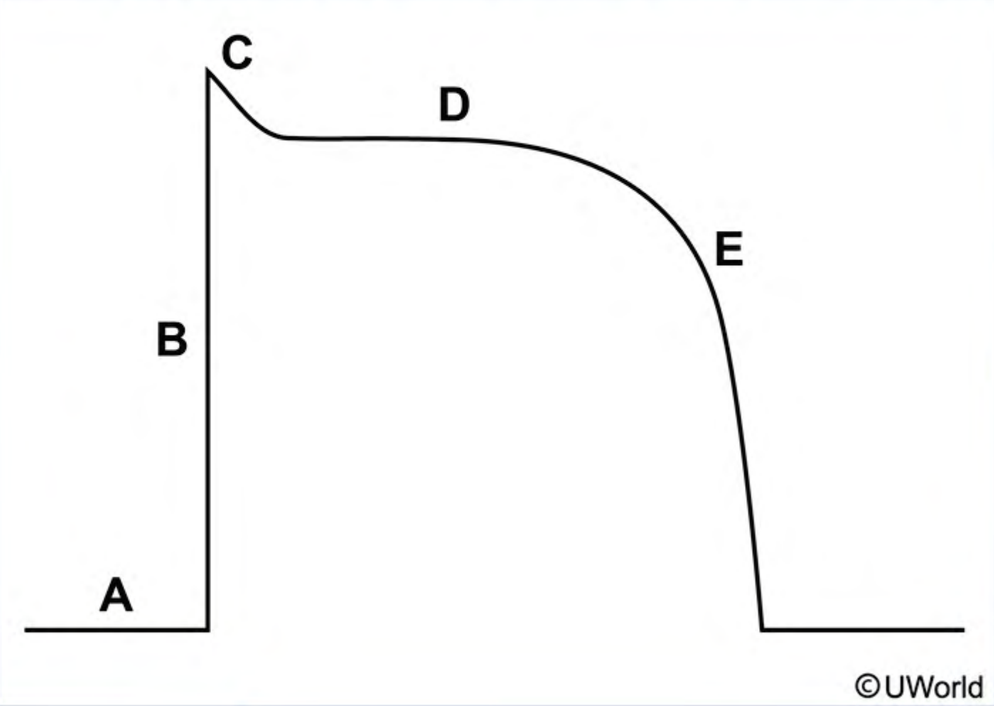
A 37-year-old man with no significant medical history comes to the office due to intermittent palpitations. He has a healthy lifestyle, exercises daily, and does not use tobacco, alcohol, or illicit drugs. ECG shows atrial fibrillation with rapid ventricular response. Echocardiogram reveals normal heart function with no significant valvular disease. The patient is started on an antiarrhythmic medication that is known to be effective against both atrial and ventricular arrhythmias. On ECG, this medication causes QRS prolongation with only minimal increase in QT interval duration. Which of the following regions of the action potential curve is most affected by this drug?



- A. A
- B. B
- C. C

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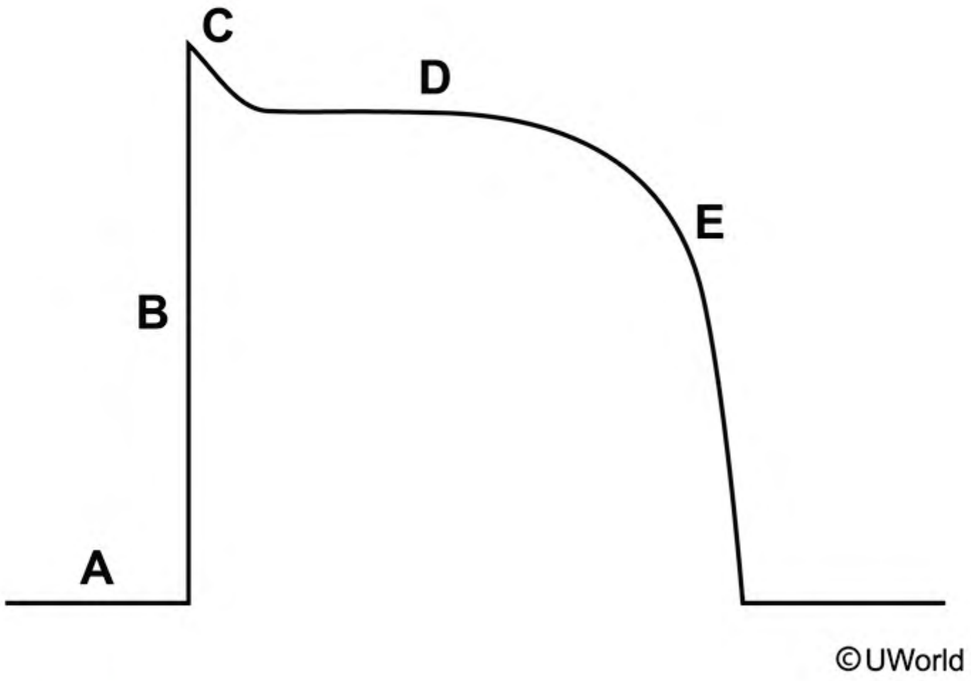

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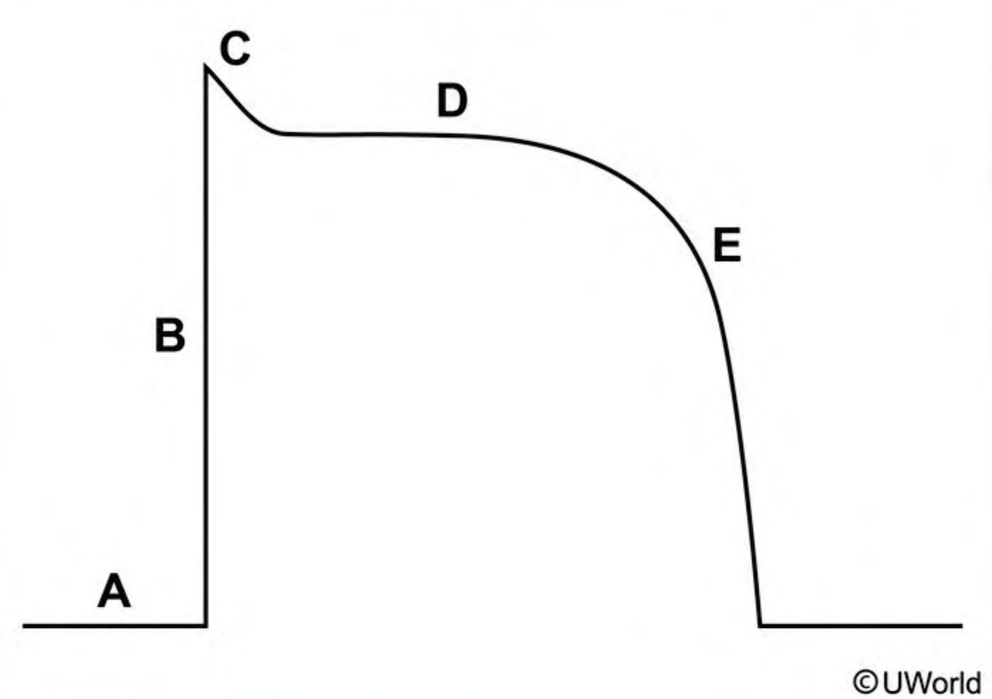

- A. A
- B. B
- C. C
- D. D
- E. E

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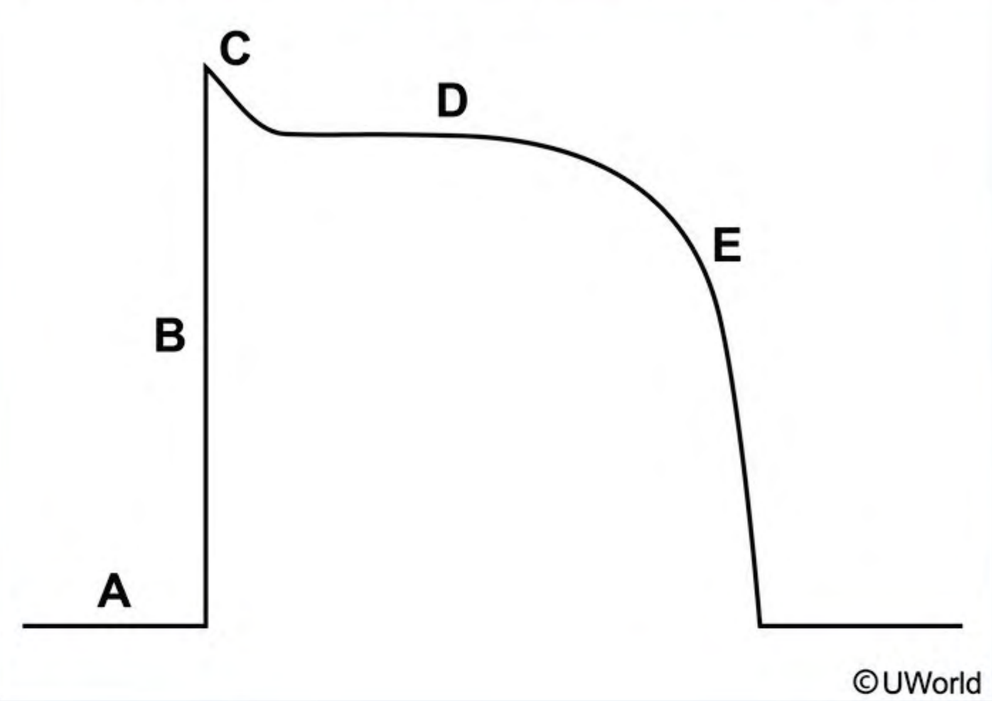

- A. A (1%)
- ✓ B. B (60%)
- C. C (3%)
- ✗ D. D (17%)
- E. E (16%)

Incorrect
Correct answer
B

60%
Answered correctly

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Incorrect
Correct answer
B

60%
Answered correctly

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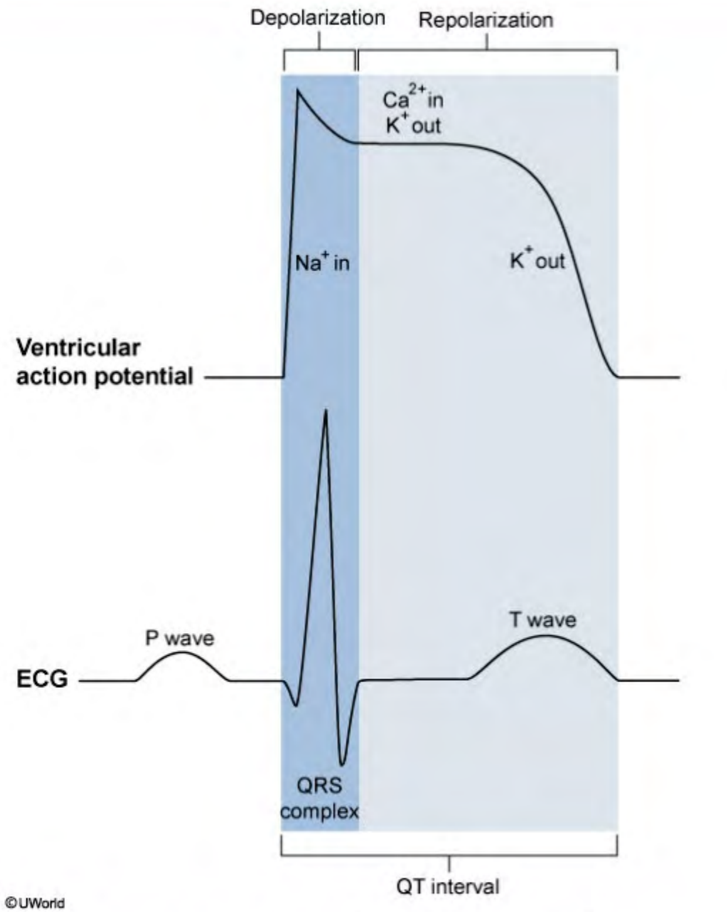

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This patient with atrial fibrillation was most likely started on a **class IC** antiarrhythmic agent, which typically cause

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This patient with atrial fibrillation was most likely started on a **class IC** antiarrhythmic agent, which typically cause QRS prolongation with little effect on QT interval duration.

The **QRS complex** represents rapid **ventricular depolarization** and corresponds to phase 0 of the ventricular myocyte action potential. The QT interval represents both ventricular depolarization and repolarization (total duration of ventricular action potential). However, since ventricular depolarization (QRS duration) contributes only a small amount of time to the interval, the **QT interval** is primarily a measure of **ventricular repolarization**.

Ventricular depolarization occurs due to the opening of **voltage-dependent sodium channels**. In general, class I antiarrhythmics act by blocking these channels, inhibiting the initial depolarization and **prolonging QRS duration**. Specific actions are as follows:

- Class 1A (eg, procainamide) drugs have intermediate sodium channel blocking activity that increases QRS duration. Their moderate potassium channel-blocking activity also causes significant QT interval prolongation.
- Class 1B drugs (eg, lidocaine) have no significant effect on the QRS duration and do not prolong the QT interval due to their rapid dissociation from the receptor.
- Class IC drugs (eg, flecainide, propafenone) are strong sodium channel blockers that prolong QRS duration in a rate-dependent manner. The QT interval may become slightly prolonged due to QRS widening.

(Choice A) Antiarrhythmic drugs do not affect phase 4 of the myocyte action potential, but they can affect phase 4 of the pacemaker cell action potential, which is characterized by slow depolarization due to sodium influx.

(Choice C) Phase 1 of the myocyte action potential refers to a brief period of repolarization caused by a sharp decrease in sodium conductance and an increase in potassium permeability. It is not modulated to an appreciable extent by antiarrhythmic medications.

(Choice D) Calcium channel blockers (eg, verapamil, diltiazem) inhibit the L-type calcium channels during phase 2 depolarization of the myocardial action potential with additional effects on cardiac slow response tissue. They slow the sinus rate, prolong conduction through the atrioventricular node, and depress myocardial contractility (negative inotropic effect).

(Choice E) Class III antiarrhythmic drugs (eg, amiodarone, sotalol, dofetilide) predominantly block potassium channels and inhibit the outward repolarizing currents during phase 3 of the myocyte action potential. This leads to

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(Choice D) Calcium channel blockers (eg, verapamil, diltiazem) inhibit the L-type calcium channels during phase 2 depolarization of the myocardial action potential with additional effects on cardiac slow response tissue. They slow the sinus rate, prolong conduction through the atrioventricular node, and depress myocardial contractility (negative inotropic effect).

(Choice E) Class III antiarrhythmic drugs (eg, amiodarone, sotalol, dofetilide) predominantly block potassium channels and inhibit the outward repolarizing currents during phase 3 of the myocyte action potential. This leads to increased action potential duration and QT interval prolongation.

Educational objective:
Class 1C antiarrhythmic agents (flecainide) block the fast sodium channels responsible for ventricular depolarization (phase 0), prolonging QRS duration with little effect on the QT interval. Class 1A and class III agents cause the most QT prolongation.

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QRS complex

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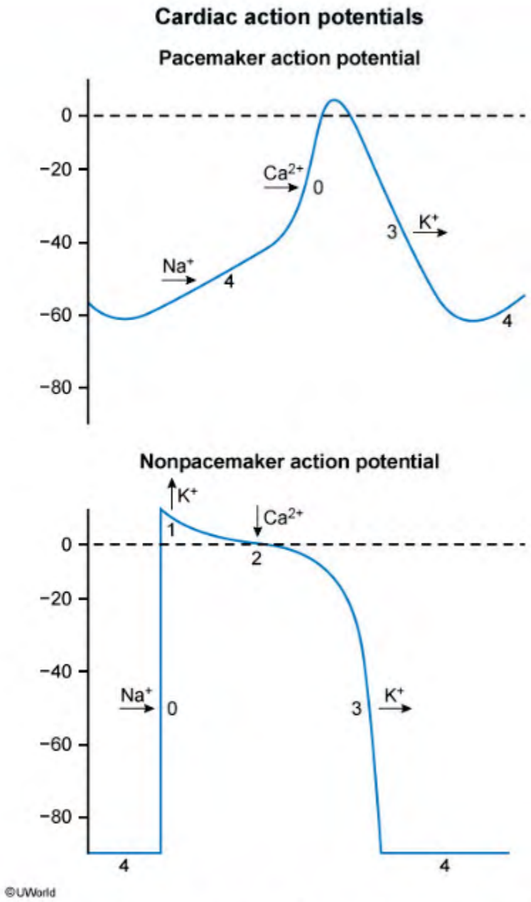

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QRS complex

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Antiarrhythmic drugs – Vaughan Williams classification

Class	Drugs	Predominant actions
IA	Sodium channel blockers • Disopyramide • Procainamide • Quinidine	• Slows AP conduction velocity • Prolongs APD
IB	• Lidocaine • Mexiletine • Phenytoin	• No effect on AP conduction velocity • Shortens APD
IC	• Flecainide • Propafenone	• Slows AP conduction velocity • Minimal effect on APD
II	Beta receptor blockers • Atenolol • Metoprolol • Esmolol • Carvedilol	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness
III	Potassium channel blockers • Amiodarone • Dronedarone • Dofetilide • Sotalol (also class II)	• No effect on AP conduction velocity • Prolongs APD
IV	Non-dihydropyridine calcium channel blockers • Verapamil • Diltiazem	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness

AP = action potential; **APD** = action potential duration; **AV** = atrioventricular.

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A 29-year-old man is brought to the emergency department by his friend due to retrosternal chest pain for the past hour. The patient is agitated and restless. Temperature is 38.1 C (100.6 F), blood pressure is 180/106 mm Hg in the right arm and 182/104 mm Hg in the left arm, pulse is 110/min, and respirations are 20/min. Physical examination shows bilaterally dilated pupils, normal heart and lung sounds, and a nontender abdomen. ECG shows sinus tachycardia with ST-segment depression in the precordial leads. Chest x-ray reveals no parenchymal opacities or pleural effusion and a normal mediastinum and cardiac silhouette. Cardiac enzymes are normal. Sublingual nitroglycerin and benzodiazepine therapy relieve the symptoms. Which of the following is the most likely cause of this patient's chest pain?

- A. Acute pericarditis
- B. Aortic dissection
- C. Coronary artery thrombosis
- D. Coronary artery vasoconstriction
- E. Pulmonary infarction

Submit

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- ✗ A. Acute pericarditis (6%)
- B. Aortic dissection (6%)
- C. Coronary artery thrombosis (8%)
- ✓ D. Coronary artery vasoconstriction (77%)
- E. Pulmonary infarction (0%)

Incorrect
Correct answer
D

77%
Answered correctly

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Explanation

Cardiovascular effects of cocaine intoxication	
Physiologic effects	• Sympathetic stimulation ◦ Hypertension & tachycardia ◦ Coronary vasoconstriction
	• Myocardial ischemia or infarction

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Explanation

Cardiovascular effects of cocaine intoxication	

Physiologic effects	• Sympathetic stimulation
	  ◦ Hypertension & tachycardia
	  ◦ Coronary vasoconstriction

Clinical manifestations	• Myocardial ischemia or infarction • Aortic dissection • Neurologic ischemia or stroke
Treatment	• Benzodiazepines & nitroglycerin • Beta blockers contraindicated

This patient is agitated and restless and has **dilated pupils**, tachycardia, and hypertension, suggesting **increased sympathetic activity** from **cocaine intoxication**. Cocaine use commonly leads to adverse cardiovascular effects that result from inhibition of the presynaptic reuptake of norepinephrine.

Overstimulation of adrenergic receptors (eg, alpha-1, beta-1) causes an increase in heart rate, blood pressure, and myocardial contractility, leading to an increase in myocardial oxygen demand. Arterial vasoconstriction, including **coronary vasoconstriction**, is also enhanced (alpha-1 effect), leading to a decrease in myocardial oxygen supply. This myocardial oxygen supply-demand mismatch often causes **myocardial ischemia**, evidenced in this patient by retrosternal chest pain and ST-segment depression on ECG. Peripheral vasoconstriction may also impair heat loss, causing **low-grade fever**.

Patients with suspected cocaine-induced chest pain are frequently treated with **nitroglycerin** (reduces cardiac preload) and **benzodiazepines** (reduces sympathetic outflow to alleviate tachycardia/hypertension) in order to **improve myocardial ischemia**. Benzodiazepines also calm cocaine-induced psychomotor agitation, which helps further decrease myocardial oxygen demand.

(Choice A) Acute pericarditis causes sharp, pleuritic chest pain and sometimes fever and may lead to widespread ST-segment elevation (but not depression) on ECG. The chest pain typically requires anti-inflammatory medication

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including **coronary vasoconstriction**, is also enhanced (alpha-1 effect), leading to a decrease in myocardial oxygen supply. This myocardial oxygen supply-demand mismatch often causes **myocardial ischemia**, evidenced in this patient by retrosternal chest pain and ST-segment depression on ECG. Peripheral vasoconstriction may also impair heat loss, causing **low-grade fever**.

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(Choice A) Acute pericarditis causes sharp, pleuritic chest pain and sometimes fever and may lead to widespread ST-segment elevation (but not depression) on ECG. The chest pain typically requires anti-inflammatory medication (eg, ibuprofen) to resolve.

(Choice B) Acute aortic dissection commonly presents with abrupt-onset, tearing chest pain in the setting of hypertension, and cocaine use increases the risk. However, a widened mediastinum is expected on chest x-ray, and blood pressure asymmetry is often present in the upper extremities. In addition, the chest pain is unlikely to resolve without pain medication.

(Choice C) Coronary artery thrombosis causes myocardial infarction associated with atherosclerotic plaque rupture, which is unlikely in this relatively young patient. Cocaine use rarely causes coronary artery thrombosis; associated chest pain is usually from myocardial ischemia due to myocardial oxygen supply-demand mismatch.

(Choice E) Acute pulmonary embolism may cause pulmonary infarction, leading to pleuritic chest pain. An associated pleural effusion is usually expected on chest x-ray, and the pain is unlikely to resolve without pain medication.

Educational objective:
Cocaine intoxication causes increased sympathetic activity, which may be recognized by agitation, dilated pupils, tachycardia, and hypertension. Cocaine-induced chest pain typically results from myocardial oxygen supply-demand mismatch (ie, myocardial ischemia) in the setting of coronary artery vasoconstriction. Sublingual nitroglycerin and benzodiazepines help improve the myocardial ischemia and relieve the chest pain.

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A 68-year-old woman comes to the office due to recurrent syncopal episodes. The most recent incident occurred 3 days ago while she was walking to her car and lasted about 1 minute. She was able to resume her normal activities afterward although she felt tired. She had no preceding chest pain or palpitations. The patient has had 2 similar syncopal episodes in the past 2 months, along with frequent episodes of intermittent dizziness. Medical history includes hypertension and chronic kidney disease. She takes lisinopril, amlodipine, and clonidine daily. Blood pressure is 155/85 mm Hg and pulse is 44/min. Heart and lung sounds are normal. After the offending medication is discontinued, she has no further syncopal episodes. The drug responsible for this patient's episodes most likely functions through which of the following mechanisms?

- A. Decreasing presynaptic release of acetylcholine
- B. Decreasing presynaptic release of norepinephrine
- C. Blockade of beta-1 receptors in the sinoatrial node
- D. Blockade of calcium channels in the atrioventricular node
- E. Blockade of potassium channels in the atrioventricular node

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- A. Decreasing presynaptic release of acetylcholine (6%)
- ✓ B. Decreasing presynaptic release of norepinephrine (41%)
- C. Blockade of beta-1 receptors in the sinoatrial node (8%)
- D. Blockade of calcium channels in the atrioventricular node (38%)
- E. Blockade of potassium channels in the atrioventricular node (6%)

Correct | 41% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Medication-induced orthostasis	
Mechanism	**Examples**
α1 blockade–mediated vasodilation	• Terazosin, prazosin, doxazosin • Antipsychotics (eg, risperidone) • Antihistamines, TCAs

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Explanation

Medication-induced orthostasis	
Mechanism	**Examples**
α1 blockade–mediated vasodilation	• Terazosin, prazosin, doxazosin • Antipsychotics (eg, risperidone) • Antihistamines, TCAs
Other vasodilation	• ACE inhibitors & ARBs • Dihydropyridine CCBs • Hydralazine, nitrates • Phosphodiesterase inhibitors
Volume depletion	• Diuretics • SGLT-2 inhibitors
Sympathetic blockade	• Beta blockers • Clonidine
ARBs = angiotensin II receptor blockers; **CCBs** = calcium channel blockers; **SGLT** = sodium-glucose cotransporter; **TCAs** = tricyclic antidepressants.	

This patient's syncopal episodes are due to bradycardia and decreased peripheral vascular resistance caused by **clonidine**. Clonidine is used in the treatment of severe or refractory hypertension. It exerts its effects on both heart rate and blood pressure by **stimulating presynaptic alpha-2 adrenergic receptors** in the rostral ventrolateral medulla (area responsible for basal and reflex control of sympathetic activity). This effect results in a subsequent **decrease in presynaptic release of norepinephrine** and decreased sympathetic outflow causing both **bradycardia** and a decrease in peripheral vascular resistance.

Normally, to maintain cerebral perfusion while standing, a baroreceptor-mediated increase in sympathetic drive causes vasoconstriction to increase peripheral resistance and increased heart rate and contractility to aid cardiac

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Normally, to maintain cerebral perfusion while standing, a baroreceptor-mediated increase in sympathetic drive causes vasoconstriction to increase peripheral resistance and increased heart rate and contractility to aid cardiac output. These changes facilitate a corrective increase in blood pressure to prevent a drop in cerebral perfusion. However, when an orthostatic stress is applied (eg, upright posture) to this patient with **attenuated sympathetic outflow** due to clonidine, she is unable to maintain cerebral perfusion and syncope results.

Lisinopril and amlodipine can also contribute to orthostatic syncope by impairing peripheral vasoconstriction, but their effects are not as profound as those of clonidine because they do not affect heart rate; in fact, they may result in reflex tachycardia.

(Choice A) Decreased presynaptic release of acetylcholine occurs with certain toxins (eg, botulinum) and some medications (eg, aminoglycoside antibiotics). This effect leads to muscle weakness and does not contribute to syncope.

(Choice C) Beta blockers (eg, atenolol) block beta-1 adrenoreceptors in the sinoatrial node, which can lead to bradycardia and contribute to orthostatic syncope. Clonidine does not block adrenoreceptors; it reduces central sympathetic output.

(Choice D) Nondihydropyridine calcium channel blockers (eg, diltiazem, verapamil) block calcium channels in the atrioventricular node and ventricular myocardium, resulting in slowed cardiac conduction (ie, heart rate) and decreased contractility, which can contribute to orthostatic syncope. Amlodipine, a dihydropyridine calcium channel blocker, has minimal effect on the heart and functions primarily as a direct vasodilator.

(Choice E) Class III antiarrhythmics (eg, dofetilide) bind to and block the potassium channels responsible for phase 3 repolarization of the cardiac action potential. This effect does not contribute to orthostatic syncope, but it

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(Choice E) Class III antiarrhythmics (eg, dofetilide) bind to and block the potassium channels responsible for phase 3 repolarization of the cardiac action potential. This effect does not contribute to orthostatic syncope, but it can prolong the QT interval and increase the risk of polymorphic ventricular tachycardia (ie, torsade de pointes), a serious cause of cardiac syncope.

Educational objective:
Clonidine, an antihypertensive agent, stimulates central alpha-2 adrenergic receptors, causing a decrease in presynaptic release of norepinephrine and a decrease in sympathetic outflow. This prevents the normal baroceptor-mediated increase in peripheral vascular resistance and heart rate during standing and can cause orthostatic syncope.

References

- A case report of clonidine induced syncope: a review of central actions of an old cardiovascular drug.

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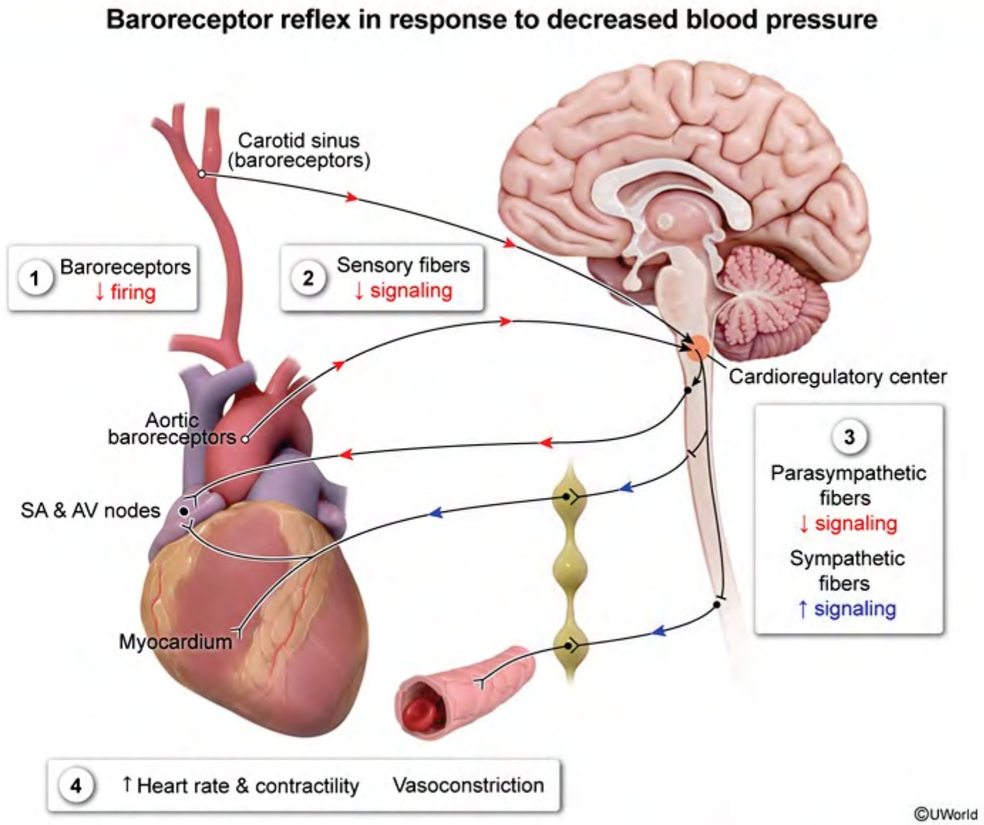

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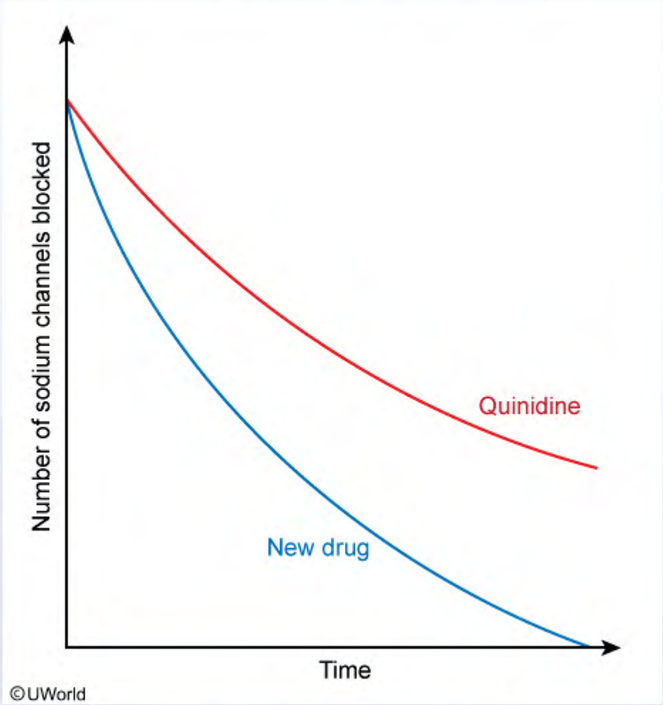
A new drug has been developed to treat cardiac arrhythmias. The drug has high affinity for activated and inactivated sodium channels but relatively little affinity for resting sodium channels. An experiment is devised to maintain sodium channels in the inactivated state for a prolonged period, during which they are exposed to the new drug. After allowing the sodium channels to return to the resting state, the speed of the drug's dissociation from the sodium channels is recorded and shown below. The dissociation curve for quinidine is given for comparison.



The pharmacologic action of the new drug is most similar to which of the following drugs?

- A. Disopyramide
- B. Flecainide
- C. Lidocaine

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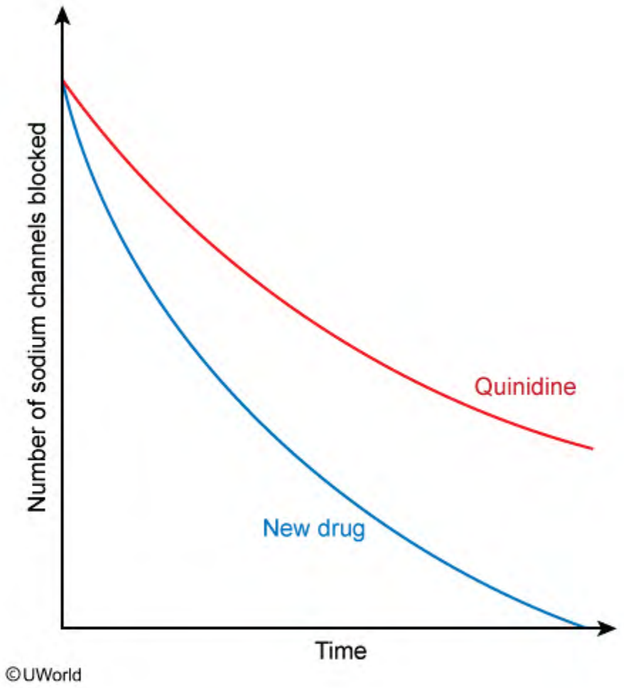

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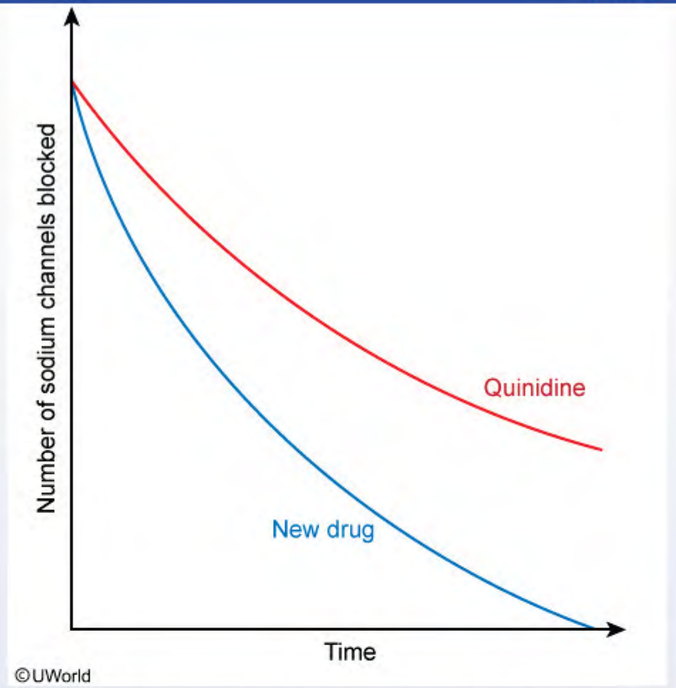

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- A. Disopyramide
- B. Flecainide
- C. Lidocaine
- D. Propranolol
- E. Sotalol
- F. Verapamil

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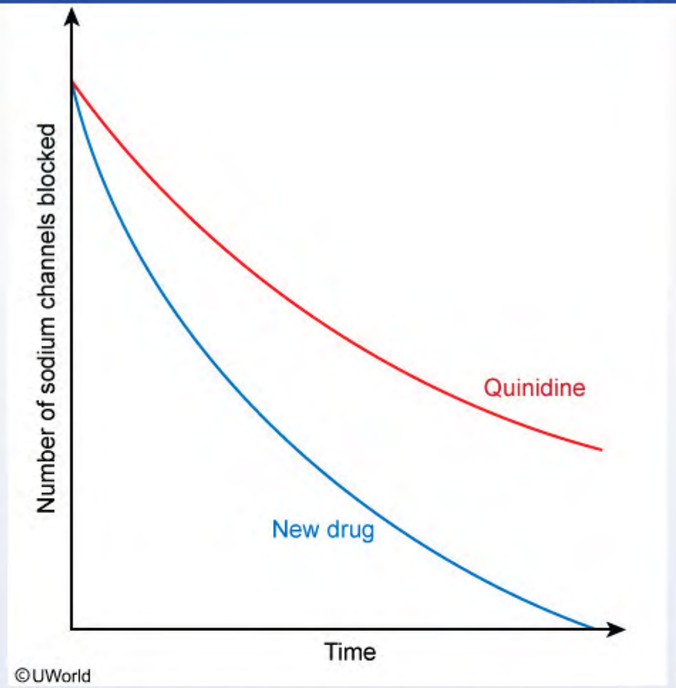

The pharmacologic action of the new drug is most similar to which of the following drugs?

- A. Disopyramide (10%)
- ✕ B. Flecainide (28%)
- ✓ C. Lidocaine (56%)
- D. Propranolol (1%)
- E. Sotalol (1%)
- F. Verapamil (1%)

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Correct answer
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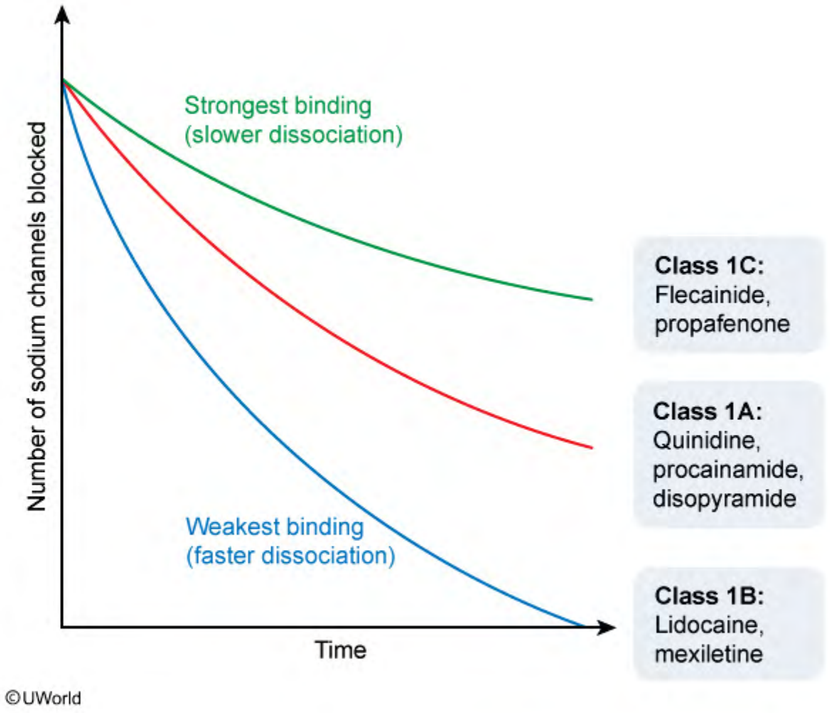

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lidocaine) having the **weakest binding** compared to other class I antiarrhythmics. Dissociation from the channels occurs so rapidly that there is minimal cumulative effect over multiple cardiac cycles, resulting in little use

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The drug described is representative of **a class I antiarrhythmic** medication, which blocks voltage-gated Na^+ channels. Most of the clinically useful agents bind within the pore of the channel during the open and/or inactivated states; dissociation of the drug occurs during the resting state. This state-dependent manner of binding causes a phenomenon in which tissues undergoing frequent depolarization become more susceptible to blockage (**use dependence**). The faster the heart rate, the more time the Na^+ channels spend in the open and inactivated states and the more that become blocked.

The binding strength of class I antiarrhythmics varies between the different subclasses, with **Class IB** agents (eg, lidocaine) having the **weakest binding** compared to other class I antiarrhythmics. Dissociation from the channels occurs so rapidly that there is minimal cumulative effect over multiple cardiac cycles, resulting in little use dependence. These drugs are more **selective for ischemic myocardium** because the elevated (less negative) resting membrane potential delays Na^+ channel transition from the inactivated to the resting state, resulting in increased drug binding. As such, class IB antiarrhythmics are useful for treating **ischemia-induced ventricular arrhythmias**.

In contrast, **class IC** agents (eg, propafenone, flecainide) have the strongest binding **(Choice B)**. They are the **slowest to dissociate** from the channel, which allows their blocking effects to accumulate over multiple cardiac cycles. This leads to substantially greater blockade at higher heart rates (**high use dependence**), making them particularly effective at **terminating tachyarrhythmias**. However, the prominent use-dependent effects of class IC drugs can cause an excessive delay in conduction speed that can promote arrhythmias, especially in patients with ischemic or structural heart disease.

(Choice A) Disopyramide, quinidine, and procainamide are class IA antiarrhythmic drugs that have an intermediate dissociation speed. The dissociation curve for the new drug shows that drug-channel dissociation is faster than with quinidine, indicating that it is most similar to a class IB antiarrhythmic agent such as lidocaine.

(Choice D) Propranolol is a nonselective beta-adrenergic blocker (class II antiarrhythmic). It causes decreased chronotropy and inotropy via beta-1 blockade.

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(Choice D) Propranolol is a nonselective beta-adrenergic blocker (class II antiarrhythmic). It causes decreased chronotropy and inotropy via beta-1 blockade.

(Choice E) Class III antiarrhythmic drugs (amiodarone, sotalol, dofetilide) act predominantly by blocking potassium channels and prolonging phase 3 repolarization. Sotalol also has beta-blocking activity.

(Choice F) Verapamil and diltiazem are calcium channel blockers that function as class IV antiarrhythmics due to their effects on cardiac slow-response tissue. Although nifedipine is a calcium channel blocker as well, it is selective for vascular smooth muscle and does not have a notable effect on the heart.

Educational objective:
For class I antiarrhythmics, sodium-channel-binding strength is **IC > IA > IB**. Use dependence describes the phenomenon in which higher heart rates lead to increased sodium channel blockade due to cumulative blocking effects over multiple cardiac cycles. Class IC antiarrhythmics demonstrate the most use dependence due to their slow dissociation from the receptor, and class IB drugs have the least use dependence as they rapidly dissociate.

Pharmacology	Cardiovascular System	Antiarrhythmic drugs
Subject	System	Topic

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procainamide, disopyramide

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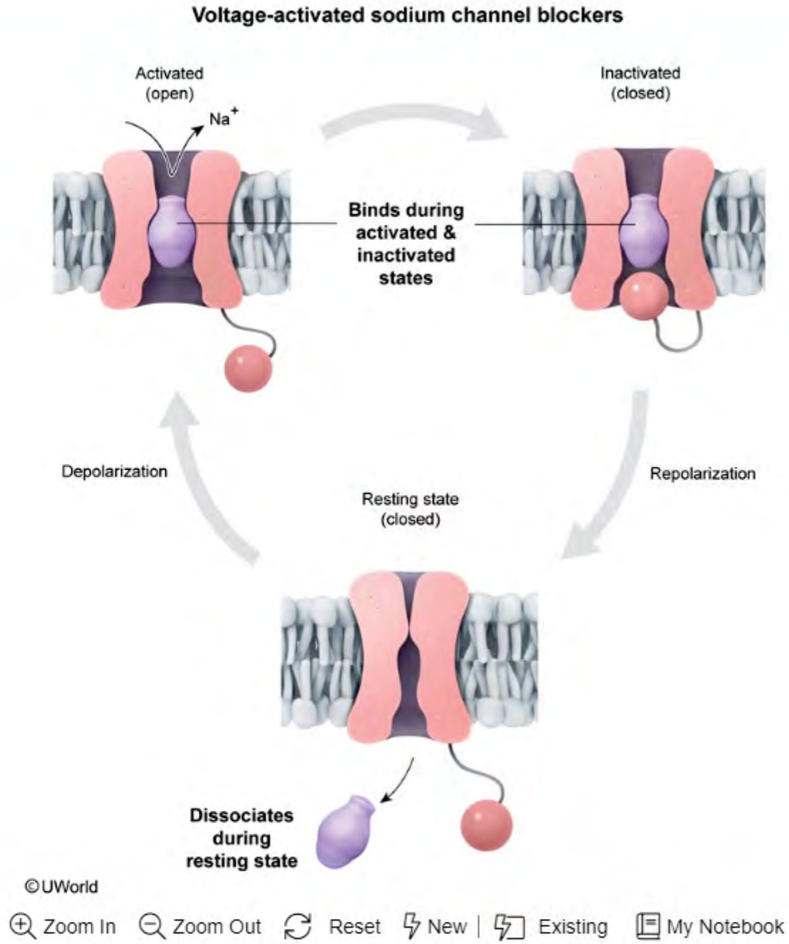

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A 35-year-old woman comes to the office for a follow-up appointment after recent hospitalization. The patient presented to the emergency department with fever and malaise and was diagnosed with infective endocarditis. She underwent a prolonged course of antibiotic therapy and her last dose was 5 days ago. She has no fever or shortness of breath and has resumed her daily activities. The patient has no other medical problems. Blood pressure is 128/72 mm Hg and pulse is 78/min. Auscultatory findings recorded at the left sternal border are shown in the phonocardiogram below.

Which of the following is the most likely cause of the murmur depicted above?

- A. Aortic stenosis
- B. Mitral regurgitation
- C. Mitral stenosis
- D. Pulmonary regurgitation
- E. Tricuspid regurgitation

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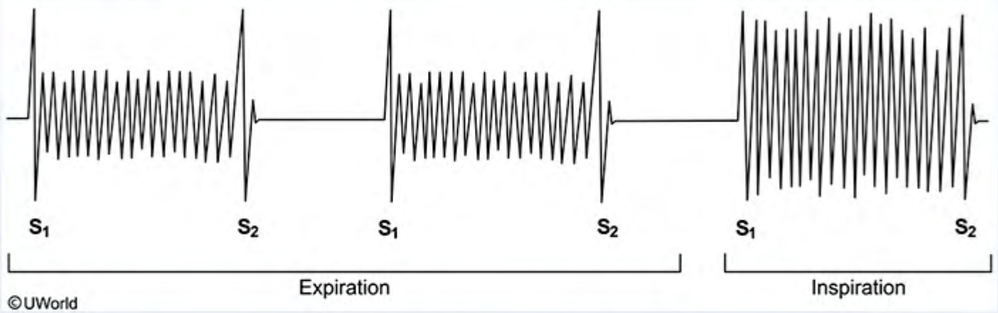

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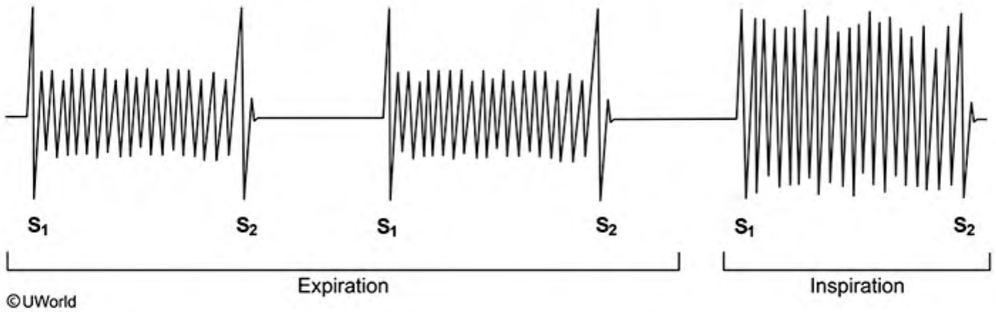

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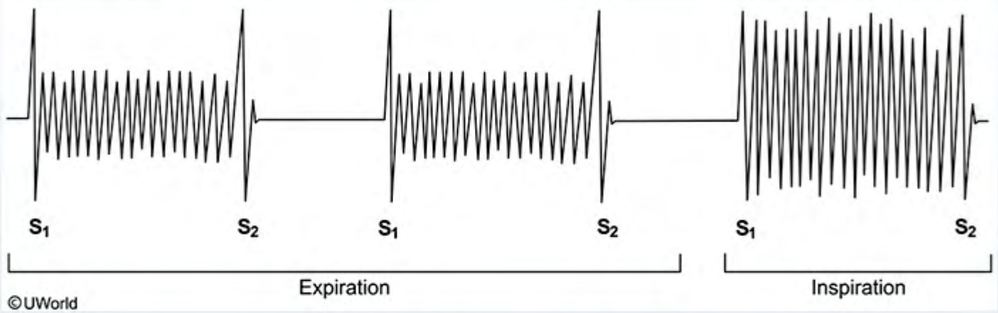

Which of the following is the most likely cause of the murmur depicted above?

- A. Aortic stenosis (5%)
- ✗ B. Mitral regurgitation (25%)
- C. Mitral stenosis (4%)
- D. Pulmonary regurgitation (6%)
- ✓ E. Tricuspid regurgitation (57%)

Incorrect
Correct answer
E

57%
Answered correctly

13 secs
Time Spent

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Explanation

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Explanation

Auscultation of holosystolic murmurs	
Mitral regurgitation	• Best heard at the apex • Radiates to the axilla
Tricuspid regurgitation	• Best heard at the left 2nd & 3rd intercostal spaces • Murmur increases with inspiration
Ventricular septal defect	• Best heard at the left 3rd & 4th intercostal spaces • Murmur is usually loud & accompanied by a thrill

This patient's phonocardiogram shows a **holosystolic murmur** that increases in intensity during inspiration. Holosystolic murmurs are associated with **tricuspid regurgitation** (TR), mitral regurgitation (MR), and ventricular septal defects (VSDs).

Intrathoracic pressure drops during inspiration, allowing more blood to return to the right heart. Right ventricular stroke volume rises due to increased venous return. However, the drop in intrathoracic pressure also increases pulmonary vessel capacity, leading to a transient decrease in left ventricular venous return. As a result, a TR murmur would be expected to increase in intensity during inspiration (Carvallo sign), whereas murmurs associated with MR or VSD would either decrease or not change. TR is further favored over MR as TR is loudest near the left sternal border at the second and third intercostal spaces. MR is heard most prominently over the cardiac apex. A

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(Choice A) Aortic stenosis is characterized by a mid-systolic ejection murmur that starts after S1 and ends before S2 and is heard best at the right second interspace. This murmur has a crescendo-decrescendo (diamond-shaped) configuration.

(Choice B) MR causes a high-pitched holosystolic murmur most prominent over the cardiac apex, which is best heard with the patient in the left lateral decubitus position. The murmur of MR displays little respiratory variation.

(Choice C) Mitral stenosis is associated with an opening snap of the mitral valve and a low-pitched rumbling murmur heard throughout diastole.

(Choice D) Pulmonic regurgitation yields an early diastolic murmur that starts with S2 and ends before S1. The murmur has a decrescendo configuration and may increase in intensity during inspiration. It has a high-pitched, blowing sound that is best heard over the left second and third intercostal spaces.

Educational objective:
A holosystolic murmur that increases in intensity on inspiration most likely represents tricuspid regurgitation. The other holosystolic murmurs (which are secondary to mitral regurgitation or a ventricular septal defect) do not

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This patient's phonocardiogram shows a **holosystolic murmur** that increases in intensity during inspiration. Holosystolic murmurs are associated with **tricuspid regurgitation** (TR), mitral regurgitation (MR), and ventricular septal defects (VSDs).

Intrathoracic pressure drops during inspiration, allowing more blood to return to the right heart. Right ventricular stroke volume rises due to increased venous return. However, the drop in intrathoracic pressure also increases pulmonary vessel capacity, leading to a transient decrease in left ventricular venous return. As a result, a TR murmur would be expected to increase in intensity during inspiration (Carvallo sign), whereas murmurs associated with MR or VSD would either decrease or not change. TR is further favored over MR as TR is loudest near the left sternal border at the second and third intercostal spaces. MR is heard most prominently over the cardiac apex. A VSD produces a holosystolic murmur that is typically loudest over the left sternal border in the third or fourth intercostal spaces.

(Choice A) Aortic stenosis is characterized by a mid-systolic ejection murmur that starts after S1 and ends before S2 and is heard best at the right second interspace. This murmur has a crescendo-decrescendo (diamond-shaped) configuration.

(Choice B) MR causes a high-pitched holosystolic murmur most prominent over the cardiac apex, which is best heard with the patient in the left lateral decubitus position. The murmur of MR displays little respiratory variation.

(Choice C) Mitral stenosis is associated with an opening snap of the mitral valve and a low-pitched rumbling murmur heard throughout diastole.

(Choice D) Pulmonic regurgitation yields an early diastolic murmur that starts with S2 and ends before S1. The murmur has a decrescendo configuration and may increase in intensity during inspiration. It has a high-pitched, blowing sound that is best heard over the left second and third intercostal spaces.

Educational objective:
A holosystolic murmur that increases in intensity on inspiration most likely represents tricuspid regurgitation. The other holosystolic murmurs (which are secondary to mitral regurgitation or a ventricular septal defect) do not typically increase in intensity during inspiration.

Physiology	Cardiovascular System	Tricuspid regurgitation
Subject	System	Topic

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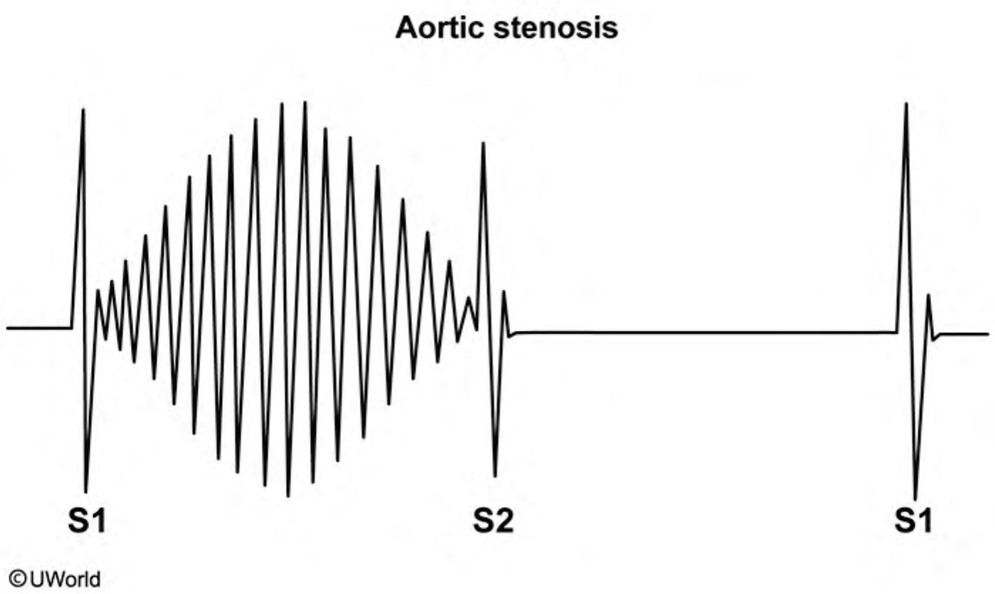

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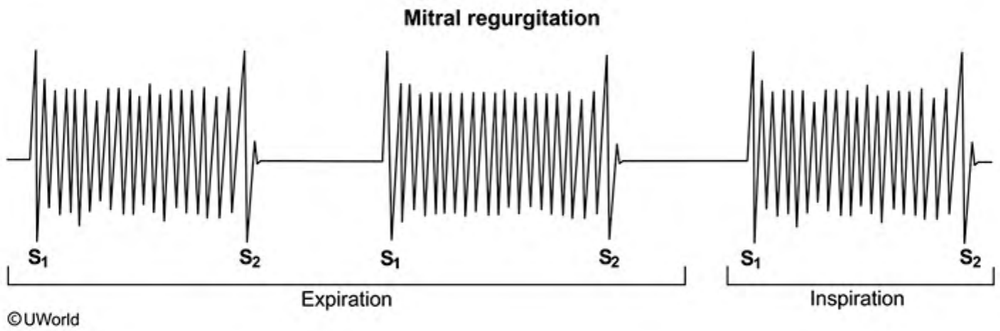

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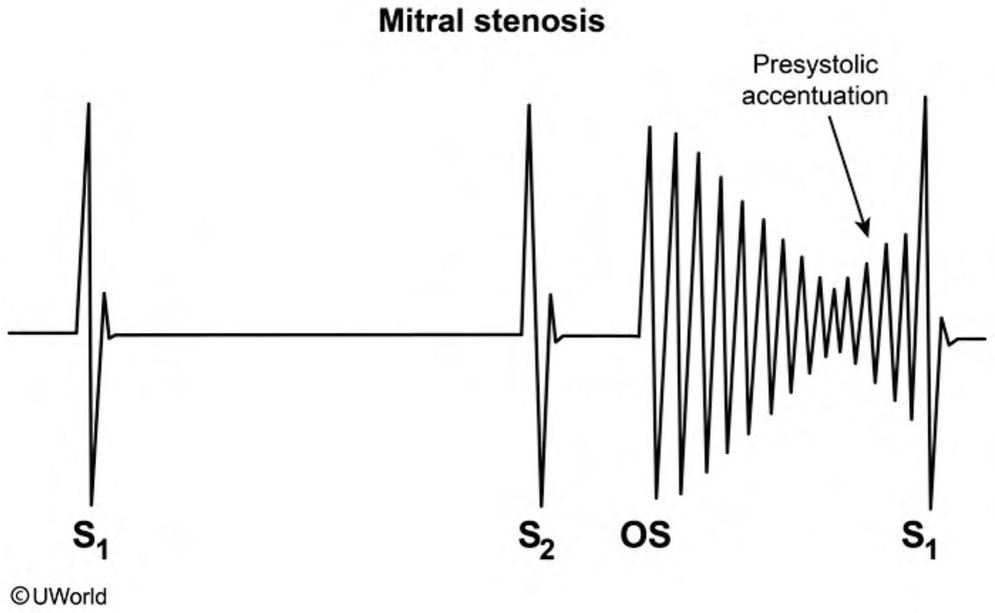

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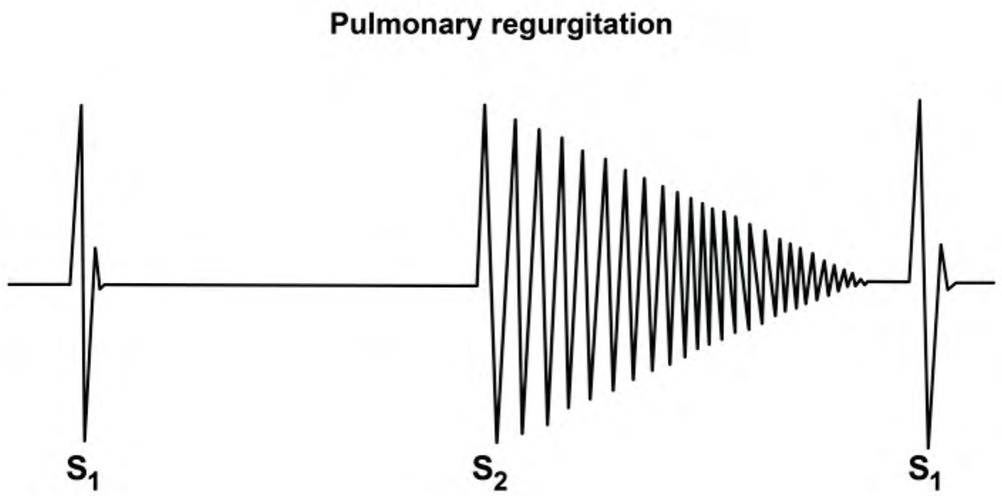

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A 53-year-old man is hospitalized due to chest pain and palpitations. Cardiac catheterization is planned to evaluate for coronary artery disease. The femoral artery is palpated in the right groin, and a catheter is introduced into the common femoral artery. The associated vasculature is visualized after injecting a contrast agent into the catheter.

Which of the following is most likely the artery indicated by the arrow in the image above?

- A. Inferior epigastric
- B. Internal iliac
- C. Medial circumflex femoral
- D. Obturator
- E. Superior gluteal

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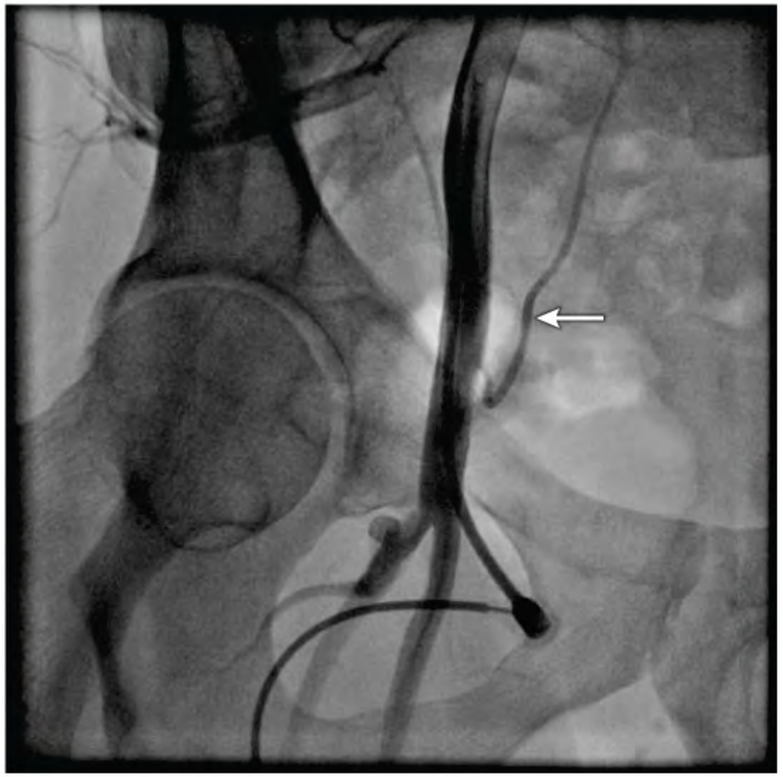

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C. Medial circumflex femoral

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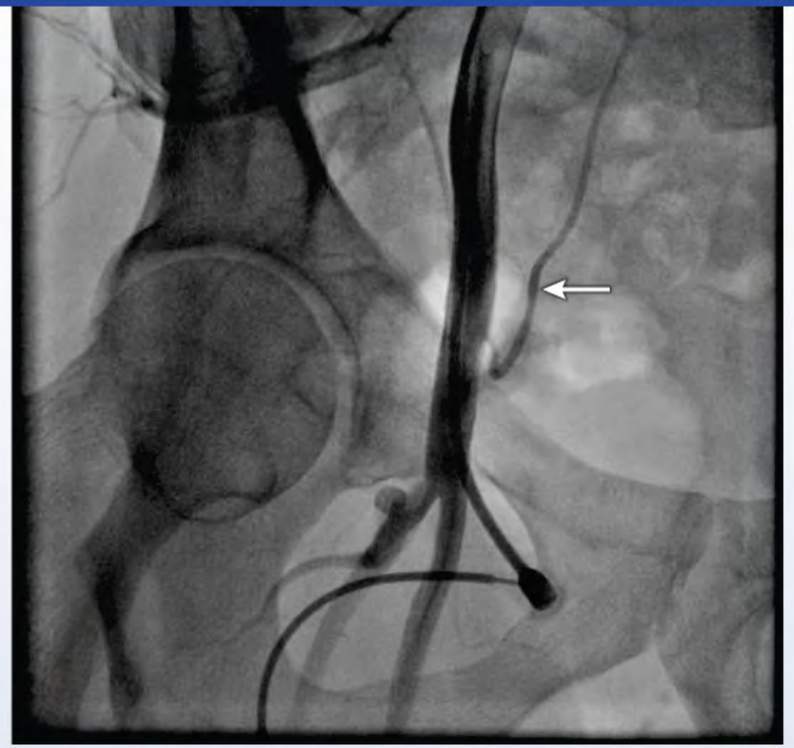

Which of the following is most likely the artery indicated by the arrow in the image above?

- A. Inferior epigastric
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- E. Superior gluteal

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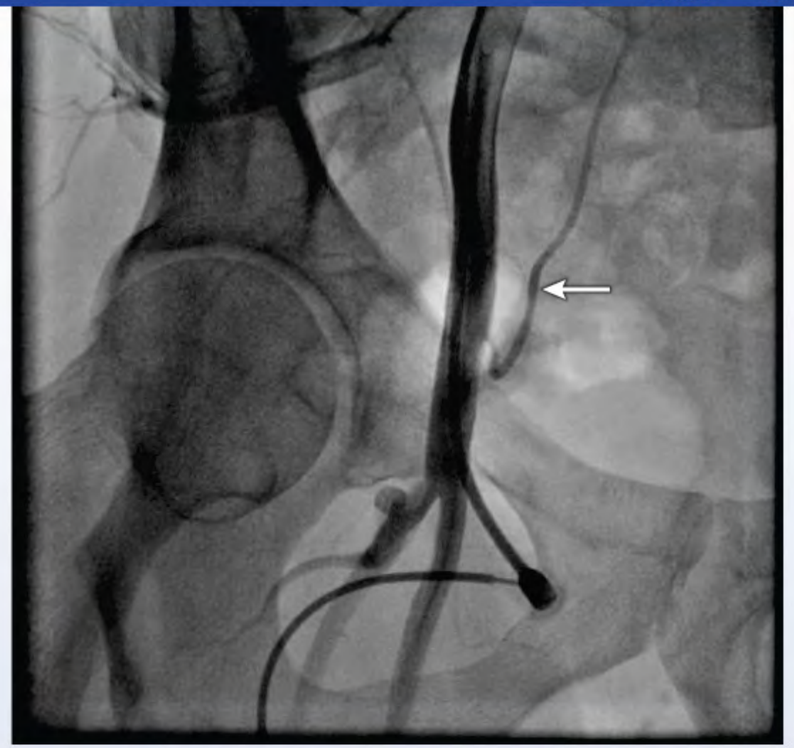

Which of the following is most likely the artery indicated by the arrow in the image above?

- ✓ A. Inferior epigastric (57%)
- B. Internal iliac (7%)
- C. Medial circumflex femoral (13%)
- D. Obturator (9%)
- E. Superior gluteal (11%)

Correct

57%
Answered correctly

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E. Superior gluteal (11%)

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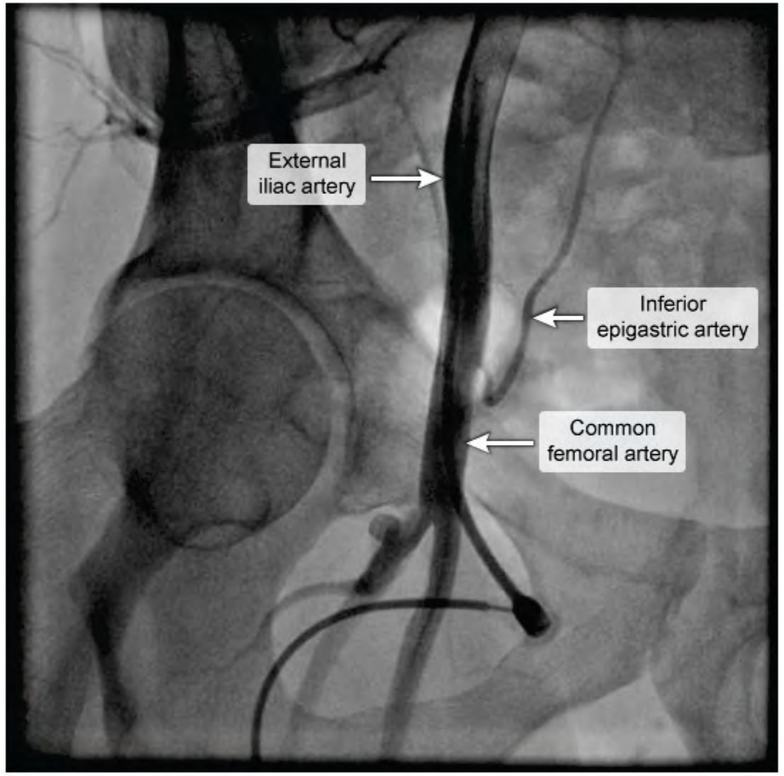
Inferior epigastric artery



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As the aorta courses through the lower abdomen, it divides into the common iliac arteries, which subsequently branch into the internal and external iliac arteries. The **inferior epigastric artery** branches off from the external iliac artery immediately proximal to the inguinal ligament before the external iliac artery becomes the common femoral artery. As the inferior epigastric artery runs **superiorly** and **medially** up the abdomen, it provides blood supply to the lower anterior abdominal wall. The other main branch off the external iliac artery is the deep circumflex iliac artery, which branches more laterally but also supplies blood to the lower abdominal wall.

(Choices B, D, and E) The internal iliac artery supplies most of the structures of the pelvis, gluteal region, and medial thigh. The obturator artery is a branch of the internal iliac artery that runs inferiorly to supply blood to the bladder and medial compartment of the thigh. The superior gluteal artery is the continuation of the internal iliac artery and runs inferiorly to supply blood to the gluteal muscles and a portion of the hip.

(Choice C) The medial circumflex femoral artery branches off the deep femoral artery to supply blood to the femoral neck.

Educational objective:

The inferior epigastric artery branches off the external iliac artery immediately proximal to the inguinal ligament. It provides blood supply to the lower anterior abdominal wall as it runs superiorly and medially up the abdomen.

References

- Percutaneous management of inferior epigastric artery injury after cardiac catheterization.
- Transcatheter coil embolization of the inferior epigastric artery in a huge abdominal wall hematoma caused by paracentesis in a patient with liver cirrhosis.
- Retroperitoneal hemorrhage caused by inferior epigastric artery injury.

Anatomy	Cardiovascular System	Cardiovascular anatomy
Subject	System	Topic

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Anatomy	Cardiovascular System	Cardiovascular anatomy
Subject	System	Topic

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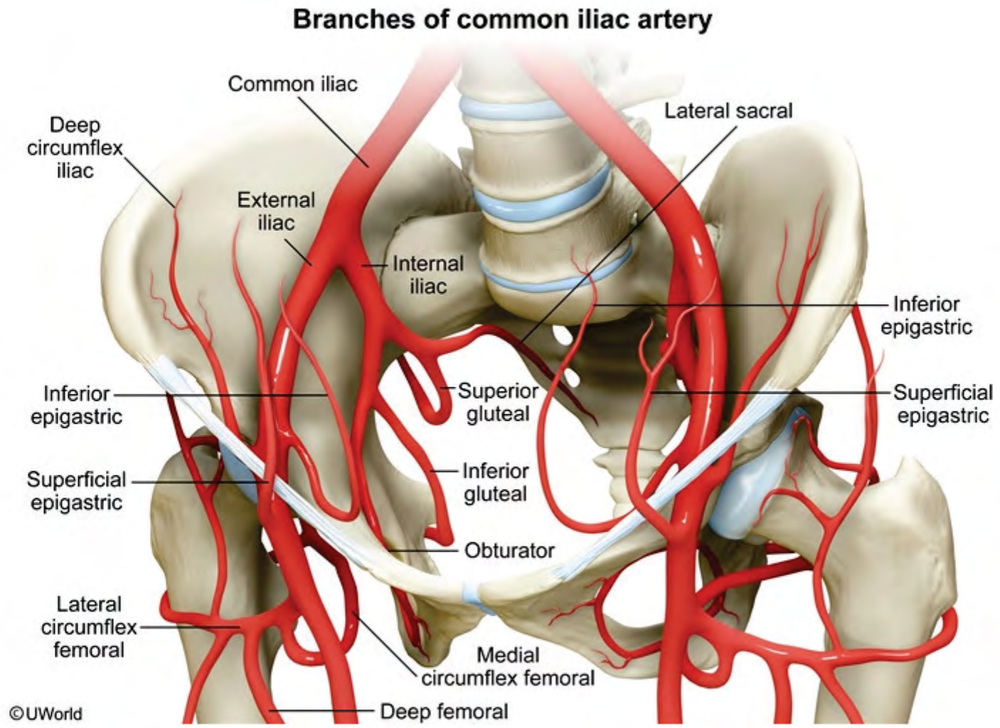

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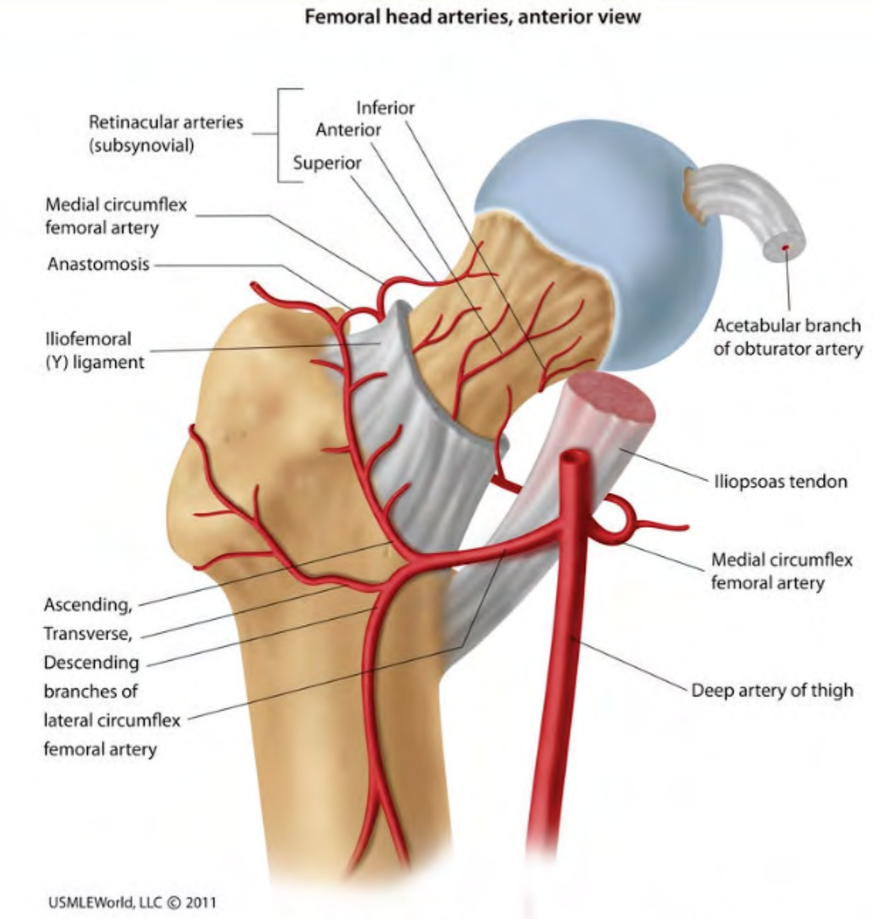

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A 75-year-old man is found unresponsive in his home. Emergency medical services are called by the neighbor, but the patient is pronounced dead at the scene. The patient had a history of hypertension, type 2 diabetes mellitus, and coronary artery disease, as well as a 25-pack-year smoking history. He worked at a coal mine in the remote past and retired as an auto mechanic 15 years ago. Autopsy is performed; a section of lung tissue stained with hematoxylin and eosin is shown below.

Which of the following is the most likely cause of this patient's abnormal histopathology findings?

- A. Chronic inflammation triggered by crystalline silica
- B. Decreased left ventricular systolic function
- C. Excessive deposition of extracellular collagen
- D. Hyperreactivity of airways to extrinsic allergens
- E. Malignant transformation of bronchial epithelial cells
- F. Neutrophil elastase-mediated alveolar septal destruction

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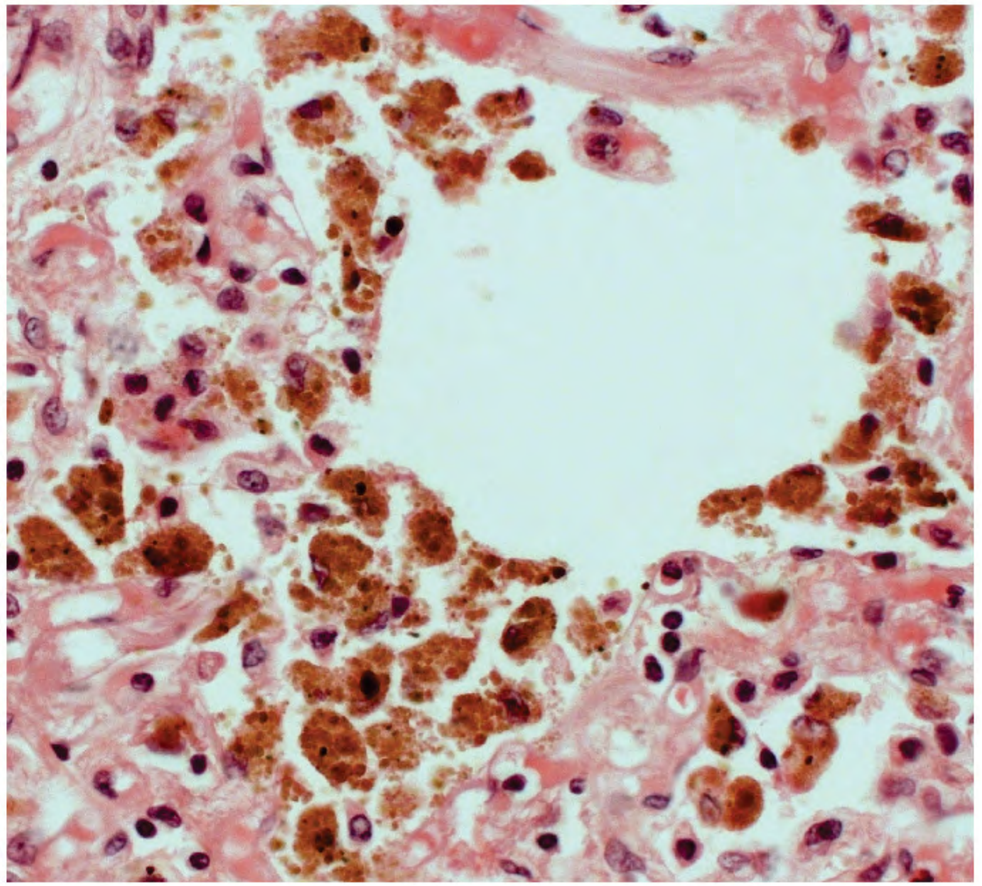

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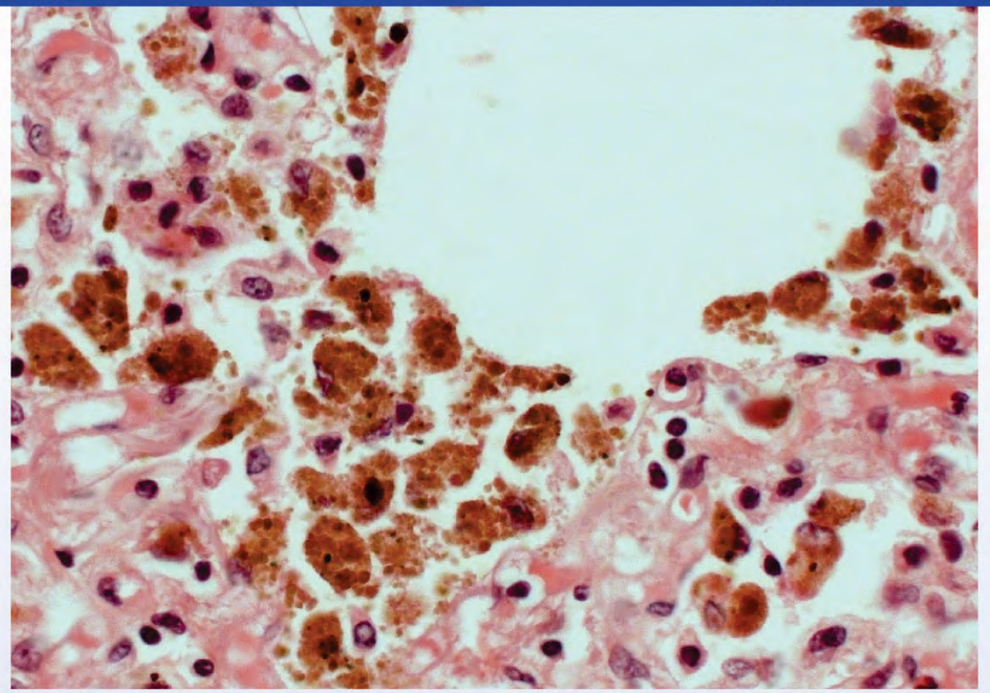

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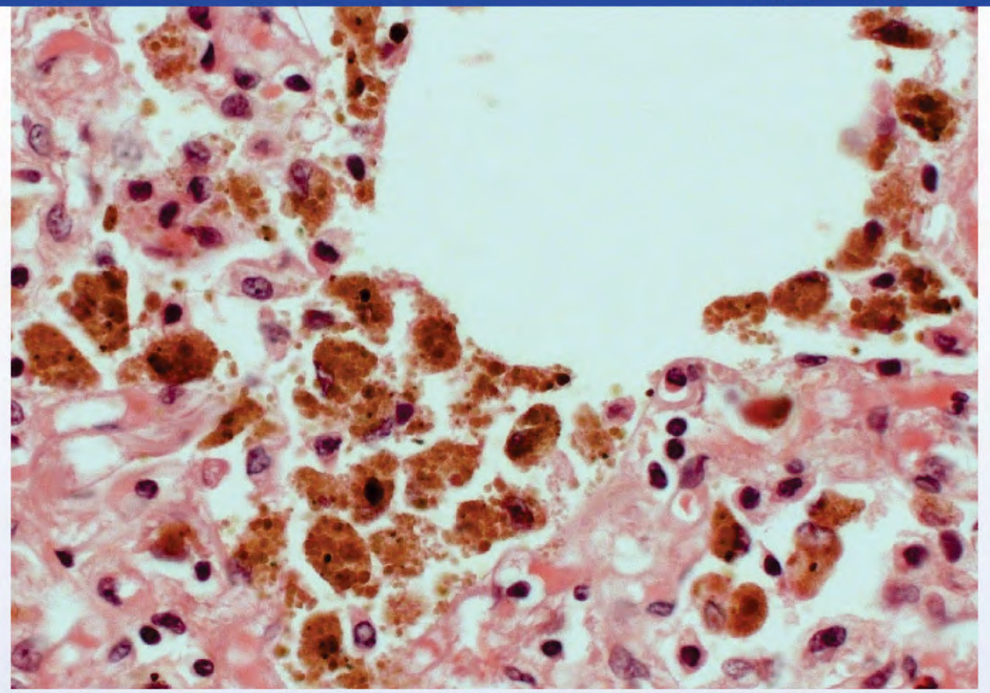

Which of the following is the most likely cause of this patient's abnormal histopathology findings?

- A. Chronic inflammation triggered by crystalline silica (16%)
- ✓ B. Decreased left ventricular systolic function (55%)
- ✗ C. Excessive deposition of extracellular collagen (8%)
- D. Hyperreactivity of airways to extrinsic allergens (6%)
- E. Malignant transformation of bronchial epithelial cells (3%)
- F. Neutrophil elastase-mediated alveolar septal destruction (8%)

Incorrect
Correct answer
B

55%
Answered correctly

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Explanation

This patient's autopsy demonstrates an abundance of alveolar macrophages filled with brown pigment in the lung parenchyma. **Hemosiderin-laden macrophages** located in the lungs are usually the result of **chronic passive lung congestion** in the setting of **heart failure** (eg, left ventricular systolic dysfunction). Elevated pulmonary venous pressure leads to transudation of fluid across the alveolar-capillary membrane (pulmonary edema) and can cause **breaks** in the endothelium with **extravasation of red blood cells** into the alveoli and lung parenchyma. As alveolar macrophages engulf and degrade the extravasated red blood cells, the released iron accumulates as intracellular hemosiderin.

Severe left ventricular systolic dysfunction is associated with an increased risk of fatal ventricular arrhythmia (ie, ventricular tachycardia or fibrillation), which was the likely cause of death in this patient.

(Choices A and C) Silicosis results from chronic inflammation triggered by inhaled particles of crystalline silica. Histopathology typically shows silicotic nodules consisting of whorls of concentric collagen fibers. As with other causes of interstitial lung disease, excessive deposition of extracellular collagen eventually leads to pulmonary fibrosis.

(Choice D) Hyperreactivity of airways to environmental allergens occurs in asthma. Microscopic analysis of sputum may reveal Curschmann spirals, which represent spiral-shaped mucus plugs.

(Choice E) Malignant transformation of bronchial epithelial cells underlies the development of most types of lung cancer. Histopathology typically shows hyperchromatic nuclei and poorly organized cell structure, such as in squamous cell carcinoma of the lung.

(Choice F) Neutrophil elastase-mediated alveolar septal destruction underlies the pathogenesis of emphysema, which can occur with smoking or alpha-1 antitrypsin deficiency. Microscopy shows cystic enlargement of the airspaces with loss of the normal lobular architecture.

Educational objective:
Left-sided heart failure leads to chronically elevated pulmonary venous and capillary pressures, with resulting pulmonary edema and extravasation of red blood cells into the alveolar parenchyma. The iron from red blood cells is taken up by alveolar macrophages and stored as hemosiderin, appearing as brown pigment on microscopy.

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Educational objective:
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Pathophysiology	Cardiovascular System	Chronic heart failure
Subject	System	Topic

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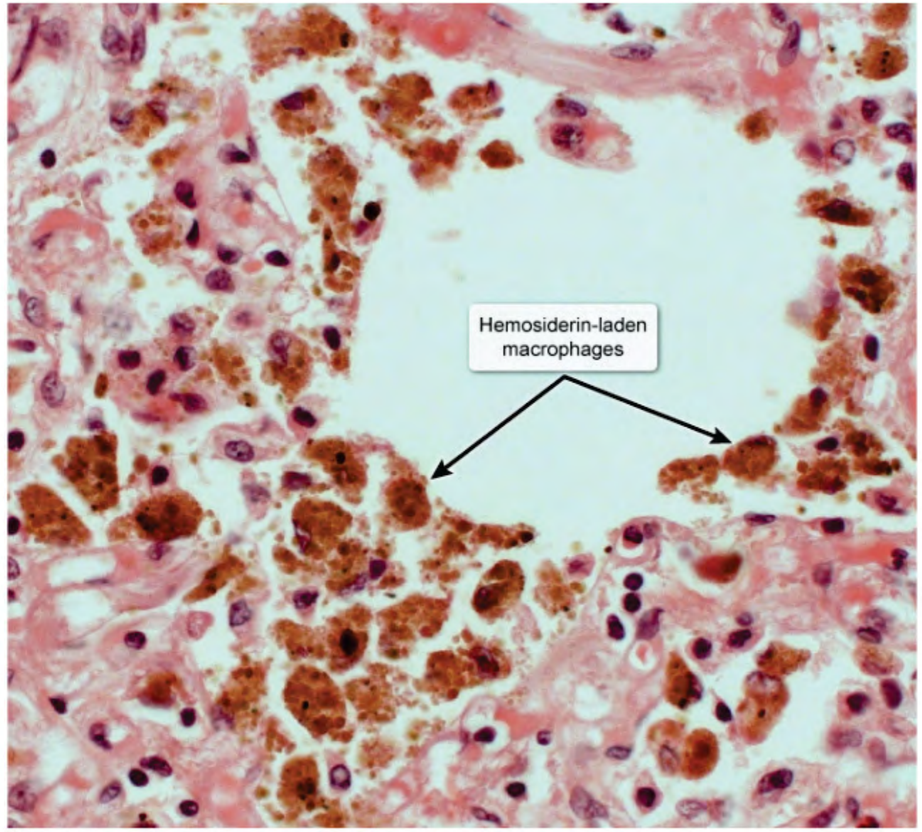
Chronic passive lung congestion



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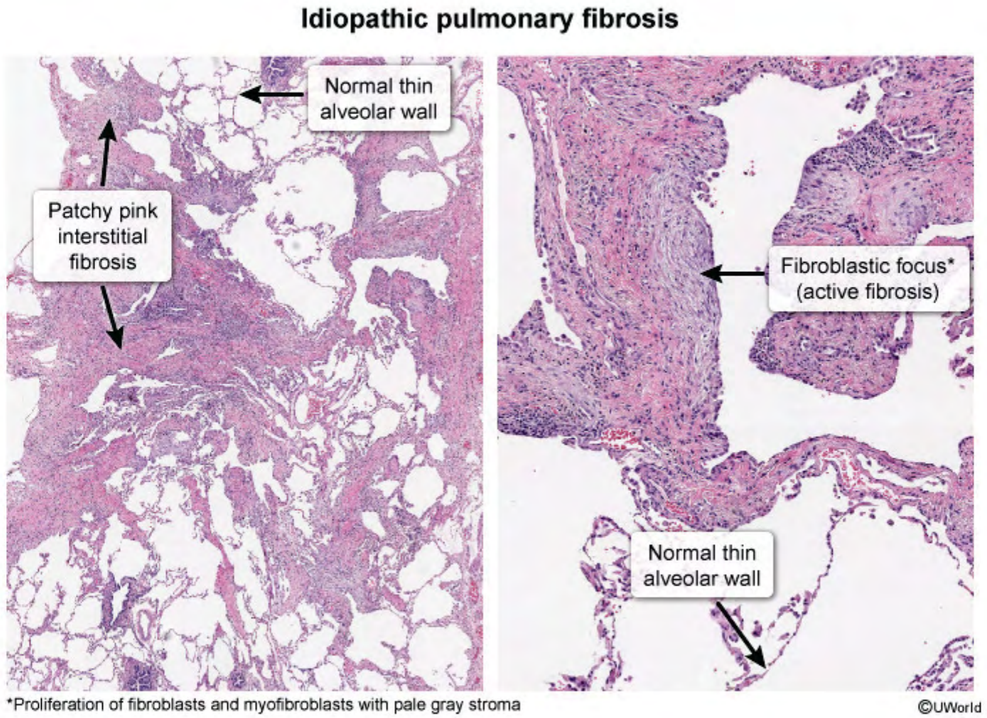

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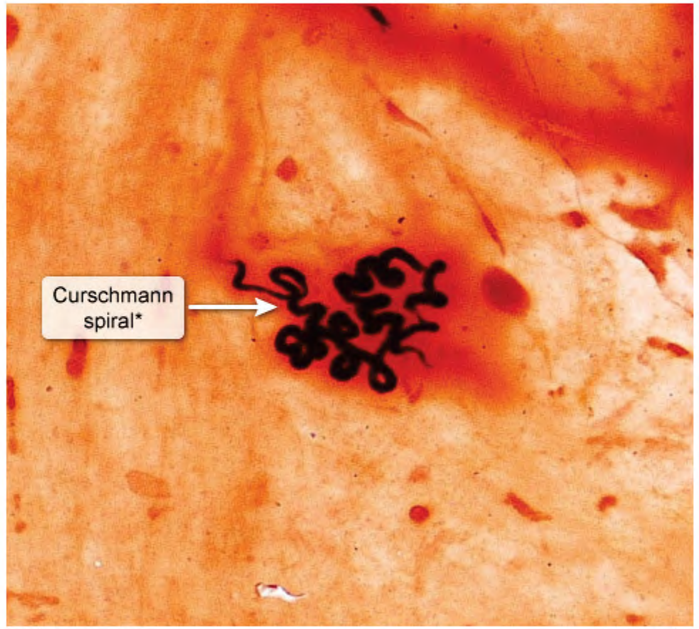
Asthma



*Inspissated mucus

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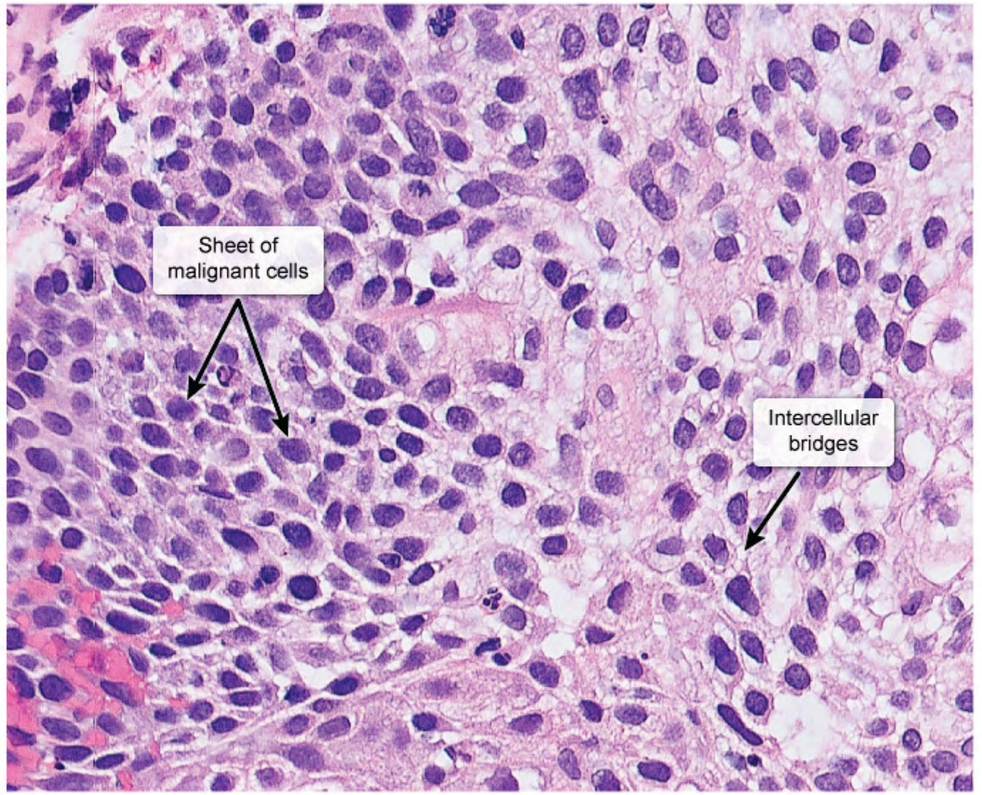
Squamous cell carcinoma



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An 18-year-old man comes to the clinic for a routine physical. The patient has no medical conditions and takes no medications. He does not use tobacco, alcohol, or recreational drugs. He plays on his high school football team. Review of systems is unremarkable. Blood pressure is 120/76 mm Hg, pulse is 68/min, and respirations are 15/min. BMI is 28 kg/m^2. Blood pressures in the upper and lower extremities are equal. There is no jugular venous distension. Carotid upstrokes are brisk. A grade 2/6 midsystolic murmur is present along the left sternal border. The murmur decreases in intensity with handgrip and passive leg elevation. Lung sounds are normal without crackles or wheezes. Echocardiography is most likely to demonstrate which of the following findings?

	Left ventricular mass	Left ventricular cavity size	Left ventricular ejection fraction	Left ventricular relaxation
○ A.	Increased	Dilated	Decreased	Normal
○ B.	Increased	Normal	Preserved	Normal
○ C.	Increased	Small	Decreased	Impaired
○ D.	Increased	Small	Preserved	Impaired
○ E.	Normal	Normal	Preserved	Impaired

Submit

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		Left ventricular mass	Left ventricular cavity size	Left ventricular ejection fraction	Left ventricular relaxation	
✗	A.	Increased	Dilated	Decreased	Normal	(10%)
	B.	Increased	Normal	Preserved	Normal	(16%)
	C.	Increased	Small	Decreased	Impaired	(22%)
✓	D.	Increased	Small	Preserved	Impaired	(47%)
	E.	Normal	Normal	Preserved	Impaired	(3%)

Incorrect
Correct answer
D

47%
Answered correctly

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Explanation

Expected echocardiographic findings				
		LV cavity	LV ejection	LV diastolic

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Explanation

Expected echocardiographic findings				
	LV mass	**LV cavity size**	**LV ejection fraction**	**LV diastolic function**
Dilated cardiomyopathy	Increased	Increased	Decreased	Normal
Hypertrophic cardiomyopathy	Increased	Decreased	Preserved	Impaired
Restrictive cardiomyopathy	Normal	Normal	Preserved	Impaired
Athlete's heart	Increased	Normal or increased	Preserved	Normal
LV = left ventricular.				

This patient with a systolic murmur along the left sternal border that **decreases in intensity** with maneuvers that **increase left ventricular (LV) blood volume** likely has **hypertrophic cardiomyopathy** (HCM). HCM typically involves hypertrophy of the interventricular septum that can partially obstruct LV outflow and cause a murmur. Increases in LV blood volume, caused by maneuvers that increase preload (eg, passive leg elevation) or afterload (eg, handgrip), alleviate LV outflow obstruction and decrease the intensity of the murmur.

Characteristic findings in HCM include **increased LV muscle mass** and a **small LV cavity**. Because LV contractility is typically intact and outflow obstruction is dynamic and commonly present only with exercise, there is usually **preserved ejection fraction** (EF) at rest (ie, carotid pulses are full). Diastolic dysfunction is typically present due to **impaired relaxation** of the hypertrophied LV wall.

Affected patients can be asymptomatic or may have exertional symptoms, depending on the level of phenotypic

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LV = left ventricular.

This patient with a systolic murmur along the left sternal border that **decreases in intensity** with maneuvers that **increase left ventricular (LV) blood volume** likely has **hypertrophic cardiomyopathy** (HCM). HCM typically involves hypertrophy of the interventricular septum that can partially obstruct LV outflow and cause a murmur. Increases in LV blood volume, caused by maneuvers that increase preload (eg, passive leg elevation) or afterload (eg, handgrip), alleviate LV outflow obstruction and decrease the intensity of the murmur.

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Affected patients can be asymptomatic or may have exertional symptoms, depending on the level of phenotypic expression and degree of LV outflow tract obstruction.

(Choices A and E) Increased LV mass with a dilated LV cavity, decreased EF, and normal LV relaxation are consistent with dilated cardiomyopathy. Normal LV mass and cavity size with preserved EF and impaired relaxation describe restrictive cardiomyopathy; the LV walls are stiff but not typically thickened. Both conditions are likely to lead to symptomatic heart failure with jugular venous distension and pulmonary edema (evidenced by crackles on lung auscultation).

(Choice B) Increased LV mass with normal relaxation and normal (or somewhat increased) LV cavity size and ejection fraction are consistent with athlete's heart, in which physiologic cardiac changes (eg, LV cavity enlargement and wall thickening) improve cardiac function in response to intense endurance training. A systolic flow murmur may be heard but does not decrease in intensity with maneuvers that increase LV blood volume.

(Choice C) Increased LV mass with small LV cavity size, decreased EF, and impaired relaxation occur in severe aortic stenosis. The fixed obstruction created by the stenotic valve leads to pressure overload and concentric LV hypertrophy, and also restricts stroke volume to decrease EF. The murmur of aortic stenosis is similar to that of HCM but is differentiated by a decrease in intensity with maneuvers that decrease, rather than increase, LV preload (eg, abrupt standing, Valsalva strain phase).

Educational objective:

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Characteristic findings in HCM include **increased LV muscle mass** and a **small LV cavity**. Because LV contractility is typically intact and outflow obstruction is dynamic and commonly present only with exercise, there is usually **preserved ejection fraction** (EF) at rest (ie, carotid pulses are full). Diastolic dysfunction is typically present due to **impaired relaxation** of the hypertrophied LV wall.

Affected patients can be asymptomatic or may have exertional symptoms, depending on the level of phenotypic expression and degree of LV outflow tract obstruction.

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Educational objective:
Hypertrophic cardiomyopathy (HCM) typically involves interventricular septal hypertrophy that obstructs left ventricular (LV) outflow and creates a systolic murmur that decreases in intensity with maneuvers that increase LV blood volume (eg, handgrip, passive leg elevation). HCM is characterized by increased LV muscle mass with a small LV cavity, preserved ejection fraction, and impaired relaxation leading to diastolic dysfunction.

Physiology	Cardiovascular System	Hypertrophic cardiomyopathy
Subject	System	Topic

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Athlete's	Increased	Normal or	Preserved	Normal

Exhibit Display

Effect of maneuvers on hypertrophic cardiomyopathy

	Physiologic effect	Left ventricular blood volume	Murmur intensity
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

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A 66-year-old man comes to the office for a routine visit. He has a history of hypertension and osteoarthritis. The patient has smoked a pack of cigarettes daily for 40 years and occasionally drinks 1 or 2 glasses of wine but does not use illicit drugs. Blood pressure is 142/82 mm Hg and pulse is 80/min. Cardiopulmonary examination is normal. There is a pulsating, central abdominal mass on physical examination. Which of the following pathologic conditions is the most likely underlying cause of this patient's abnormal findings?

- A. Chronic transmural inflammation
- B. Cystic medial necrosis
- C. Focal intimal tear
- D. Malignant endothelial proliferation
- E. Microbial infection of aortic wall
- F. Vasa vasorum endarteritis

Submit

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- ✓ A. Chronic transmural inflammation (22%)
- B. Cystic medial necrosis (29%)
- C. Focal intimal tear (28%)
- ✗ D. Malignant endothelial proliferation (4%)
- E. Microbial infection of aortic wall (1%)
- F. Vasa vasorum endarteritis (13%)

Incorrect
Correct answer
A

22%
Answered correctly

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Explanation

This patient's pulsating, central abdominal mass most likely represents an **abdominal aortic aneurysm** (AAA), which is a focal dilation of the abdominal aorta that typically occurs **below** the renal arteries.

AAA is associated with several risk factors (eg, age >60, smoking, hypertension, male sex, family history) that lead to oxidative stress, vascular smooth muscle apoptosis, and **chronic transmural inflammation** of the aorta. Inflammatory cells (particularly macrophages) release matrix metalloproteinases and elastases that degrade extracellular matrix components (eg, elastin, collagen), leading to weakening and **progressive expansion** of the

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(Choice B) Cystic medial necrosis is characterized by loss of smooth muscle, collagen, and elastic tissue with formation of cystic mucoid spaces in the aortic media. Patients with Marfan syndrome develop cystic medial degeneration of the aortic root at a young age, predisposing them to ascending aortic aneurysm and dissection.

(Choice C) An intimal tear in the aortic wall is the primary event involved in the pathophysiology of aortic dissection. Patients typically present with tearing chest pain that radiates to the back. Proximal dissections involving the ascending aorta can cause unequal blood pressure in the arms, aortic valve regurgitation (eg, diastolic decrescendo murmur at the right sternal border), and cardiac tamponade.

(Choice D) Malignant endothelial proliferation is characteristic of angiosarcoma, a neoplasm that arises from blood or lymphatic vessels in the subcutaneous tissue and most frequently affects the scalp and face.

(Choice E) Microbial infection (usually bacterial) can cause localized dilation of an artery due to destruction of the vessel wall. Trauma, bacteremic seeding, or septic emboli (mycotic aneurysm) can cause infected aneurysms, which typically present with painful, pulsatile masses and systemic signs such as fever and malaise.

(Choice F) Vasa vasorum endarteritis ("endarteritis obliterans") in the thoracic aorta is thought to be the mechanism by which syphilis causes thoracic aortic aneurysm.

Educational objective:

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Educational objective:
Abdominal aortic aneurysm is associated with risk factors (eg, age >60, smoking, hypertension, male sex, family history) that lead to chronic transmural inflammation and extracellular matrix degradation within the wall of the aorta. This leads to weakening and progressive expansion of the aortic wall, resulting in aneurysm formation, typically below the renal arteries.

References

- Understanding the pathogenesis of abdominal aortic aneurysms.

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A 26-year-old man collapsed while playing basketball and could not be resuscitated. Prior to the episode, he had exertional dyspnea and lightheadedness. Autopsy is performed. The lungs are completely normal. The heart is shown in the exhibit. If the patient had been examined prior to death, which of the following would most likely have been present?

- A. Delayed carotid upstroke
- B. Early systolic murmur at the apex
- C. Mid-diastolic murmur at the apex
- D. S4
- E. Single S2

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A 26-year-old man collapsed while playing basketball and could not be resuscitated. Prior to the episode, he had exertio
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Exhibit Display

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A 26-year-old man collapsed while playing basketball and could not be resuscitated. Prior to the episode, he had exertional dyspnea and lightheadedness. Autopsy is performed. The lungs are completely normal. The heart is shown in the exhibit. If the patient had been examined prior to death, which of the following would most likely have been present?

- A. Delayed carotid upstroke (6%)
- ✗ B. Early systolic murmur at the apex (15%)
- C. Mid-diastolic murmur at the apex (5%)
- ✓ D. S4 (69%)
- E. Single S2 (3%)

Incorrect
Correct answer
D

69%
Answered correctly

41 secs
Time Spent

2023
Version

Explanation

Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain
	• Myocardial disarray & coronary microvascular dysfunction

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Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain
Phenotypic manifestations	• Myocardial disarray & coronary microvascular dysfunction • Asymmetric septal hypertrophy with dynamic LVOT obstruction • LV stiffening with diastolic dysfunction • Increased risk of ventricular arrhythmias & SCD
Clinical presentation	• Often asymptomatic • Exertional dyspnea, angina, palpitations, light-headedness, syncope • Systolic murmur that increases in intensity with ↓ LV blood volume • Audible S4
LV = left ventricular; **LVOT** = left ventricular outflow tract; **SCD** = sudden cardiac death.	

Gross examination of this patient's heart reveals **asymmetric** left ventricular **hypertrophy** (LVH) (ie, wall thickening) that is most prominent in the **interventricular septum**, which is characteristic of **hypertrophic cardiomyopathy** (HCM).

HCM results from genetic mutations that affect cardiac sarcomere proteins; the phenotypic manifestations of the

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HCM results from genetic mutations that affect cardiac sarcomere proteins; the phenotypic manifestations of the disease typically begin during adolescence and involve myocardial disarray and left ventricular (LV) thickening that mostly affects the interventricular septum. The septal thickening commonly leads to dynamic LV outflow tract obstruction that causes a mid-systolic murmur and reduces cardiac output, often leading to exertional symptoms (eg, dyspnea, angina, lightheadedness, syncope). **Sudden cardiac death** (SCD) can also occur.

In addition to obstructing cardiac output, the LVH in HCM also frequently **impairs LV diastolic filling**, with an **S4** commonly heard as blood strikes the thick, stiffened LV wall.

(Choices A and E) A weak and delayed carotid upstroke (ie, pulsus parvus et tardus) as well as a soft and single S2 (caused by delay of aortic valve closure to the point that it occurs simultaneously with pulmonic valve closure) are characteristic of aortic stenosis (AS). Severe AS commonly causes exertional symptoms (eg, dyspnea, lightheadedness) and can cause SCD, but it usually results from age-related calcification and affects older patients (eg, age >40 for a bicuspid aortic valve or older for a congenitally normal valve). Concentric LVH can develop with severe AS; the wall thickening is evenly distributed and does not favor the interventricular septum as it does in HCM.

(Choices B and C) An early systolic murmur at the cardiac apex is consistent with mitral regurgitation (MR), and a mid-diastolic murmur at the cardiac apex is classic for mitral stenosis (MS). Both MR and MS commonly present with heart failure symptoms (eg, dyspnea, orthopnea, peripheral edema), and SCD is relatively uncommon. In addition, asymmetric LVH affecting the interventricular septum does not occur (eccentric LVH can develop with severe MR, and no LVH develops with MS as the left ventricle does not undergo pressure or volume overload).

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Educational objective:
Hypertrophic cardiomyopathy typically involves asymmetric left ventricular hypertrophy (LVH) that is most prominent in the interventricular septum and commonly causes left ventricular (LV) outflow tract obstruction. The LVH also impairs diastolic LV filling, with an S4 commonly heard as blood strikes the thick, stiffened LV wall.

Physiology	Cardiovascular System	Hypertrophic cardiomyopathy
Subject	System	Topic

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blood volume

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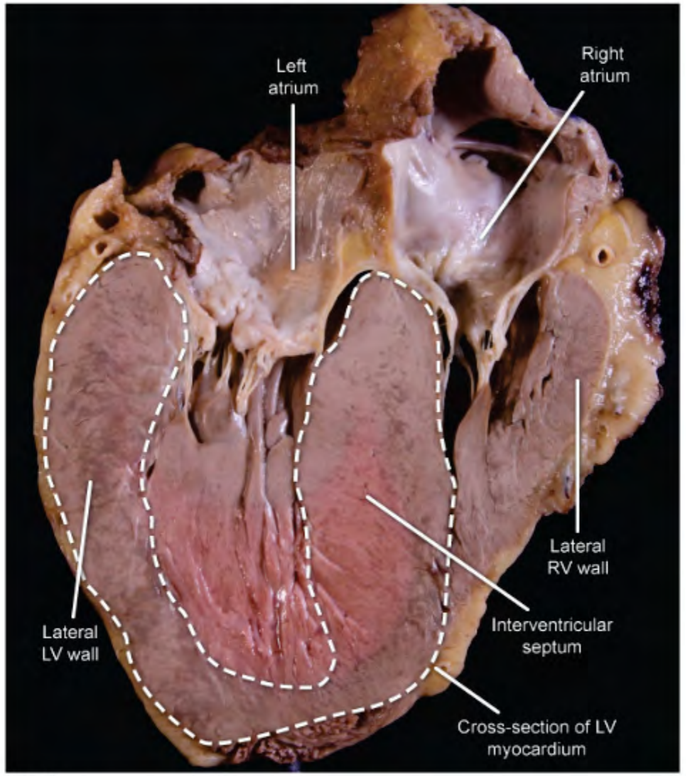
Hypertrophic cardiomyopathy



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presentation
- Systolic murmur that increases in intensity with ↓ EV blood volume

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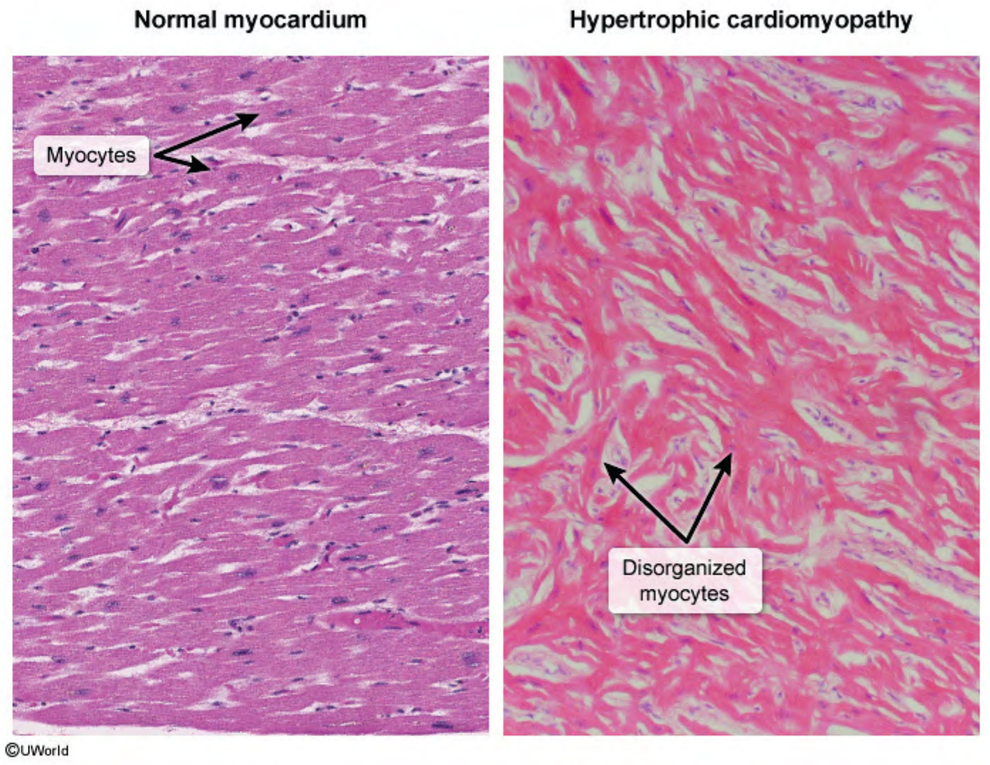

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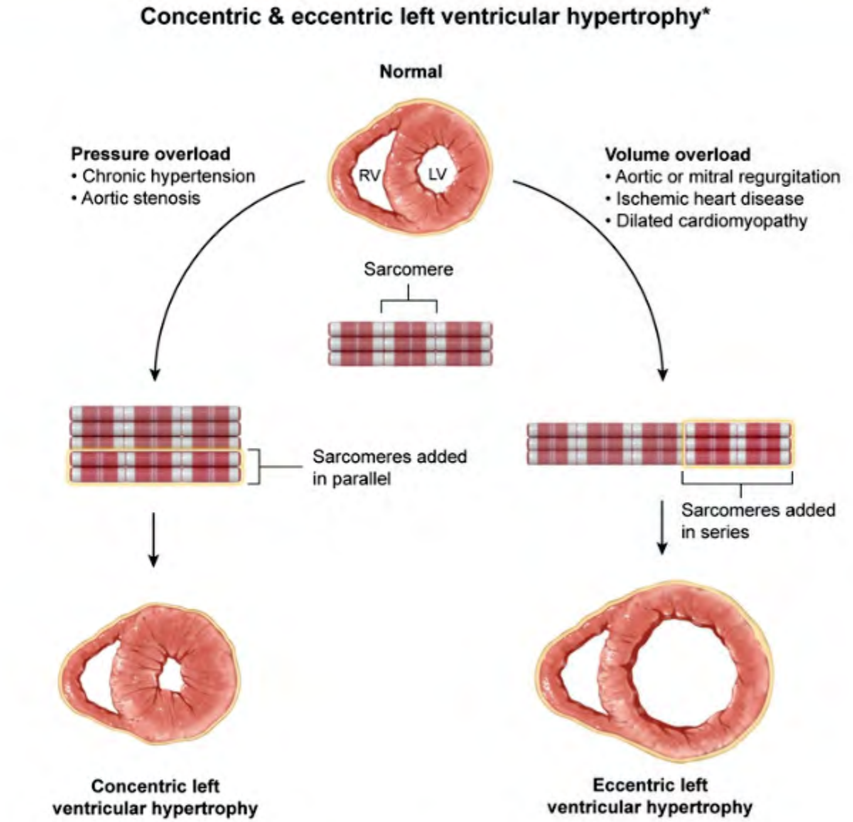

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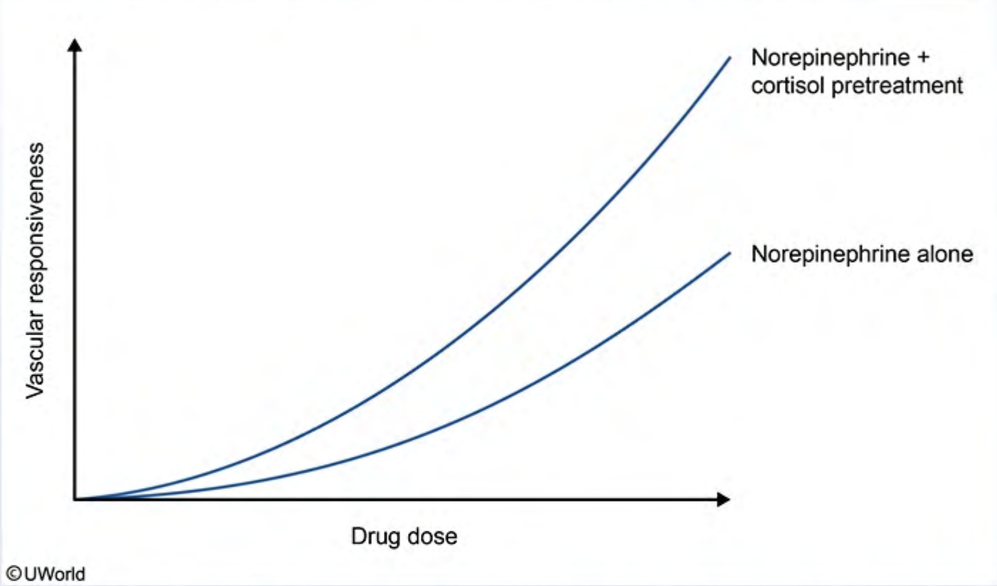
Physiologists conduct a series of experiments on hypophysectomized animals to investigate the effects of cortisol on vascular reactivity. Their initial tests show that administration of cortisol alone does not elicit a vascular response. Next, the researchers measure vascular reactivity to an infusion of norepinephrine both with and without pretreatment with cortisol. The results of their experiments are shown in the graph below.



Which of the following pharmacologic principles best describes the effect of cortisol in this experiment?

- A. Additive effect
- B. Alteration in metabolism
- C. Permissiveness
- D. Synergistic effect
- E. Tachyphylaxis

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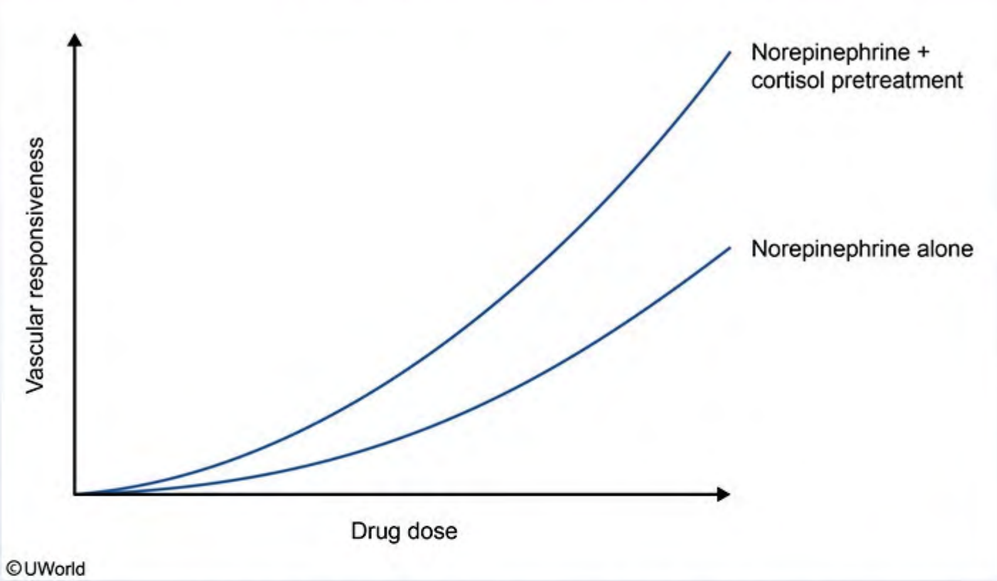
pretreatment with cortisol. The results of their experiments are shown in the graph below.



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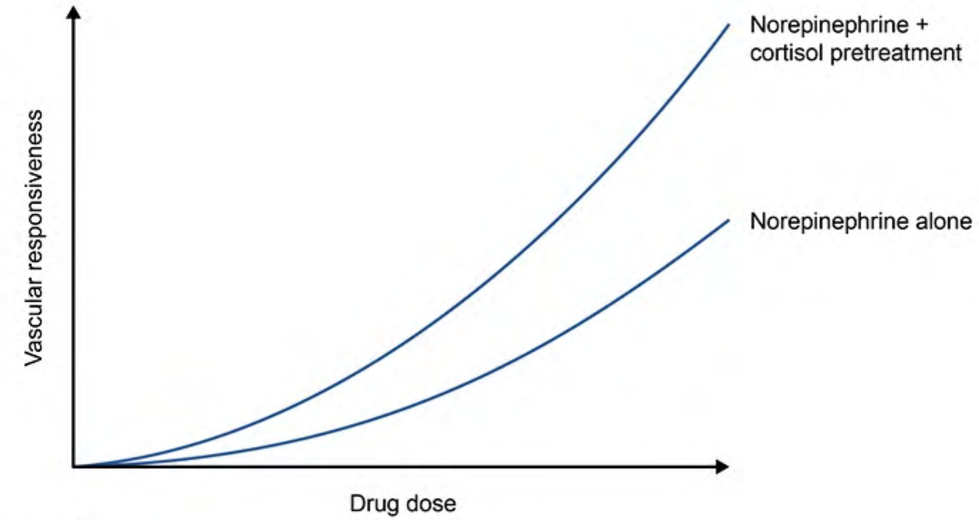

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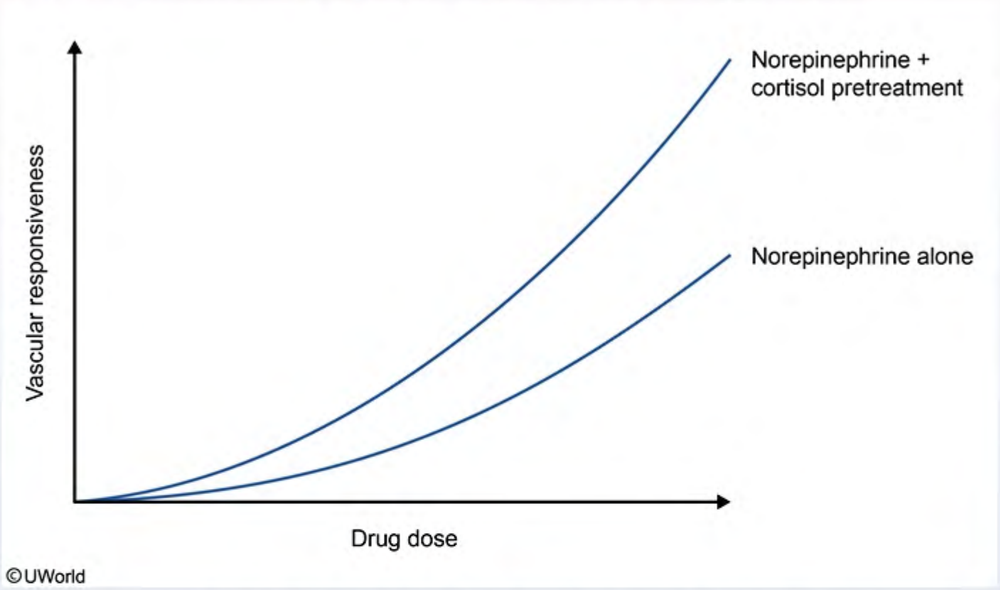

Which of the following pharmacologic principles best describes the effect of cortisol in this experiment?

- A. Additive effect (17%)
- B. Alteration in metabolism (2%)
- ✓ C. Permissiveness (29%)
- ✗ D. Synergistic effect (50%)
- E. Tachyphylaxis (0%)

Incorrect
Correct answer
C

29%
Answered correctly

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Although **cortisol** has no direct vasoactive properties, it **augments the vasoconstrictive effects of catecholamines** and angiotensin II. The above graph shows that the administration of norepinephrine produced a limited degree of vasoconstriction that was markedly increased following pretreatment with cortisol. This phenomenon is termed **permissiveness**; it occurs when a hormone has **no direct effect** on a physiologic process but **allows another hormone to exert its maximal effect** on that process. Cortisol exerts its potentiating effect on vasoconstriction in part through upregulation of alpha-1 adrenergic receptors on vascular smooth muscle cells. In adrenal insufficiency, low cortisol levels can contribute to hypotensive crisis by decreasing vascular responsiveness to norepinephrine and angiotensin II.

Cortisol also has a permissive effect on catecholamine-mediated bronchodilation and glucagon-mediated release of glucose from the liver.

(Choices A and D) The combination of 2 drugs with similar actions can have a synergistic or additive effect. When the combined effect of 2 drugs is equal to the sum of their individual effects, it is termed an additive response (eg, 1 + 1 = 2). When the combined effect exceeds the sum of the individual drugs' effects, the interaction is called synergistic (eg, 1 + 1 > 2). In this case, cortisol's influence on vascular responsiveness is best described as permissive rather than synergistic because cortisol has no intrinsic vasoconstrictive effect (eg, 1 + 0 > 1).

(Choice B) Alterations in metabolism can change a drug's pharmacokinetic and pharmacodynamic properties. For example, certain drugs (eg, amiodarone, fluconazole) can inhibit the P450 cytochrome oxidase system, decreasing the metabolism of other drugs metabolized through this pathway, which can lead to additive or synergistic responses.

(Choice E) Tachyphylaxis describes decreased drug responsiveness in a short period following one or more doses (ie, rapidly developing tolerance). Time is not quantified in the graph; therefore, drug response over time cannot be assessed.

Educational objective:
A permissive hormone has no effect on a physiologic process by itself but allows another hormone to exert its

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Educational objective:
A permissive hormone has no effect on a physiologic process by itself but allows another hormone to exert its maximal effect on that process. Cortisol exerts a permissive effect on catecholamines to potentiate vasoconstriction and bronchodilation; it also has a permissive effect on glucagon to increase glucose release from the liver.

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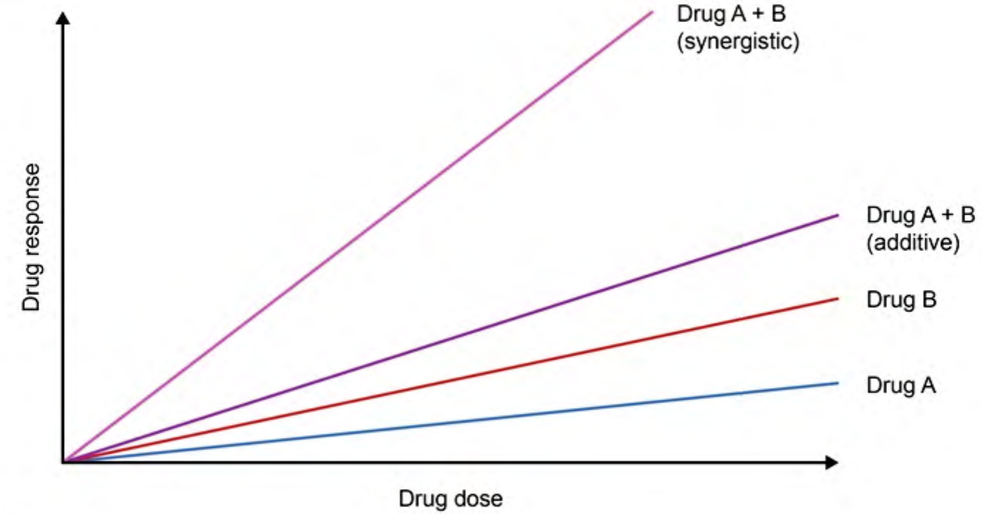
Differences between synergistic and additive response



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A research center studying cardiovascular pathology is conducting trials in which experimental rabbits are fed sweet peas containing substances that alter connective tissue synthesis and maturation. The animals are monitored for several weeks prior to euthanasia. Autopsy shows myxomatous degeneration with pooling of proteoglycans in the media layer and an intact intima layer in large arteries. The pathologic findings seen in the experiment are most similar to which of the following conditions?

- A. Aortic aneurysm
- B. Atherosclerosis
- C. Berry aneurysm
- D. False aneurysm
- E. Giant cell arteritis
- F. Malignant hypertension

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- ✓ A. Aortic aneurysm (48%)
- ✗ B. Atherosclerosis (11%)
- C. Berry aneurysm (6%)
- D. False aneurysm (14%)
- E. Giant cell arteritis (10%)
- F. Malignant hypertension (7%)

Incorrect
Correct answer
A

48%
Answered correctly

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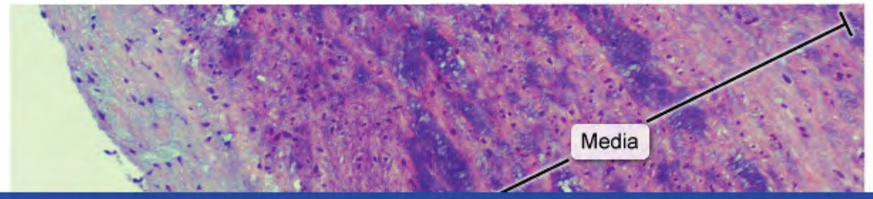
Explanation

Cystic medial degeneration of aorta



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F. Malignant hypertension (7%)

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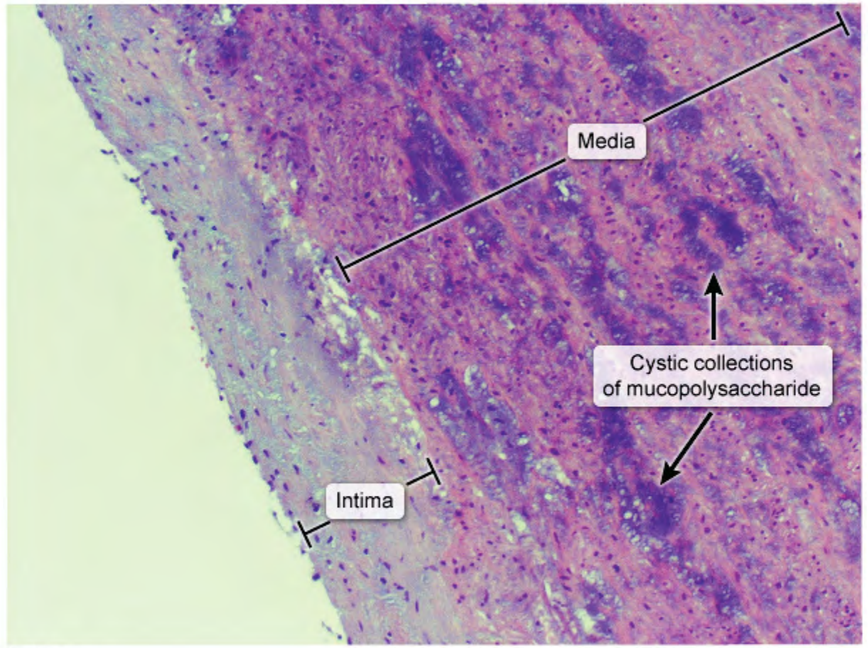
Cystic medial degeneration of aorta



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Myxomatous changes in the media of large arteries is found in **cystic medial degeneration**, a form of pathologic weakening of connective tissue that occurs in large arteries. Medial degeneration is characterized by the

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Myxomatous changes in the media of large arteries is found in **cystic medial degeneration**, a form of pathologic weakening of connective tissue that occurs in large arteries. Medial degeneration is characterized by the fragmentation of elastic tissue and separation of the elastic and fibromuscular components of the tunica media by small, clefted spaces that become filled with amorphous extracellular matrix (ie, mucopolysaccharide).

Marfan syndrome is a frequent cause of cystic medial degeneration in younger patients. It is characterized by an autosomal dominant defect in the extracellular glycoprotein fibrillin-1, which is a major component of the extracellular matrix microfibrils that form the scaffolding for elastic fibers. Mutation of the fibrillin-1 gene results in elastic tissue fragmentation, which predisposes affected patients to **aortic aneurysms** (with an intact intima) and dissections (with an intimal tear).

Beta-aminopropionitrile (a chemical found in certain kinds of sweet peas) causes inhibition of lysyl oxidase, an enzyme responsible for cross-linking elastin fibers and collagen fibers. Ingestion of this compound can cause **connective tissue disruption** in the aorta that mimics the myxomatous degeneration seen in patients with Marfan syndrome.

(Choice B) Atherosclerosis is the formation of intimal atheromas in response to chronic or repetitive endothelial injury. Myxomatous degeneration does not play a significant role in the pathogenesis of atherosclerosis.

(Choice C) The pathogenesis of berry aneurysms is multifactorial. Contributory factors include hemodynamic stress, hypertension, and certain connective tissue disorders. Type IV Ehlers-Danlos syndrome (which results in abnormalities in type III collagen) predisposes patients to the development of berry aneurysms. Myxomatous degeneration is not associated with berry aneurysms.

(Choice D) A false aneurysm, or pseudoaneurysm, is the result of a breach in the continuity of all the layers of a blood vessel, leading to blood leakage outside the vessel wall (eg, hematoma). The intima would not be intact in a false aneurysm.

(Choice E) Giant cell arteritis involves granulomatous inflammation of the media and fragmentation of the internal

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dissections (with an intimal tear).

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(Choice D) A false aneurysm, or pseudoaneurysm, is the result of a breach in the continuity of all the layers of a blood vessel, leading to blood leakage outside the vessel wall (eg, hematoma). The intima would not be intact in a false aneurysm.

(Choice E) Giant cell arteritis involves granulomatous inflammation of the media and fragmentation of the internal elastic lamina, perhaps due to autoimmunity to elastin. It is not related to myxomatous changes of the arterial wall.

(Choice F) Hyperplastic arteriolosclerosis of the renal arterioles is associated with malignant hypertension. It results from concentric lamellar reduplication of the intimal smooth muscle cells ("onion-skinning"). Myxomatous degeneration is not a contributing factor.

Educational objective:
Myxomatous changes, with pooling of mucopolysaccharides in the media layer of large arteries, are found in cystic medial degeneration, which predisposes affected patients to the development of aortic aneurysms. Medial degeneration in younger individuals is frequently due to Marfan syndrome.

References

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Cystic collections

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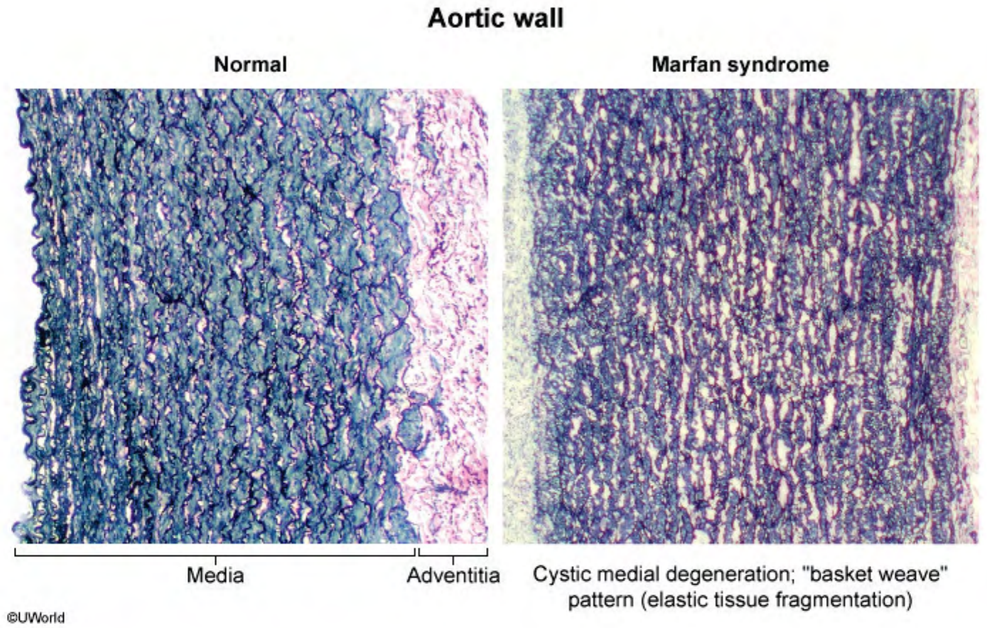

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results from concentric lamellar reduplication of the intimal smooth muscle cells ("onion-skinning"). Myxomatous

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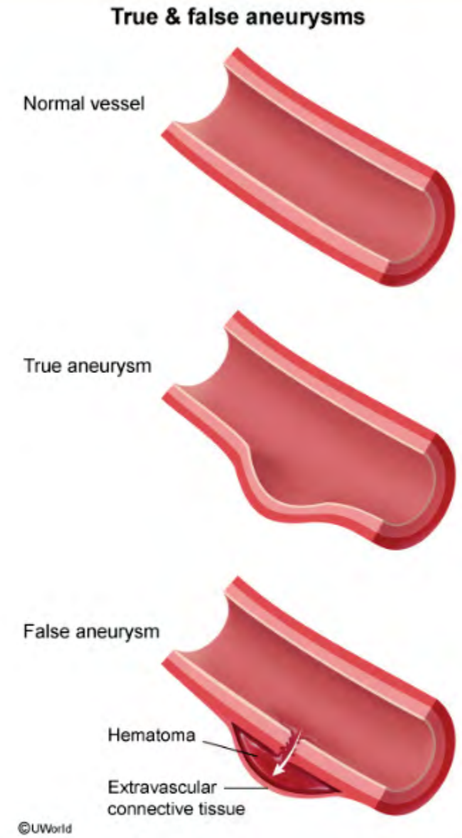

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An 82-year-old woman is sent to the hospital from a nursing home after 2 days of fever, confusion, and lethargy. The patient has a history of mild dementia, hypertension, and osteoarthritis. Her temperature is 38.8 C (102 F), blood pressure is 90/60 mm Hg, and pulse is 116/min. She has mild suprapubic and right costovertebral angle tenderness. Her leukocyte count is 18,000/mm^3, and urinalysis is consistent with acute cystitis. A femoral venous access is planned for administration of intravenous fluids and antibiotics. The femoral artery pulsation is palpable immediately below the inguinal ligament. Which of the following describes the optimal cannulation site for the femoral vein?

- A. Immediately lateral to the femoral artery
- B. Immediately medial to the femoral artery
- C. Midway between the iliac crest and the femoral artery
- D. Superior and medial to the pubic tubercle
- E. Superior to the inguinal ligament

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- ✗ A. Immediately lateral to the femoral artery (16%)
- ✓ B. Immediately medial to the femoral artery (77%)
- C. Midway between the iliac crest and the femoral artery (2%)
- D. Superior and medial to the pubic tubercle (1%)
- E. Superior to the inguinal ligament (1%)

Incorrect
Correct answer
B

77%
Answered correctly

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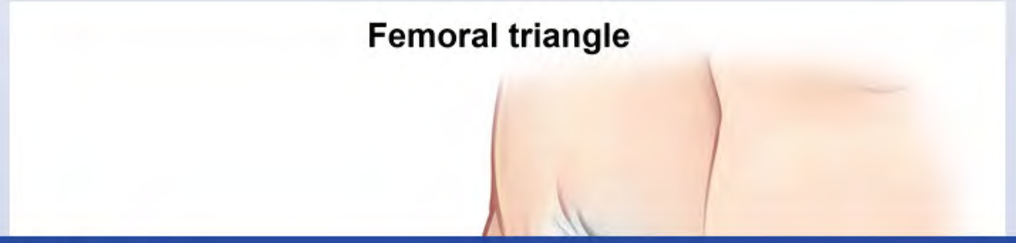
Explanation



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✓ B. Immediately medial to the femoral artery (77%)

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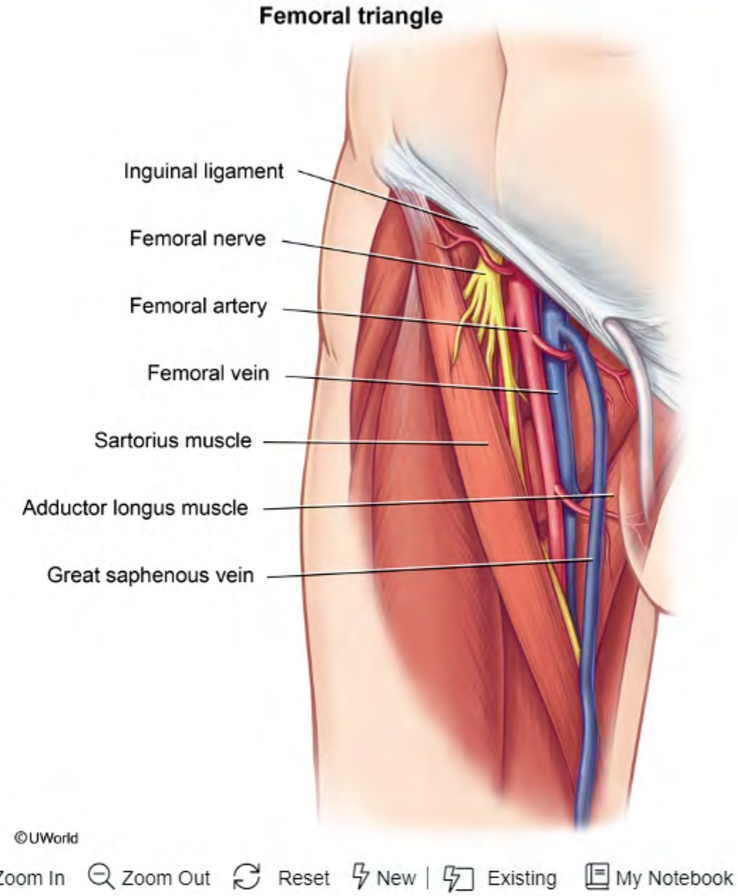

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Sartorius muscle

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Femoral vein catheter placement is usually done in patients with unavailable preferred access sites (eg, subclavian, jugular vein) or those at increased risk of complications (eg, emergency access, uncooperative patient). The femoral vein is the major deep vein of the lower extremity and passes through the femoral triangle beneath the inguinal ligament before entering the pelvis as the external iliac vein. The femoral triangle is a subfascial space in the upper thigh that is bordered by the inguinal ligament (superiorly), adductor longus muscle (medially), and sartorius muscle (laterally).

Within the femoral triangle lies (lateral to medial) the femoral nerve, femoral artery, femoral vein, and deep inguinal nodes/lymphatic vessels. The femoral artery is located at the midinguinal point (midway between the pubic symphysis and anterior superior iliac spine), and the **femoral vein** lies **immediately medial** to the artery. Cannulation of the femoral vein should occur approximately 1 cm below the inguinal ligament and 0.5-1.0 cm medial to the femoral artery pulsation **(Choices C, D, and E).**

(Choice A) The femoral nerve is approximately 1 cm lateral to the femoral artery pulsation and could be damaged by needle insertion at this site.

Educational objective:
The femoral triangle (lateral to medial) consists of the femoral nerve, femoral artery, femoral vein, and deep inguinal nodes/lymphatic vessels. Cannulation of the femoral vein should occur approximately 1 cm below the inguinal ligament and just medial to the femoral artery pulsation.

Anatomy	Cardiovascular System	Central venous catheter
Subject	System	Topic

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A 49-year-old woman comes to the emergency department with a 2-day history of fever, dysuria, and flank pain. The patient has a history of poorly controlled type 2 diabetes mellitus. Temperature is 38.3 C (101 F), blood pressure is 80/44 mm Hg, pulse is 134/min, and respirations are 34/min. On physical examination, the patient appears lethargic, flushed, and diaphoretic. Neck veins are flat. The lungs are clear on auscultation and heart sounds are normal. There is right-sided costovertebral tenderness. Which of the following hemodynamic parameters are most likely to be present in this patient?

	Pulmonary capillary wedge pressure (PCWP)	Cardiac index (CI)	Systemic vascular resistance (SVR)	Mixed venous O2 saturation (SvO2)
○ A.	↑	↓	↑	↓
○ B.	↑	↑	↓	↑
○ C.	↓	↓	↑	↓
○ D.	↓	↓	↓	↓
○ E.	↓	↑	↓	↑

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	Pulmonary capillary wedge pressure (PCWP)	Cardiac index (CI)	Systemic vascular resistance (SVR)	Mixed venous O2 saturation (SvO2)	
✗ A.	↑	↓	↑	↓	(6%)
B.	↑	↑	↓	↑	(9%)
C.	↓	↓	↑	↓	(17%)
D.	↓	↓	↓	↓	(28%)
✓ E.	↓	↑	↓	↑	(37%)

Incorrect
Correct answer
E

37%
Answered correctly

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Explanation

Hemodynamic measurements in shock				
Parameter	Hypovolemic	Cardiogenic	Obstructive	Distributive

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Hemodynamic measurements in shock				
Parameter	**Hypovolemic shock**	**Cardiogenic shock**	**Obstructive shock**	**Distributive shock**
CVP (right-sided preload)	↓	↑	↑	↓
PCWP (left-sided preload)	↓	↑	↓*	↓
Cardiac index (LV output)	↓	↓	↓	↑**
SVR (afterload)	↑	↑	↑	↓
SvO_2	↓	↓	↓	↑**

*In tamponade, left-sided preload is decreased, but measured PCWP is paradoxically increased due to external compression by pericardial fluid.

**Cardiac index & SvO_2 are usually decreased in neurogenic shock due to impaired sympathetic reflexes.

CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **SvO_2** = mixed venous oxygen saturation; **SVR** = systemic vascular resistance.

This patient with fever, dysuria, flank pain, and right costovertebral angle tenderness likely has **pyelonephritis**. In addition, her **hypotension** in the setting of infection suggests **septic shock**. Septic shock is the most common type of distributive shock; other types include anaphylactic shock and neurogenic shock.

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This patient with fever, dysuria, flank pain, and right costovertebral angle tenderness likely has **pyelonephritis**. In addition, her **hypotension** in the setting of infection suggests **septic shock**. Septic shock is the most common type of distributive shock; other types include anaphylactic shock and neurogenic shock.

The primary disturbance in septic shock is **widespread vasodilation** leading to **decreased systemic vascular resistance** (SVR). Increased vascular permeability (third-spacing) and peripheral venous dilation also cause a reduction in central venous pressure and pulmonary capillary wedge pressure (PCWP) **(Choice B)**. Initially, the hypotension and inflammation from sepsis trigger a compensatory increase in sympathetic drive that increases heart rate and myocardial contractility, leading to **increased cardiac index** (cardiac output per body surface area). The high blood flow rates prevent complete extraction of oxygen by the tissues, resulting in **high mixed venous oxygen saturation**. As shock progresses, tissue ischemia and accumulation of cytotoxic mediators eventually cause a reduction in cardiac output with end-organ dysfunction.

(Choice A) Low cardiac index with high PCWP is consistent with cardiogenic shock. The primary disturbance is typically left ventricular failure with a compensatory increase in SVR in an effort to maintain adequate blood pressure for organ perfusion.

(Choice C) Low PCWP with low cardiac index and high SVR occurs in both hypovolemic shock and some types of obstructive shock (eg, pulmonary embolism, tension pneumothorax). Central venous pressure is low in hypovolemic shock and high in obstructive shock.

(Choice D) Low PCWP with low cardiac index and low SVR occurs in neurogenic shock. As in sepsis, the primary disturbance is peripheral vasodilation. However, due to nerve injury there is no sympathetic-mediated increase in heart rate and myocardial contractility; therefore, cardiac output is low. These parameters are also seen in the late stages of septic shock; however, this patient's flushing is consistent with the high cardiac output seen in early septic shock.

Educational objective:
Septic shock causes widespread arteriolar vasodilation, which leads to a decrease in systemic vascular resistance and a compensatory increase in cardiac output. Central venous pressure and pulmonary capillary wedge pressure

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The primary disturbance in septic shock is **widespread vasodilation** leading to **decreased systemic vascular resistance** (SVR). Increased vascular permeability (third-spacing) and peripheral venous dilation also cause a reduction in central venous pressure and pulmonary capillary wedge pressure (PCWP) **(Choice B)**. Initially, the hypotension and inflammation from sepsis trigger a compensatory increase in sympathetic drive that increases heart rate and myocardial contractility, leading to **increased cardiac index** (cardiac output per body surface area). The high blood flow rates prevent complete extraction of oxygen by the tissues, resulting in **high mixed venous oxygen saturation**. As shock progresses, tissue ischemia and accumulation of cytotoxic mediators eventually cause a reduction in cardiac output with end-organ dysfunction.

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Educational objective:
Septic shock causes widespread arteriolar vasodilation, which leads to a decrease in systemic vascular resistance and a compensatory increase in cardiac output. Central venous pressure and pulmonary capillary wedge pressure are also decreased due to pooling of blood in the dilated veins. Increased flow rates through the peripheral capillaries lead to incomplete oxygen extraction by the tissues and high mixed venous oxygen saturation.

References

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Cardiac index & SvO_2 are usually decreased in hypogenic shock due to impaired sympathetic reflexes.

CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **SvO_2** = mixed ve

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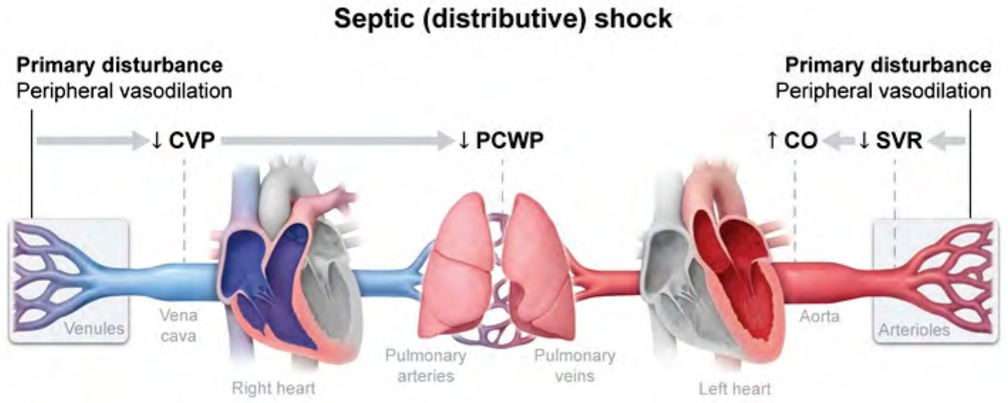

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Cardiac index & SvO_2 are usually decreased in hypovolemic shock due to impaired sympathetic reflexes.

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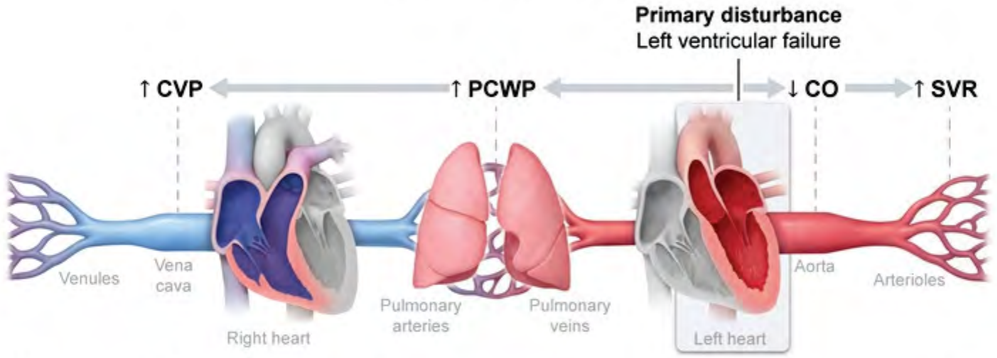
Cardiogenic shock



CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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Cardiac index & SvO_2 are usually decreased in hypogenic shock due to impaired sympathetic reflexes.

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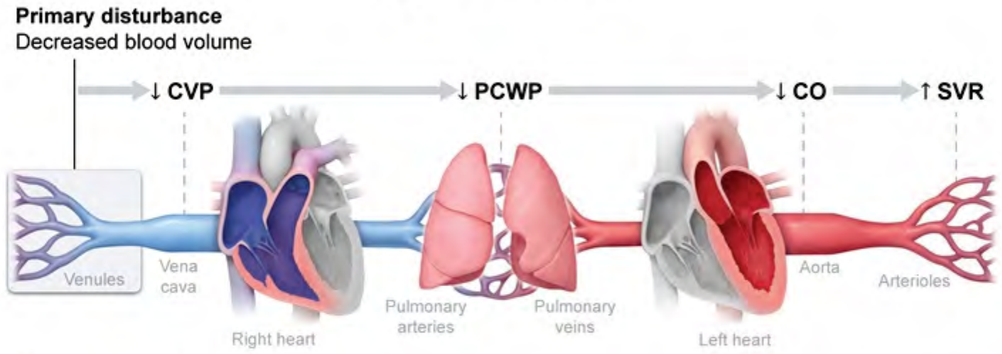

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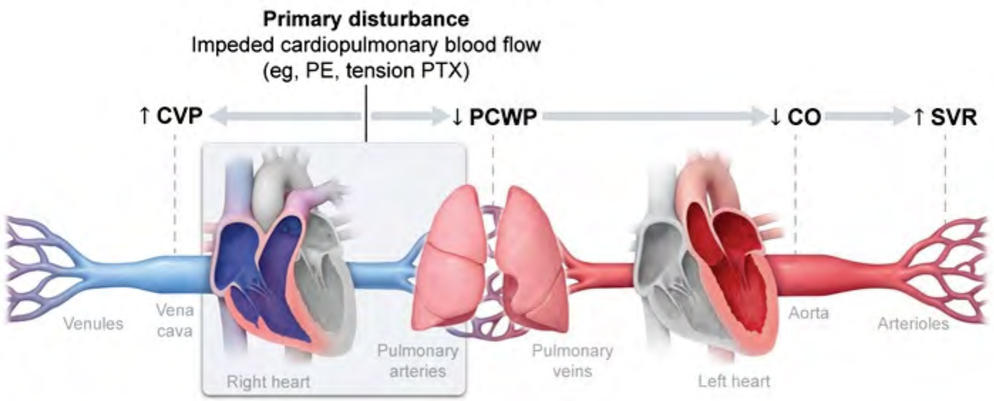

CO = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **PE** = pulmonary embolism; **PTX** = pneumothorax; **SVR** = systemic vascular resistance.

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A 78-year-old man is admitted to the hospital due to fatigue, orthopnea, and exertional dyspnea. The patient takes no medications and has not seen a physician in many years. He is a lifelong nonsmoker. Blood pressure is 110/60 mm Hg and pulse is 64/min. Examination reveals bibasilar crackles, elevated jugular venous pressure, and bilateral lower extremity pitting edema. Left ventricular diastolic pressure-volume curve is shown in the exhibit (purple curve). Which of the following is the most likely cause of this patient's condition?

- A. Alcohol use
- B. Doxorubicin therapy
- C. Selenium deficiency
- D. Transthyretin deposition
- E. Viral myocarditis

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A 78-year-old man is admitted to the hospital due to fatigue, orthopnea, and exertional dyspnea. The patient takes
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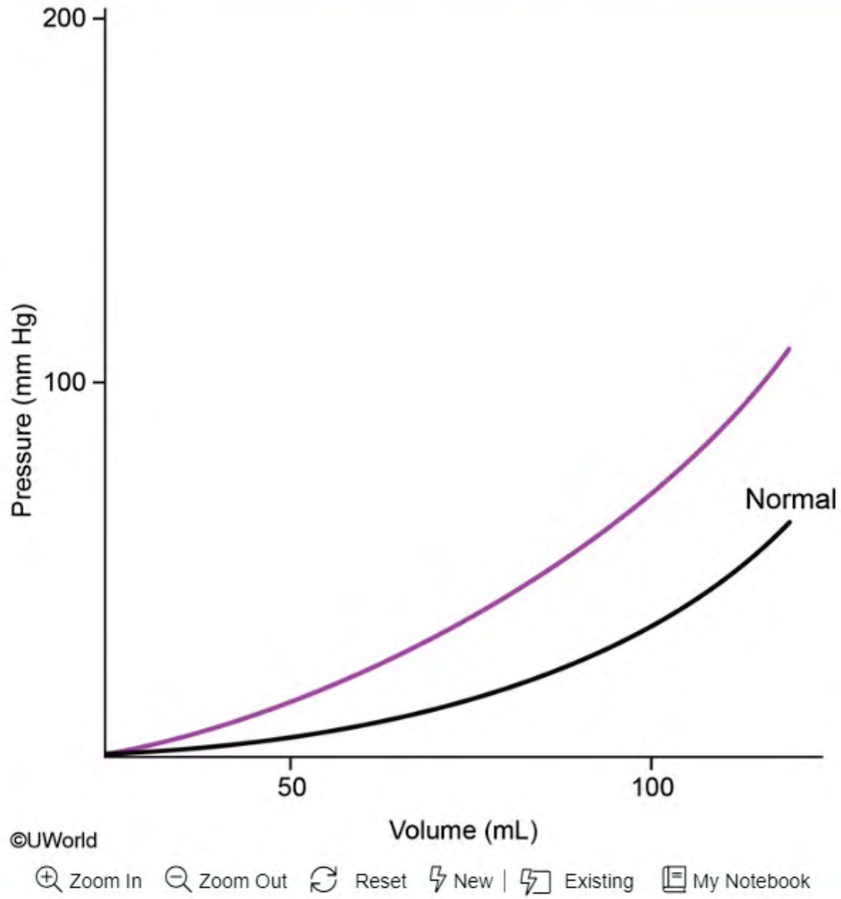

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- A. Alcohol use (16%)
- B. Doxorubicin therapy (15%)
- ✗ C. Selenium deficiency (3%)
- ✓ D. Transthyretin deposition (48%)
- E. Viral myocarditis (16%)

Incorrect
Correct answer
D

48%
Answered correctly

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Time Spent

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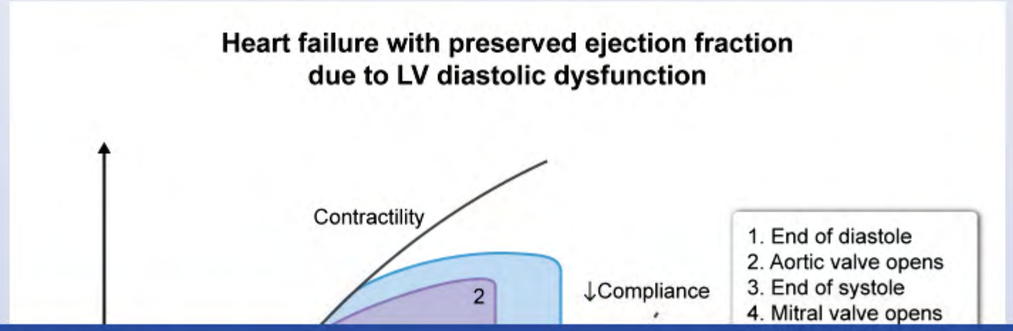
Version

Explanation



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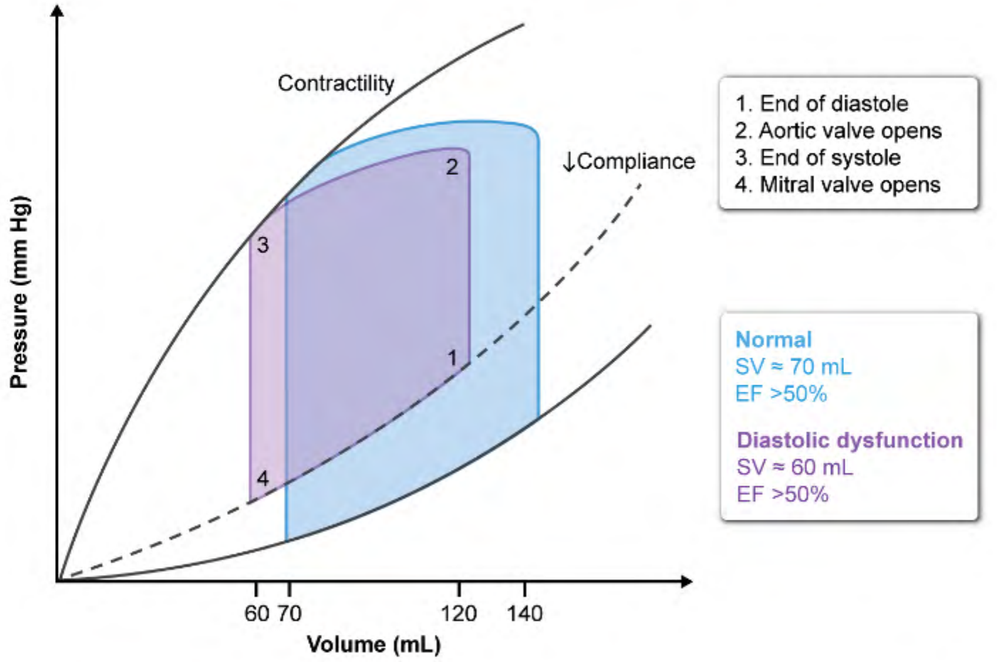
✓ D. Transthyretin deposition (48%)

Exhibit Display

Heart failure with preserved ejection fraction due to LV diastolic dysfunction



EF = ejection fraction; **LV** = left ventricular; **SV** = stroke volume.

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Volume (mL)

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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This patient's fatigue, exertional dyspnea, and orthopnea as well as lung crackles, jugular venous distension (JVD), and lower extremity edema on physical examination are consistent with **decompensated heart failure**. The left ventricular (LV) diastolic pressure-volume curve shows elevated pressure at any given LV volume. This finding indicates **reduced LV compliance** (ie, diastolic dysfunction), as occurs in conditions causing heart failure with preserved ejection fraction (eg, hypertensive heart disease, **restrictive cardiomyopathy**).

Cardiac amyloidosis is a common cause of restrictive cardiomyopathy resulting from abnormal accumulation of amyloid proteins, particularly immunoglobulin light chains or **transthyretin** (protein carrier of thyroxine and retinol). Transthyretin amyloidosis most often affects the heart of elderly patients and lead to an **infiltrative** restrictive cardiomyopathy. The **ventricular walls** become **thickened and stiff** and unable to relax during diastole; higher pressures are required to fill the ventricle. Elevated hydrostatic pressure backs up into the lungs (pulmonary edema) and into the right side of the heart and systemic venous system (JVD and peripheral edema).

LV contractility is intact with infiltrative restrictive cardiomyopathy (especially early in the disease process), but it can decrease as the disease progresses, eventually leading to LV cavity dilation and a picture consistent with dilated cardiomyopathy.

(Choices A, B, C, and E) Excessive alcohol use, doxorubicin chemotherapy, selenium deficiency, and viral myocarditis are all potential causes of dilated cardiomyopathy; they do not cause restrictive cardiomyopathy. Dilated cardiomyopathy is characterized by LV cavity enlargement and reduced LV contractility leading to systolic dysfunction with reduced ejection fraction. LV wall compliance is typically normal or increased.

Educational objective:
Reduced ventricular compliance is seen with restrictive cardiomyopathy and other causes of diastolic dysfunction and heart failure with preserved ejection fraction (eg, hypertensive heart disease). It involves ventricular wall thickening and/or stiffening that leads to increased diastolic filling pressure at any given ventricular volume.

References

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EF = ejection fraction; LV = left ventricular; SV = stroke volume

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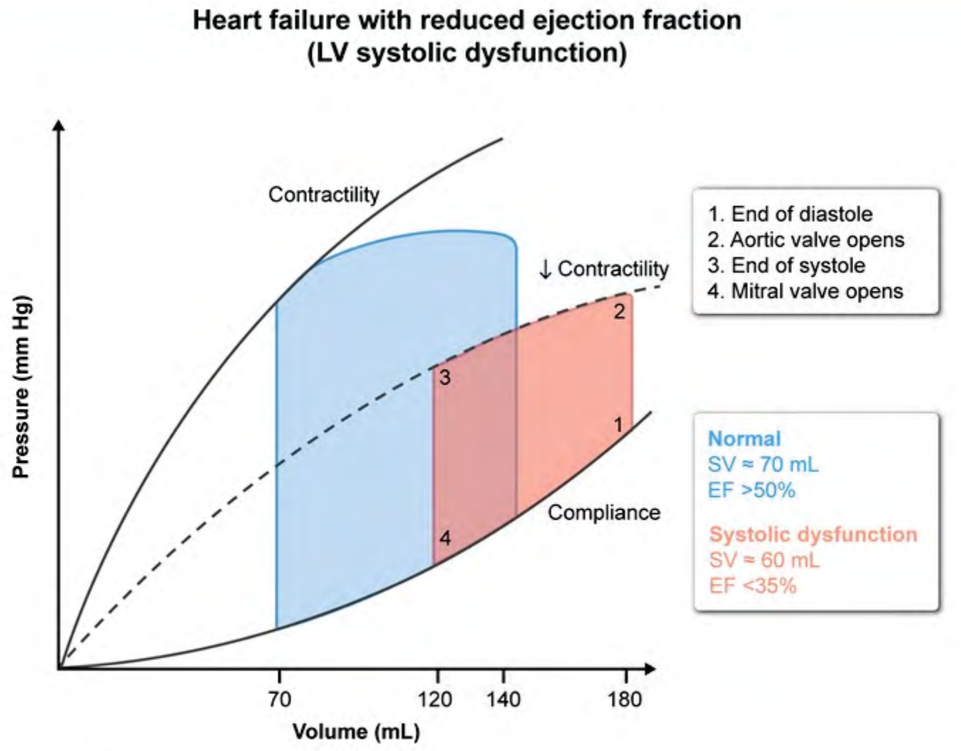

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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A 5-week-old boy is receiving care in the neonatal intensive care unit. The patient was born at 26 weeks gestation with a birth weight of 0.79 kg (1 lb 10 oz). Echocardiography reveals a patent ductus arteriosus and a dilated left atrium. The patient has moderate pulmonary edema. During surgical ligation of the ductus arteriosus, the surgeons perform a left-sided thoracotomy, visualize the ductus, and place a small metal clamp across the vessel. Which of the following changes is most likely to take place immediately after ligation?

- A. Decreased right ventricular output
- B. Decreased right ventricular preload
- C. Increased left ventricular afterload
- D. Increased left ventricular output
- E. Increased right ventricular afterload

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- A. Decreased right ventricular output (5%)
- ✗ B. Decreased right ventricular preload (12%)
- ✓ C. Increased left ventricular afterload (49%)
- D. Increased left ventricular output (17%)
- E. Increased right ventricular afterload (15%)

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Correct answer
C

49%
Answered correctly

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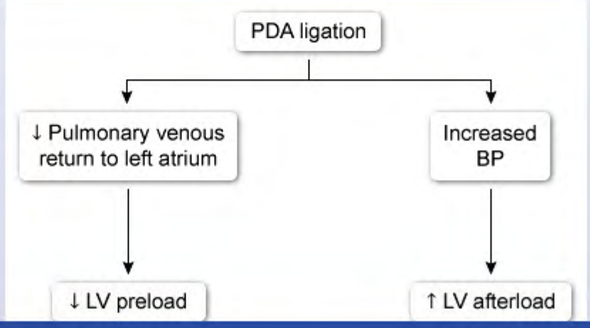
Explanation



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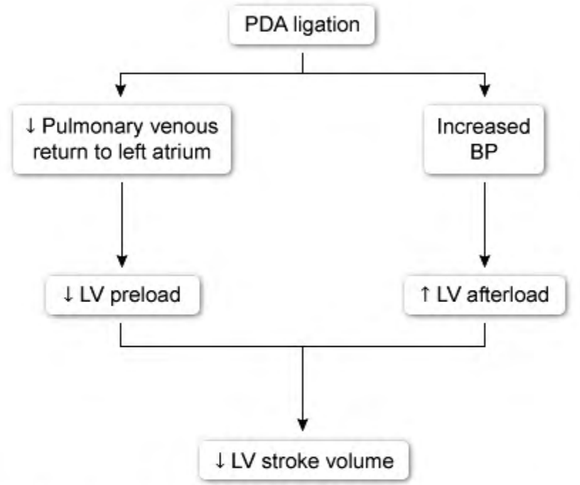

BP = blood pressure; **LV** = left ventricular;
PDA = patent ductus arteriosus.

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↓ LV preload
↑ LV afterload

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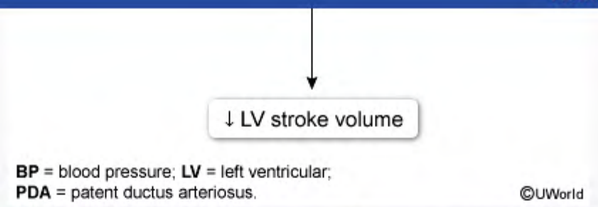

A **patent ductus arteriosus** (PDA) allows blood to flow from the aorta into the relatively low-pressure pulmonary artery. When a PDA persists longer than the initial postnatal period (eg, >48 hours), the **left-to-right shunt** causes overcirculation of the lungs and left side of the heart, which can ultimately lead to left-sided heart failure and pulmonary edema. Closure of a persistent PDA can be achieved with a medication that inhibits prostaglandin synthesis (eg, indomethacin, ibuprofen) or by **surgical ligation**.

When the PDA shunt is ablated during surgery, there is an immediate reduction in pulmonary venous return to the left atrium (ie, **decreased LV preload**) and increased diastolic blood pressure in the aorta (ie, **increased LV afterload**). The combination of these two immediate changes can result in an acute decrease in LV stroke volume and cardiac output **(Choice D)**.

(Choices A, B, and E) In patients with a left-to-right shunt through a PDA, the increased volume load is handled entirely by the left ventricle. The right ventricle receives deoxygenated blood from the body via the superior and inferior vena cava and pumps that blood to the pulmonary artery. This does not change when the PDA is closed, so there are no postligation changes to preload, afterload, or cardiac output of the right ventricle.

Educational objective:
Closure of a patent ductus arteriosus results in decreased pulmonary venous return to the left atrium (decreased left ventricular preload) and increased diastolic blood pressure in the aorta (increased left ventricular afterload). Together, these changes acutely lower left ventricular stroke volume and cardiac output.

Pathophysiology	Cardiovascular System	Patent ductus arteriosus
Subject	System	Topic

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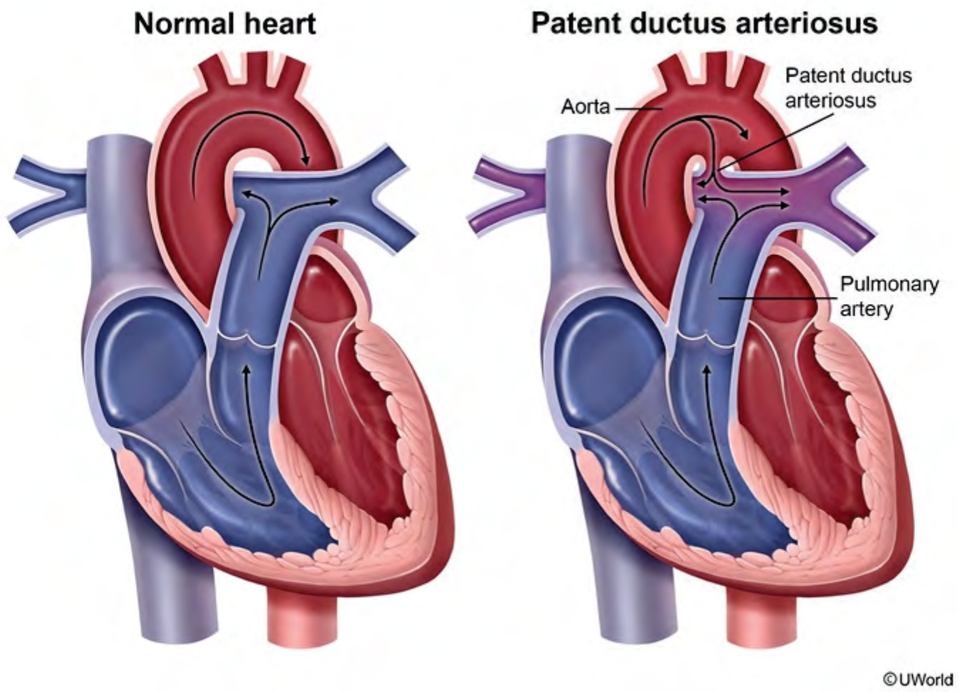

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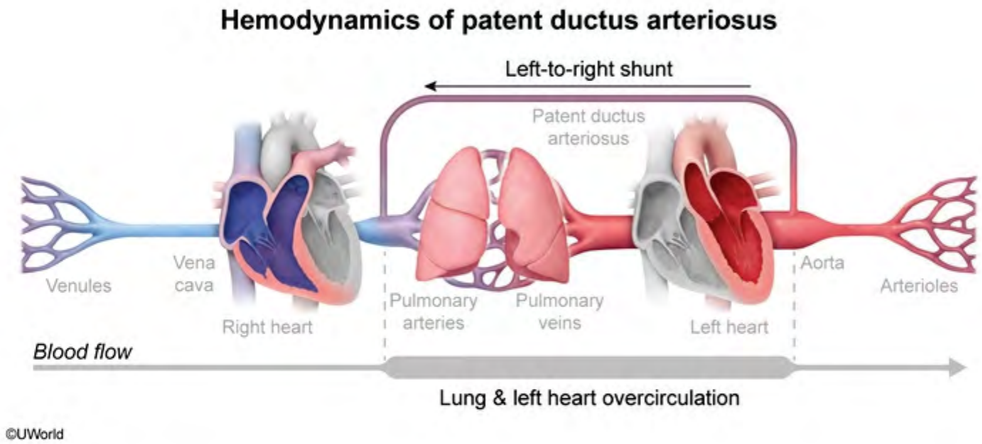

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A 65-year-old immigrant from Eastern Europe comes to the physician because of a four-week history of low-grade fever, multiple joint pains, and a well-demarcated erythematous rash on his face and trunk. The patient denies hair loss, mucosal ulceration, or photosensitivity. His serum test is positive for the presence of anti-nuclear antibody (ANA) in very high titers. The patient reports a past medical history of coronary artery disease, congestive heart failure, and "cardiac rhythm abnormalities." This patient should be specifically questioned about the intake of which of the following medications?

- A. Amiodarone
- B. Propranolol
- C. Lidocaine
- D. Procainamide
- E. Verapamil
- F. Adenosine

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A 65-year-old immigrant from Eastern Europe comes to the physician because of a four-week history of low-grade fever, multiple joint pains, and a well-demarcated erythematous rash on his face and trunk. The patient denies hair loss, mucosal ulceration, or photosensitivity. His serum test is positive for the presence of anti-nuclear antibody (ANA) in very high titers. The patient reports a past medical history of coronary artery disease, congestive heart failure, and "cardiac rhythm abnormalities." This patient should be specifically questioned about the intake of which of the following medications?

- A. Amiodarone (14%)
- ✗ B. Propranolol (1%)
- C. Lidocaine (1%)
- ✓ D. Procainamide (76%)
- E. Verapamil (3%)
- F. Adenosine (2%)

Incorrect
Correct answer
D

76%
Answered correctly

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Explanation

Features of drug-induced lupus erythematosus
• New onset lupus symptoms • Anti-nuclear antibodies (ANA) and **anti-histone antibodies** present in > 95% of cases • Anti-dsDNA antibodies are rarely seen

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Features of drug-induced lupus erythematosus

• New onset lupus symptoms
• Anti-nuclear antibodies (ANA) and **anti-histone antibodies** present in > 95% of cases
• Anti-dsDNA antibodies are rarely seen
• Exposure to implicated drugs
  ◦ **Hydralazine**
  ◦ **Procainamide**
  ◦ **Isoniazid**
  ◦ Minocycline
  ◦ Quinidine

Drug-induced lupus erythematosus (DILE) should be considered in patients presenting with signs and symptoms of systemic lupus erythematosus (SLE). Both hydralazine and procainamide are categorized as high-risk drugs for the development of DILE. Procainamide is metabolized via hepatic acetylation. Individuals who are "slow acetylators" are at greatest risk for DILE, while individuals who metabolize the drug more rapidly are less likely to experience this adverse effect. Drug-induced lupus erythematosus occurs in up to 20% of patients taking procainamide.

The presence of autoantibodies can be used to help differentiate DILE from SLE. Although anti-histone antibodies are very common in DILE, they are seen in only 50% of SLE patients. Likewise, anti-dsDNA antibodies are present in 80% of SLE patients, but are rarely seen in DILE. Anti-nuclear antibodies are frequently positive in both diseases.

(Choice A) The most notable side effects of amiodarone include thyroid dysfunction, lung fibrosis, and liver toxicity, as well as a blue or grey discoloration of the skin.

(Choice B) The most frequently encountered adverse effects of nonselective beta-blockers are negative inotropy

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experience this adverse effect. Drug-induced lupus erythematosus occurs in up to 20% of patients taking procainamide.

The presence of autoantibodies can be used to help differentiate DILE from SLE. Although anti-histone antibodies are very common in DILE, they are seen in only 50% of SLE patients. Likewise, anti-dsDNA antibodies are present in 80% of SLE patients, but are rarely seen in DILE. Anti-nuclear antibodies are frequently positive in both diseases.

(Choice A) The most notable side effects of amiodarone include thyroid dysfunction, lung fibrosis, and liver toxicity, as well as a blue or grey discoloration of the skin.

(Choice B) The most frequently encountered adverse effects of nonselective beta-blockers are negative inotropy and bronchoconstriction in patients with asthma or COPD.

(Choice C) The adverse effects of lidocaine are characteristically nonfocal neurologic signs such as tremor, drowsiness, and change in mental status, although lidocaine can also lead to generalized seizures.

(Choice E) Verapamil is a calcium channel blocker and class 4 antiarrhythmic. The most frequently noted adverse reactions include negative inotropy, constipation, and gingival hyperplasia.

(Choice F) Adenosine is used as a vasodilator in chemical cardiac stress tests and as a fast acting antiarrhythmic for stopping acute supraventricular tachycardias. Adenosine has an extremely short half-life, and its immediate adverse effects include chest burning, flushing, and transient hypotension.

Educational objective:
Procainamide and hydralazine have the highest risk of causing drug-induced lupus erythematosus (DILE), which is characterized by the development of lupus-like symptoms in addition to positive ANA and anti-histone antibodies. Unlike with SLE, anti-dsDNA antibodies are rarely seen.

References

- Drug-induced lupus.
- Drug-induced lupus erythematosus: incidence, management and prevention.

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A 45-year-old woman participates in a cardiovascular health study. She undergoes continuous 24-hour ECG monitoring with a wearable device. A few instances of the following pattern were found on her ECG:

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Which of the following best represents the relative left ventricular end-diastolic volumes just prior to the heart beats represented by the letters?

- A. X=Y=Z
- B. X>Y>Z
- C. Y>X>Z
- D. Y=Z>X
- E. Z>X>Y

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Which of the following best represents the relative left ventricular end-diastolic volumes just prior to the heart beats represented by the letters?

- A. X=Y=Z (14%)
- ✗ B. X>Y>Z (5%)
- C. Y>X>Z (19%)
- D. Y=Z>X (4%)
- ✓ E. Z>X>Y (55%)

Incorrect
Correct answer
E

55%
Answered correctly

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Explanation

Duration of ventricular diastole prior to each beat

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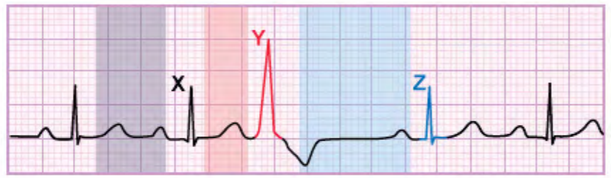
Duration of ventricular diastole prior to each beat



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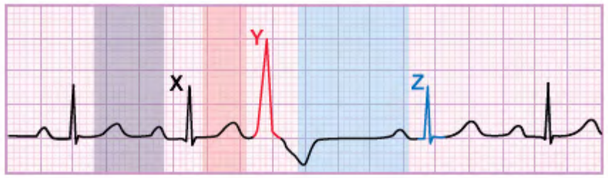
Duration of ventricular diastole prior to each beat



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The beat marked by the **letter Y** in this patient is a **premature ventricular contraction** (PVC), recognized by a wide QRS complex, opposite-facing T wave, and brief ensuing sinus pause (ie, delayed P wave). PVCs may result from increased ventricular automaticity leading to spontaneous ventricular depolarization. Occasional PVCs are normal and considered benign.

When a PVC occurs, it **interrupts diastolic filling** of the left ventricle, resulting in **decreased end-diastolic volume** (EDV) compared to the previous normal beat (**beat X > Y**). The sinus pause that occurs after a PVC allows for **longer-than-normal filling** time before the next ventricular contraction (ie, prolonged ventricular diastole), which creates **increased EDV** at the time of the post-PVC beat (**beat Z > X**). The greater-than-normal EDV causes a large-stroke volume ventricular ejection, which may be responsible for the palpitations that some patients experience with PVCs.

Educational objective:
A premature ventricular contraction is recognized on ECG by a wide QRS complex, opposite-facing T wave, and brief ensuing sinus pause. Left ventricular filling is less than normal at the time of the PVC. The ensuing sinus pause allows for greater-than-normal filling during the post-PVC beat.

Physiology	Cardiovascular System	Premature ventricular contractions
Subject	System	Topic

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A 63-year-old man comes to the emergency department due to dyspnea. Over the past several days, the patient has experienced progressively worsening shortness of breath while walking his dog around the block. In addition, he could not breathe while lying in bed last night and fell asleep only after moving to a recliner. The patient had an acute myocardial infarction 2 years ago and has been nonadherent with his medications and follow-up appointments. Temperature is 36.7 C (98 F), blood pressure is 122/74 mm Hg, pulse is 94/min, and respirations are 22/min. Physical examination shows bibasilar lung crackles, jugular venous distension, and bilateral pitting edema in the lower extremities. Chest x-ray reveals cardiomegaly and pulmonary venous congestion. Which of the following factors is most likely contributing to this patient's symptoms?

- A. Decreased arteriolar resistance
- B. Decreased plasma renin activity
- C. Decreased ventricular end-diastolic pressure
- D. Increased plasma brain natriuretic peptide concentration
- E. Increased sympathetic nervous system activity

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- ✗ A. Decreased arteriolar resistance (5%)
- B. Decreased plasma renin activity (2%)
- C. Decreased ventricular end-diastolic pressure (21%)
- D. Increased plasma brain natriuretic peptide concentration (17%)
- ✓ E. Increased sympathetic nervous system activity (52%)

Incorrect
Correct answer
E

52%
Answered correctly

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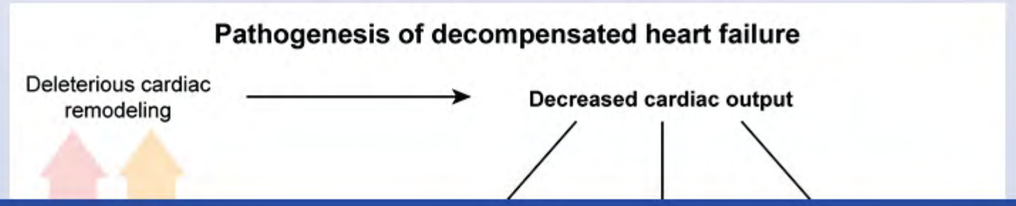
Explanation



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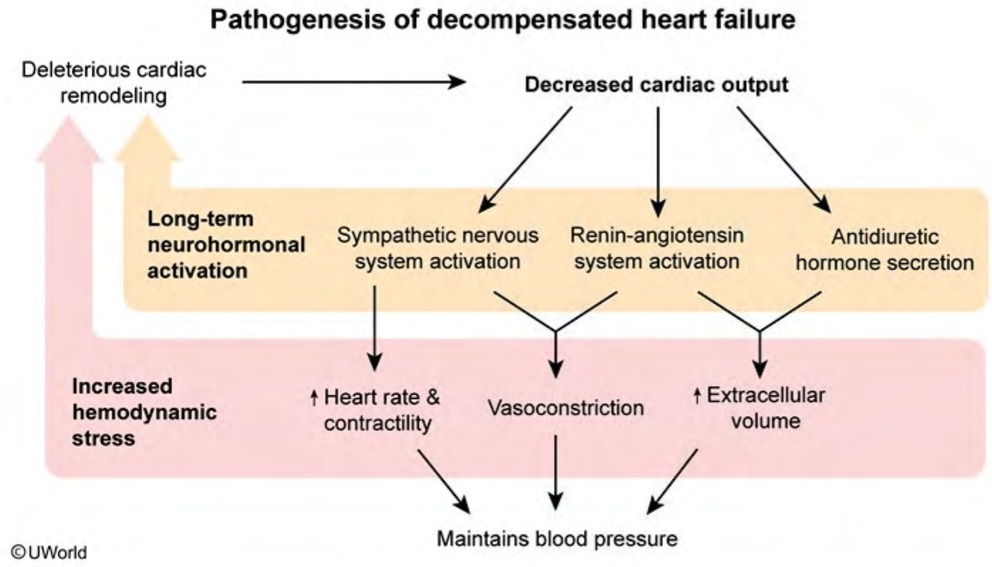

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This patient's progressive dyspnea, orthopnea, and lower extremity swelling are consistent with acute decompensated **heart failure**. Heart failure occurs when a structural or functional cardiac disorder impairs ventricular filling or ejection of blood, causing **reduced cardiac output** and elevated filling pressure. The reduced cardiac output causes an initial drop in blood pressure, which triggers compensatory neurohormonal activity mainly via **stimulation** of the **sympathetic nervous system** and **renin-angiotensin-aldosterone system** (RAAS).

These **adaptations** function together in an effort to **improve organ perfusion** by increasing cardiac chronotropy and inotropy (improves cardiac output), promoting arterial and venous vasoconstriction (maintains perfusion pressure), and expanding the extracellular fluid compartment (improves circulating blood volume). However, **over time**, these responses lead to adverse consequences that perpetuate a downward spiral of **progressive cardiac deterioration**. Chronic hemodynamic stress (eg, increased preload and afterload) and prolonged exposure to sympathetic drive (catecholamines) and RAAS stimulation (angiotensin II, aldosterone) lead to **deleterious remodeling** with worsening cardiac function and eventual symptomatic decompensation.

(Choices A and B) Decreased cardiac output in heart failure stimulates increased RAAS activity and sympathetic output, resulting in increased arteriolar resistance.

(Choice C) Ventricular end-diastolic and end-systolic pressures are increased in heart failure due to increased preload and afterload.

(Choice D) Heart failure triggers the myocardium to secrete natriuretic peptides (ie, atrial natriuretic peptide, brain natriuretic peptide) in response to myocardial stretch from volume overload. These peptides stimulate vasodilation and increase salt and water excretion to improve the symptoms of heart failure; however, these positive effects are overpowered by the negative effects of increased sympathetic drive and RAAS activation.

Educational objective:
The reduced cardiac output in heart failure triggers compensatory activation of the sympathetic nervous system and renin-angiotensin-aldosterone pathway, resulting in vasoconstriction (increased afterload), fluid retention

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decompensated **heart failure**. Heart failure occurs when a structural or functional cardiac disorder impairs ventricular filling or ejection of blood, causing **reduced cardiac output** and elevated filling pressure. The reduced cardiac output causes an initial drop in blood pressure, which triggers compensatory neurohormonal activity mainly via **stimulation** of the **sympathetic nervous system** and **renin-angiotensin-aldosterone system** (RAAS).

These **adaptations** function together in an effort to **improve organ perfusion** by increasing cardiac chronotropy and inotropy (improves cardiac output), promoting arterial and venous vasoconstriction (maintains perfusion pressure), and expanding the extracellular fluid compartment (improves circulating blood volume). However, **over time**, these responses lead to adverse consequences that perpetuate a downward spiral of **progressive cardiac deterioration**. Chronic hemodynamic stress (eg, increased preload and afterload) and prolonged exposure to sympathetic drive (catecholamines) and RAAS stimulation (angiotensin II, aldosterone) lead to **deleterious remodeling** with worsening cardiac function and eventual symptomatic decompensation.

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Educational objective:
The reduced cardiac output in heart failure triggers compensatory activation of the sympathetic nervous system and renin-angiotensin-aldosterone pathway, resulting in vasoconstriction (increased afterload), fluid retention (increased preload), and deleterious cardiac remodeling. These mechanisms perpetuate a downward spiral of cardiac deterioration, leading to symptomatic decompensated heart failure.

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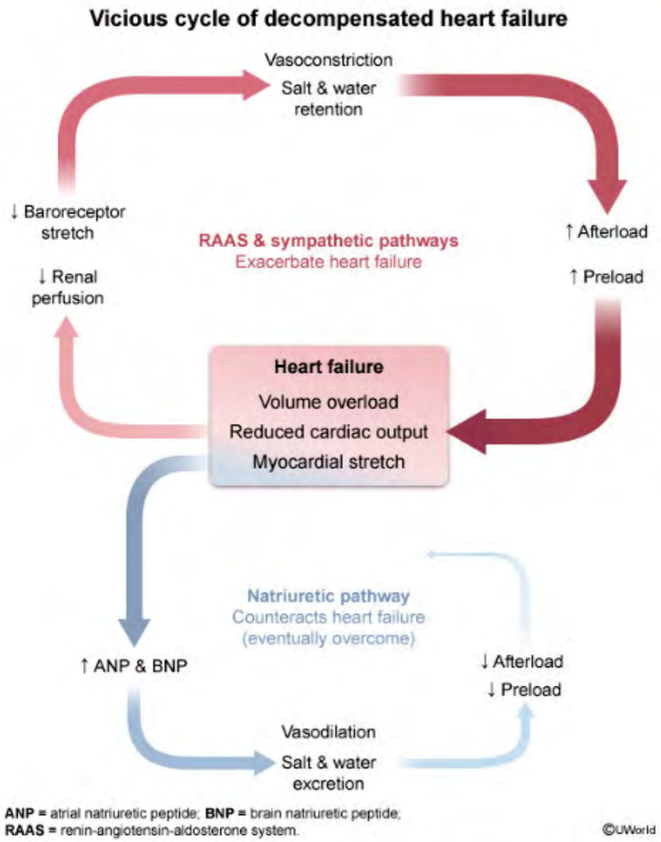

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A 55-year-old man comes to the office for follow-up of abnormal serum chemistries found on routine laboratory testing. He has a history of hypertension, for which he is being treated with pharmacologic therapy, weight loss, and dietary salt restriction. Blood pressure is 130/80 mm Hg and pulse is 80/min. BMI is 27 kg/m^2. Physical examination reveals no abnormalities. Laboratory studies are as follows:

Serum chemistry	
Sodium	134 mEq/L
Potassium	3.8 mEq/L
Blood urea nitrogen	18 mg/dL
Creatinine	0.8 mg/dL
Calcium	11.0 mg/dL
Glucose	98 mg/dL
Albumin	4 g/dL
Parathyroid hormone	decreased

Which of the following is most likely responsible for these findings?

- A. Familial hypocalciuric hypercalcemia
- B. Hypothyroidism
- C. Medication effect
- D. Primary hyperparathyroidism
- E. Secondary hyperparathyroidism

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Blood urea nitrogen	18 mg/dL
Creatinine	0.8 mg/dL
Calcium	11.0 mg/dL
Glucose	98 mg/dL
Albumin	4 g/dL
Parathyroid hormone	decreased

Which of the following is most likely responsible for these findings?

- ✗ A. Familial hypocalciuric hypercalcemia (20%)
- B. Hypothyroidism (1%)
- ✓ C. Medication effect (63%)
- D. Primary hyperparathyroidism (4%)
- E. Secondary hyperparathyroidism (9%)

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Correct answer
C

63%
Answered correctly

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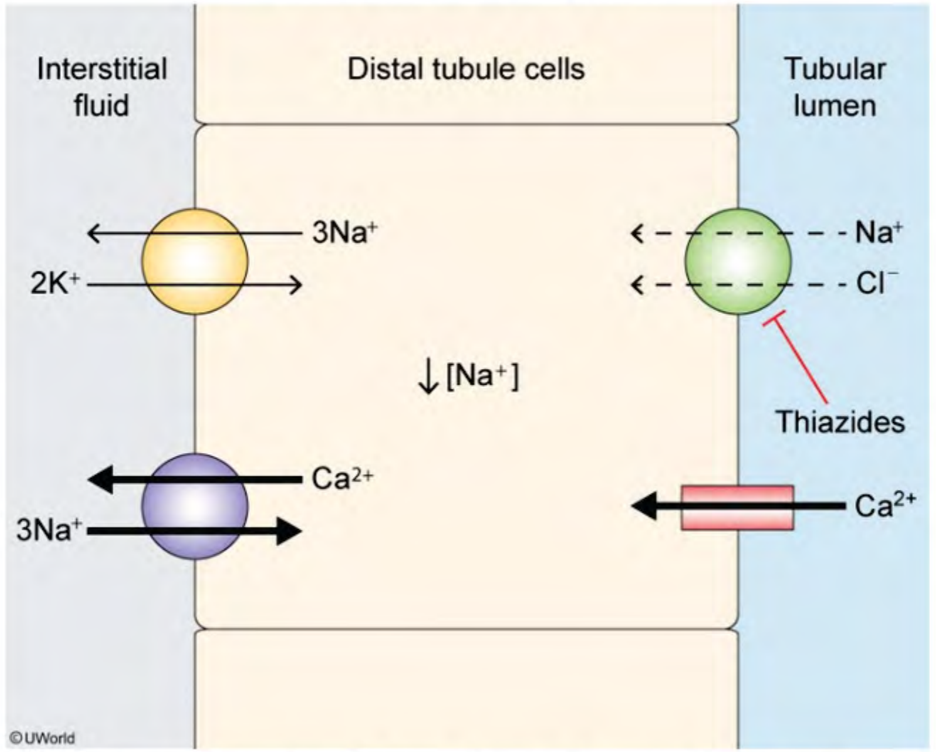
Effect of thiazide diuretics on distal tubular calcium reabsorption



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Thiazide diuretics (eg, chlorthalidone, hydrochlorothiazide) are some of the most widely used agents for treatment of hypertension. However, thiazides, especially in higher doses, can cause a variety of metabolic complications, including hypercalcemia, hyperglycemia, hyperlipidemia, hyponatremia, and hypokalemia. The incidence of the individual side effects is relatively low, and, patients rarely experience all of the potential adverse effects.

Thiazides increase renal Ca^{2+} reabsorption, leading to **hypercalcemia** and **hypocalciuria**. This occurs through 2 major mechanisms:

1. Thiazides **inhibit the Na^+/Cl^- cotransporter** on the apical side of distal convoluted tubule cells, which decreases intracellular Na^+ concentration. This activates the basolateral **Na^+/Ca^{2+} antiporter**, which pumps Na^+ into the cell in exchange for Ca^{2+}. The resulting decrease in intracellular Ca^{2+} enhances luminal Ca^{2+} reabsorption across the apical membrane.
2. **Hypovolemia** induced by thiazides increases Na^+ and H_2O reabsorption in the proximal tubule, leading to a passive increase in paracellular Ca^{2+} reabsorption.

The increase in circulating calcium suppresses parathyroid hormone (PTH) release, leading to a new steady state with a mildly elevated serum calcium concentration and low serum PTH.

(Choice A) Familial hypocalciuric hypercalcemia is a benign disorder caused by mutations in the calcium-sensing receptor in the parathyroid gland and kidneys. Higher calcium levels are required to suppress secretion of PTH, leading to mild hypercalcemia and hypocalciuria. However, unlike in patients taking thiazide diuretics, PTH is high-normal or mildly elevated.

(Choice B) Hyperthyroidism can cause hypercalcemia due to increased osteoclastic bone resorption, but serum calcium in hypothyroidism is usually normal.

(Choices D and E) Primary hyperparathyroidism is caused by parathyroid hyperplasia or adenoma; it is characterized by hypercalcemia with elevated or inappropriately normal PTH levels. Secondary hyperparathyroidism is usually seen in patients with chronic kidney disease and is due to phosphate retention and

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1. Thiazides **inhibit the Na^+/Cl^- cotransporter** on the apical side of distal convoluted tubule cells, which decreases intracellular Na^+ concentration. This activates the basolateral **Na^+/Ca^{2+} antiporter**, which pumps Na^+ into the cell in exchange for Ca^{2+}. The resulting decrease in intracellular Ca^{2+} enhances luminal Ca^{2+} reabsorption across the apical membrane.
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(Choices D and E) Primary hyperparathyroidism is caused by parathyroid hyperplasia or adenoma; it is characterized by hypercalcemia with elevated or inappropriately normal PTH levels. Secondary hyperparathyroidism is usually seen in patients with chronic kidney disease and is due to phosphate retention and decreased production of 1,25-dihydroxyvitamin D; these disturbances lead to decreased intestinal calcium absorption, hypocalcemia, hyperphosphatemia, and a compensatory increase in PTH secretion. This patient has a decreased PTH level.

Educational objective:
Thiazide diuretics cause hypercalcemia by increasing the distal tubular reabsorption of Ca^{2+}. The increased circulating calcium levels result in suppression of parathyroid hormone, which distinguishes this effect from hyperparathyroidism.

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An autopsy is being performed on a 72-year-old man with a history of myocardial infarction. Light microscopy of a portion of the left ventricle reveals the findings shown in the image below:

This patient most likely sustained the myocardial infarction during which of the following time frames prior to his death?

- A. Less than 4 hours
- B. 1 to 3 days
- C. 7 to 10 days
- D. 2 weeks to 2 months
- E. More than 2 months

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An autopsy is being performed on a 72-year-old man with a history of myocardial infarction. Light microscopy of a portion

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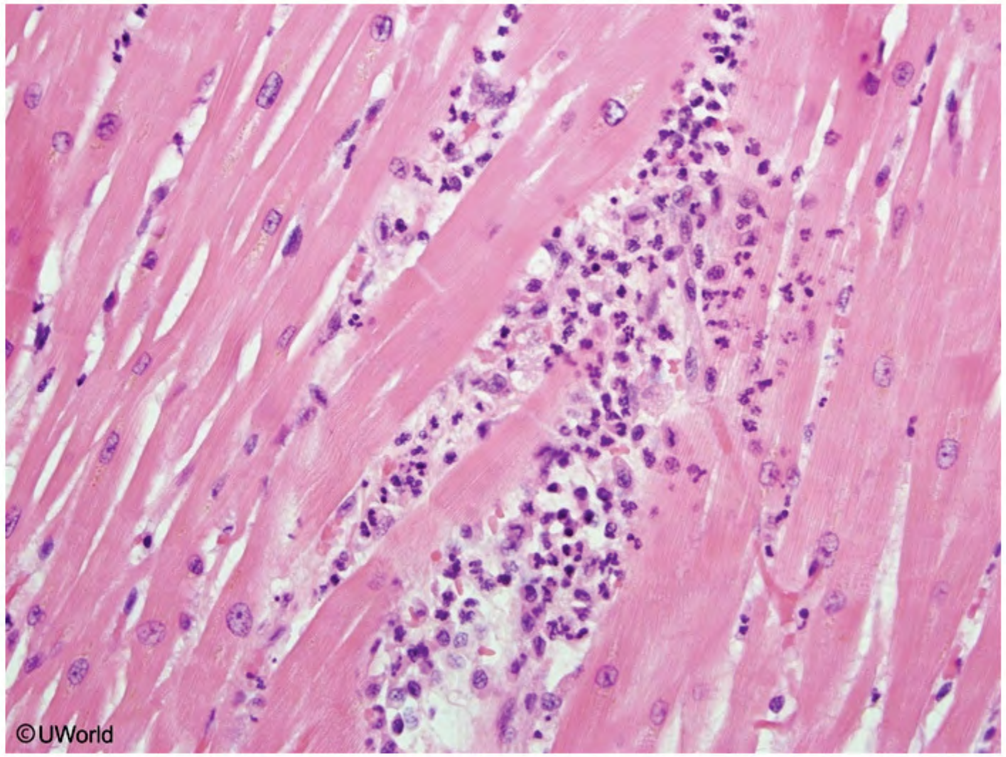

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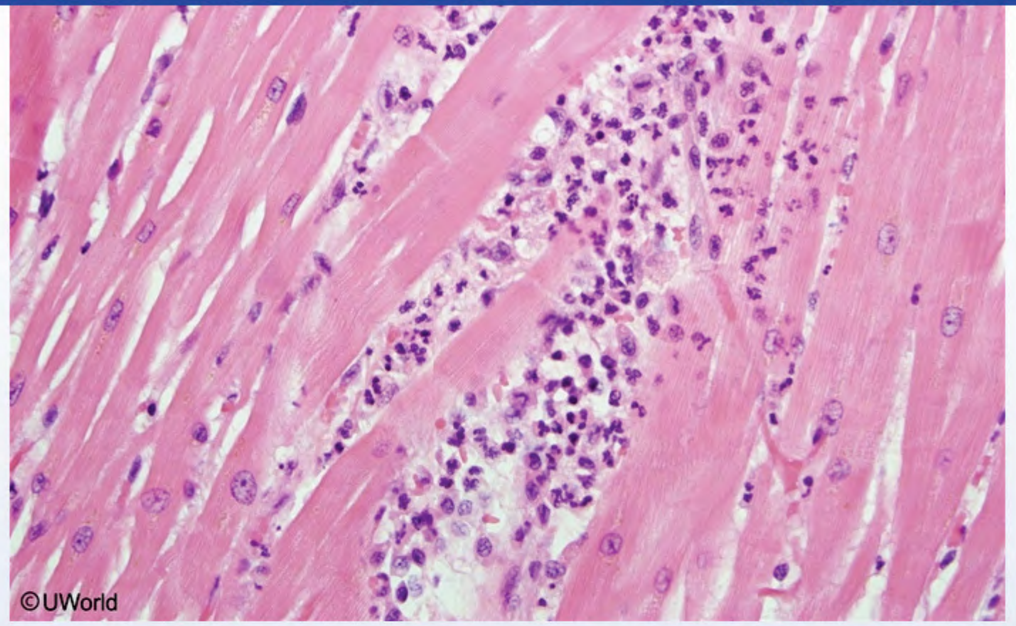

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- A. Less than 4 hours
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- C. 7 to 10 days
- D. 2 weeks to 2 months
- E. More than 2 months

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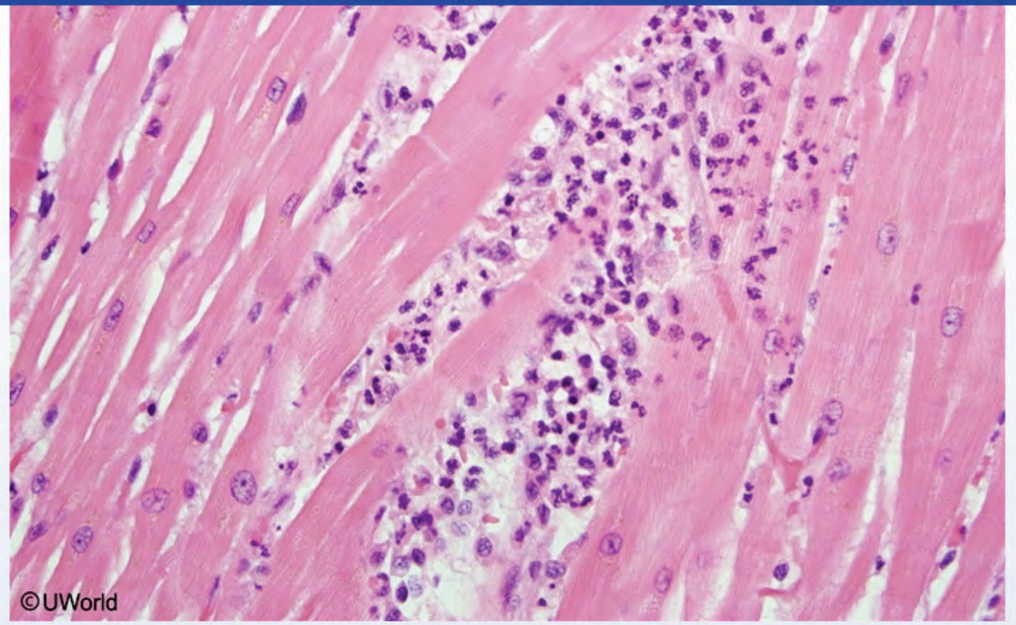

This patient most likely sustained the myocardial infarction during which of the following time frames prior to his death?

- A. Less than 4 hours (8%)
- ✓ B. 1 to 3 days (76%)
- C. 7 to 10 days (11%)
- D. 2 weeks to 2 months (1%)
- E. More than 2 months (1%)

Correct

76% Answered correctly

12 secs Time Spent

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Time after myocardial infarction	Predominant light microscopic changes
0-4 hours	• No visible change
4-12 hours	• Wavy fibers with narrow, elongated myocytes
12-24 hours	• Myocyte hypereosinophilia with pyknotic (shrunken) nuclei
1-3 days	• Coagulation necrosis (loss of nuclei & striations) • Prominent neutrophilic infiltrate
3-7 days	• Disintegration of dead neutrophils & myofibers • Macrophage infiltration at border areas
7-10 days	• Robust phagocytosis of dead cells by macrophages • Beginning formation of granulation tissue at margins
10-14 days	• Well-developed granulation tissue with neovascularization
2-8 weeks	• Progressive collagen deposition & scar formation

Postmortem histopathology of this patient's myocardium reveals a dense neutrophilic infiltrate surrounded by relatively normal myocytes consistent with the morphologic changes expected in the border zone of ischemic injury **1-3 days after** myocardial infarction (MI). Myocardial cells are highly metabolically active and susceptible to **ischemic injury** after as little as 3-4 minutes of oxygen deprivation due to coronary artery blockage.

After irreversible myocardial injury, a series of characteristic **microscopic changes** occur over predictable time frames due to **coagulative necrosis** of myocardial fibers and infiltration of **inflammatory cells**, which play an essential role in the healing process:

- During the first 4 hours post-MI, no visible change occurs although cardiac-specific enzymes can be detected in the patient's bloodstream **(Choice A)**.
- From 4 to 24 hours post-MI, changes due to digestion of cytoplasmic organelles, denaturation of proteins, and

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10-14 days	• Well-developed granulation tissue with neovascularization
2-8 weeks	• Progressive collagen deposition & scar formation

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- During the first 4 hours post-MI, no visible change occurs although cardiac-specific enzymes can be detected in the patient's bloodstream **(Choice A)**.
- From 4 to 24 hours post-MI, changes due to digestion of cytoplasmic organelles, denaturation of proteins, and loss of RNA become visible (eg, wavy, hypereosinophilic myocytes with shrunken nuclei), especially in the central portion of the infarct.
- From **1 to 3 days** post-MI, **neutrophils infiltrate** the **border zone** of the injured tissue in response to proinflammatory cytokines (eg, IL-6, IL-8), eventually migrating to the central portion of the infarct. These neutrophils phagocytize dead myocytes and release **lysosomal enzymes**, reactive oxygen species, and **cytokines** (eg, IL-1, TNF-α) that can prevent infiltration by pathogens and facilitate wound healing.
- From 3 to 10 days post-MI, macrophages infiltrate and phagocytize dead myocytes and neutrophils while initiating the formation of granulation tissue by producing cytokines (eg, IL-10) and growth factors (TGF-β) **(Choice C)**.
- From 10 to 14 days post-MI, prominent fibroblast proliferation, collagen deposition, and neovascularization of granulation tissue are present.
- From 2 weeks to 2 months post-MI, collagen remodeling and fibroblast differentiation into myofibroblasts drive

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- From **1 to 3 days** post-MI, **neutrophils infiltrate** the **border zone** of the injured tissue in response to proinflammatory cytokines (eg, IL-6, IL-8), eventually migrating to the central portion of the infarct. These neutrophils phagocytize dead myocytes and release **lysosomal enzymes**, reactive oxygen species, and **cytokines** (eg, IL-1, TNF-α) that can prevent infiltration by pathogens and facilitate wound healing.
- From 3 to 10 days post-MI, macrophages infiltrate and phagocytize dead myocytes and neutrophils while initiating the formation of granulation tissue by producing cytokines (eg, IL-10) and growth factors (TGF-β) **(Choice C)**.
- From 10 to 14 days post-MI, prominent fibroblast proliferation, collagen deposition, and neovascularization of granulation tissue are present.
- From 2 weeks to 2 months post-MI, collagen remodeling and fibroblast differentiation into myofibroblasts drive the formation of scar tissue **(Choice D)**.
- After 2 months post-MI, remodeling continues to a slower rate as the scar matures and contracts **(Choice E)**.

Educational objective:
Irreversible myocardial ischemic injury initiates a series of predictable morphologic changes over the hours to weeks after the initial injury. From 1 and 3 days after the initial injury, neutrophils infiltrate the border zone of the injured tissue (where myocytes are relatively normal) in response to proinflammatory cytokines (eg, IL-6, IL-8), eventually migrating to the central portion of the infarct.

Histology Cardiovascular System Myocardial infarction

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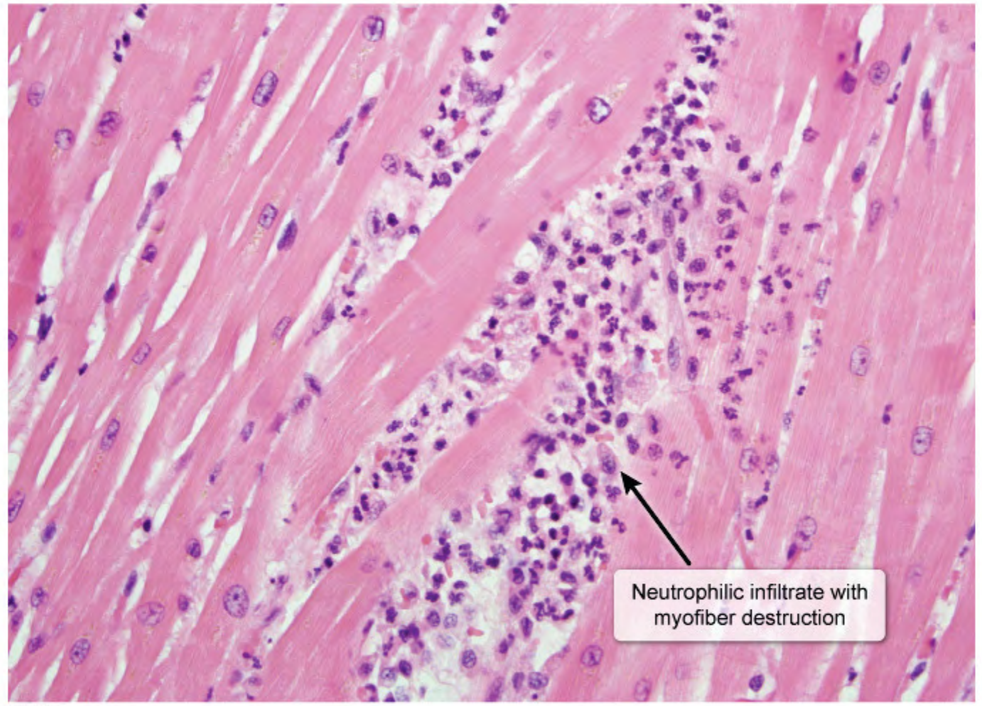
Acute myocardial infarction



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After irreversible myocardial injury, a series of characteristic **microscopic changes** occur over predictable time frames essent

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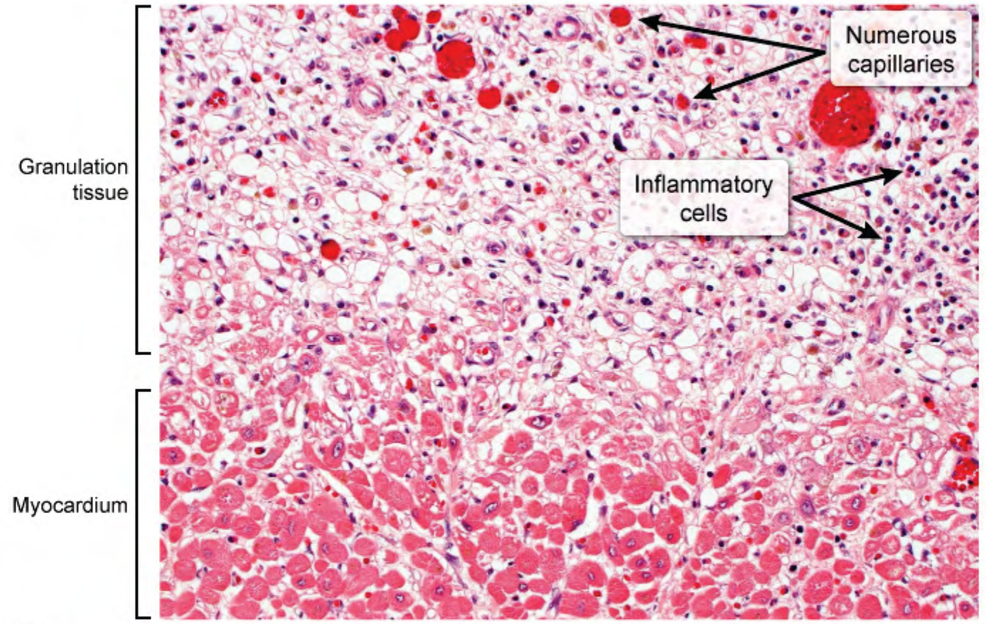
weeks after the initial injury. From 1 and 3 days after the initial injury, neutrophils infiltrate the border zone of the injured tissue (where myocytes are relatively normal) in response to proinflammatory cytokines (eg, IL-6, IL-8)

Exhibit Display

Healing myocardial infarction



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After irreversible myocardial injury, a series of characteristic **microscopic changes** occur over predictable time frames essent

- D in
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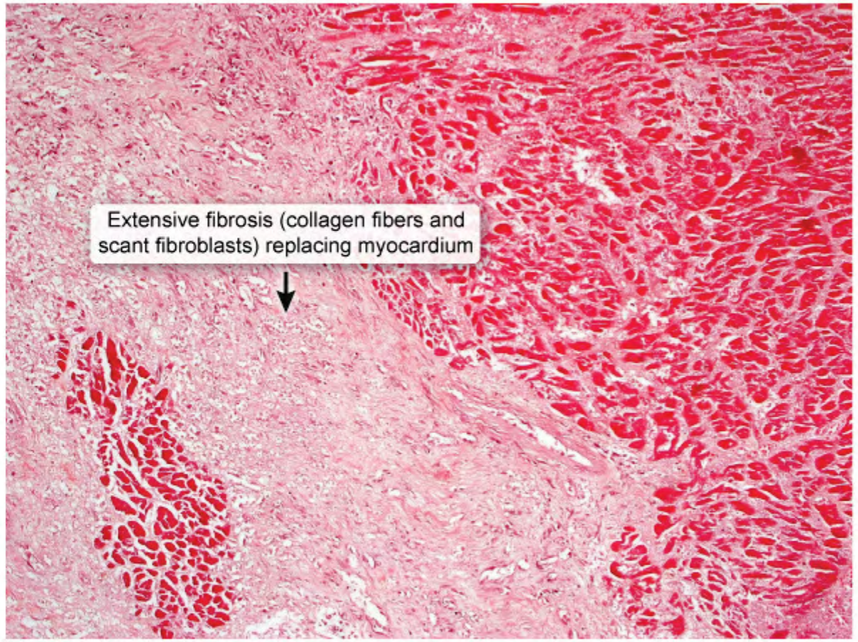
weeks after the initial injury. From 1 and 3 days after the initial injury, neutrophils infiltrate the border zone of the injured tissue (where myocytes are relatively normal) in response to proinflammatory cytokines (eg, IL-6, IL-8)

Exhibit Display

Resolved myocardial infarcts



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Special electrodes are used to detect the change in membrane potential of a specific type of cardiac cell. These changes are recorded on the graph below.

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The deflection indicated by the arrow is most likely caused by movement of which of the following ions?

- A. Sodium
- B. Potassium
- C. Calcium
- D. Chloride
- E. Sodium and potassium
- F. Calcium and potassium

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Special electrodes are used to detect the change in membrane potential of a specific type of cardiac cell. These changes are recorded on the graph below.

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The deflection indicated by the arrow is most likely caused by movement of which of the following ions?

- A. Sodium (20%)
- ✗ B. Potassium (2%)
- ✓ C. Calcium (71%)
- D. Chloride (0%)
- E. Sodium and potassium (2%)
- F. Calcium and potassium (2%)

Incorrect
Correct answer
C

71%
Answered correctly

20 secs
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Explanation

Cardiac action potentials

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Incorrect
Correct answer
C

71%
Answered correctly

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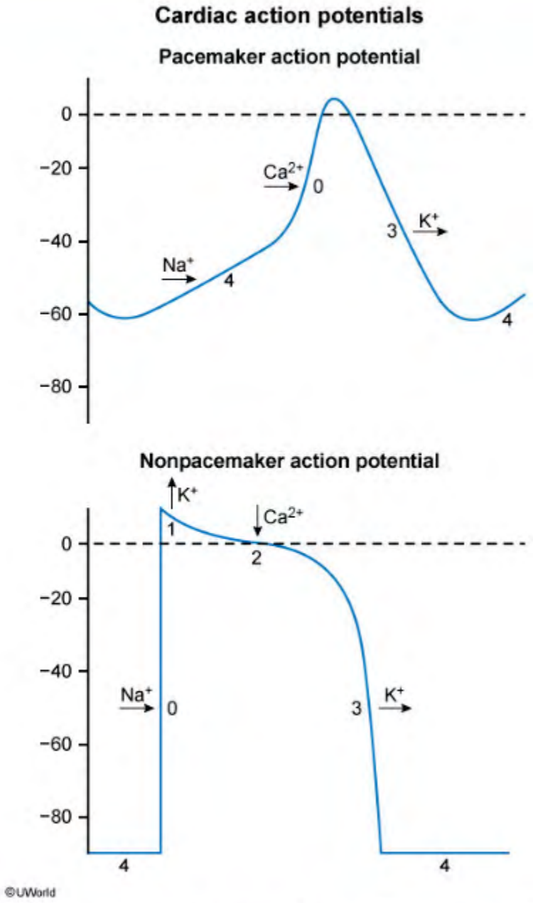

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The graph in the question stem illustrates an action potential typical of cardiac **pacemaker cells**, such as those found in the sinoatrial node. These action potentials are different from non-pacemaker cardiac action potentials in that phases 1 and 2 are absent and phase 4 represents a slow depolarization phase.

Pacemaker cells are unique in that they exhibit automaticity (an inherent ability to depolarize without any external influences). Automaticity is made possible by a inward, mixed sodium-potassium current (the **funny current**) that occurs during phase 4 of the action potential **(Choice E)**. This current gradually brings the membrane potential closer to threshold, at which point phase 0 depolarization occurs due to a **large increase in calcium influx (arrow)**. Pacemaker cell depolarization is then followed by phase 3 (repolarization), which is caused by an outward potassium current **(Choice B)**.

In cardiomyocytes and Purkinje cells (**non-pacemaker cells**), phase 4 corresponds to the resting membrane potential and is typically stable at approximately -90 mV (near the equilibrium potential of potassium). Phase 0 of the non-pacemaker action potential is mediated by rapid sodium influx **(Choice A)**, in contrast to the slower calcium-mediated depolarization of pacemaker cells. Phase 1 is an early short repolarization occurring immediately after depolarization that is caused by increased outflow of potassium and decreased sodium conductance. Phase 2 is the plateau phase of the action potential, during which an inward calcium current counteracts the outward movement of potassium ions that started in phase 1 **(Choice F)**. Phase 3 (repolarization) occurs as the inward calcium current ceases while potassium continues to rush out of the cell.

Educational objective:
In cardiac pacemaker cells, phase 0 depolarization is mediated by an inward flux of calcium. This differs from phase 0 of cardiomyocytes and Purkinje cells, which results from an inward sodium current.

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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A 62-year-old woman comes to the office due to a vague feeling of heaviness in her legs, especially when standing for long periods. She has no history of trauma to the legs. The patient's other medical problems include osteoarthritis and gastroesophageal reflux disease. She has worked as a cashier at a department store for the last 20 years. Vital signs are normal. Jugular venous pressure is normal. The heart has regular rate and rhythm with no murmurs or extra sounds. Lung sounds are clear with normal air movement. The abdomen is soft and without masses. Examination of the lower extremities shows dilated, tortuous veins at the calves and ankles bilaterally, with no erythema or edema. Upper extremity examination is unremarkable. This patient's current condition increases the risk for which of the following?

- A. Intermittent claudication
- B. Ischemic stroke
- C. Myocardial infarction
- D. Phlegmasia alba dolens
- E. Pulmonary embolism
- F. Skin ulcerations

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- ✗ A. Intermittent claudication (13%)
- B. Ischemic stroke (0%)
- C. Myocardial infarction (0%)
- D. Phlegmasia alba dolens (5%)
- E. Pulmonary embolism (44%)
- ✓ F. Skin ulcerations (35%)

Incorrect
Correct answer
F

35%
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Explanation

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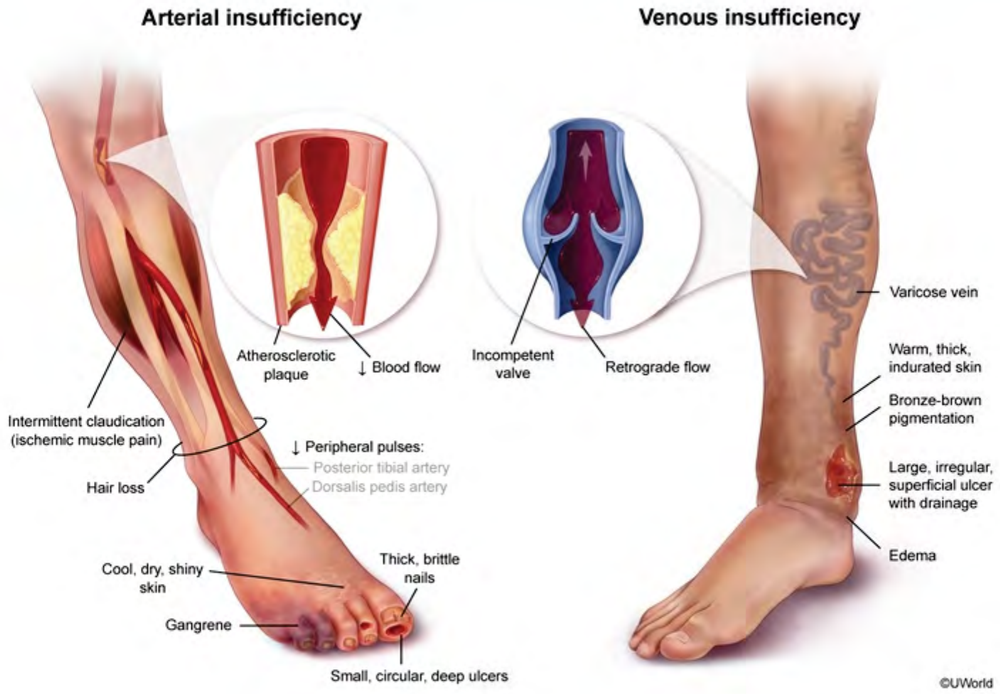

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the venous valves. The resulting backflow of blood exacerbates the venous hypertension, precipitating a vicious

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This patient has **varicose veins** - dilated, tortuous veins typically involving the superficial venous drainage of the leg. Increased intraluminal pressure or loss of vessel wall tensile strength can lead to venous dilation and failure of the venous valves. The resulting backflow of blood exacerbates the venous hypertension, precipitating a vicious cycle that leads to further valvular incompetence and venous congestion.

Varicose veins are most common in patients age >50, and the risk is increased by conditions that cause elevated venous pressure (eg, prolonged standing, pregnancy, obesity). There is also often a familial association, suggesting possible genetic factors involving the venous wall or valves. Common complications include chronic edema, stasis dermatitis, skin ulcerations, poor wound healing, and infection.

(Choices A, B, and C) Intermittent claudication is achy or crampy pain in the legs caused by inadequate arterial blood flow, usually due to atherosclerosis in the branches of the femoral artery. In contrast to venous disease, in which the pain is worsened by prolonged standing or sitting with the legs dependent, claudication due to arterial disease is worse with exertion and relieved with rest. Because peripheral artery atherosclerosis is often associated with atherosclerotic disease elsewhere, patients with intermittent claudication have an increased risk for ischemic stroke and myocardial infarction.

(Choices D and E) Deep venous thrombosis of the lower extremity can lead to pulmonary embolism. In addition, massive iliofemoral thrombosis can cause an acute rise in tissue pressure that impairs arterial inflow, leading to phlegmasia alba dolens (painful white "milk leg"). Although varicose veins can sometimes be complicated by thrombophlebitis, it primarily involves the superficial rather than deep venous system and does not increase the risk for pulmonary embolism or phlegmasia alba dolens.

Educational objective:
Varicose veins are dilated, tortuous veins most commonly found in the superficial veins of the leg. They are caused by chronically increased intraluminal pressure and/or loss of tensile strength in the vessel wall, leading to incompetence of the venous valves. Common complications include edema, stasis dermatitis, skin ulcerations, poor wound healing, and infections.

References

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This patient has **varicose veins** - dilated, tortuous veins typically involving the superficial venous drainage of the leg. In the ve
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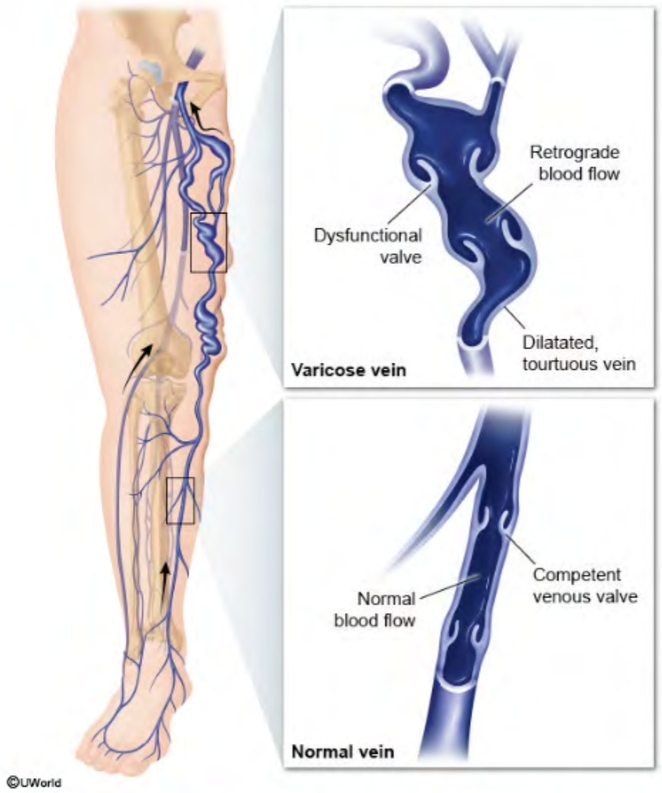

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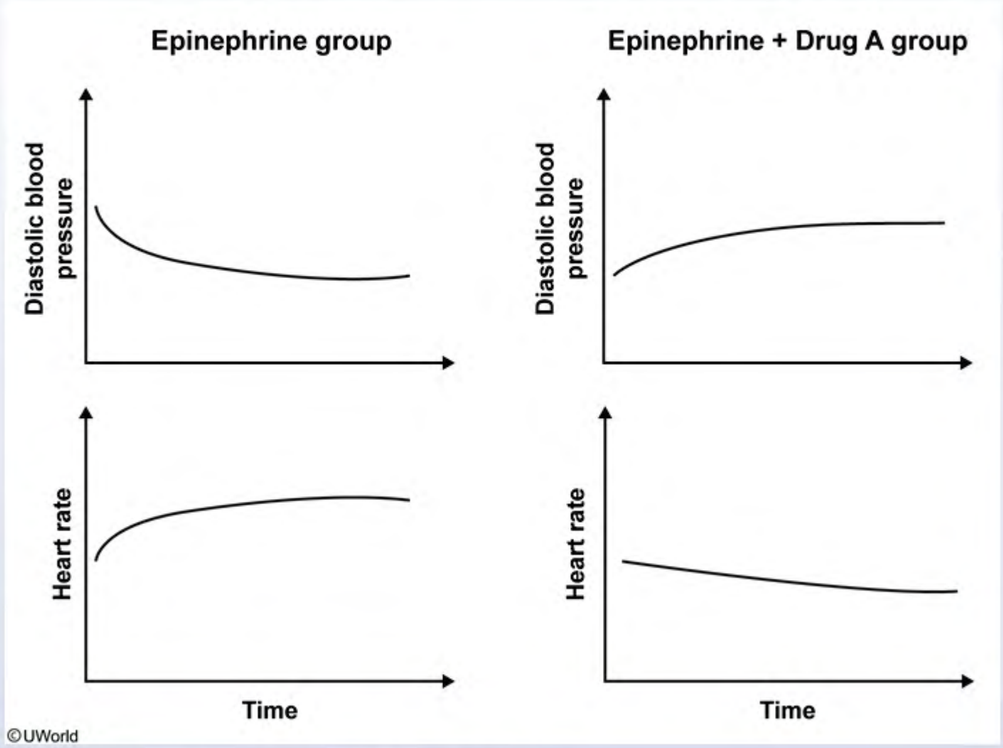
An experiment is conducted to assess the effects of drug A on the hemodynamic actions of epinephrine. Two groups of healthy volunteers are selected. The first group receives only low-dose epinephrine, and its effects on diastolic blood pressure and heart rate are monitored and shown below (epinephrine group). The second group is pretreated with drug A and then infused with low-dose epinephrine to produce a new set of curves (epinephrine + drug A group).



Which of the following medications is most likely to be drug A?

A. Atropine

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An experiment is conducted to assess the effects of drug A on the hemodynamic actions of epinephrine. Two groups
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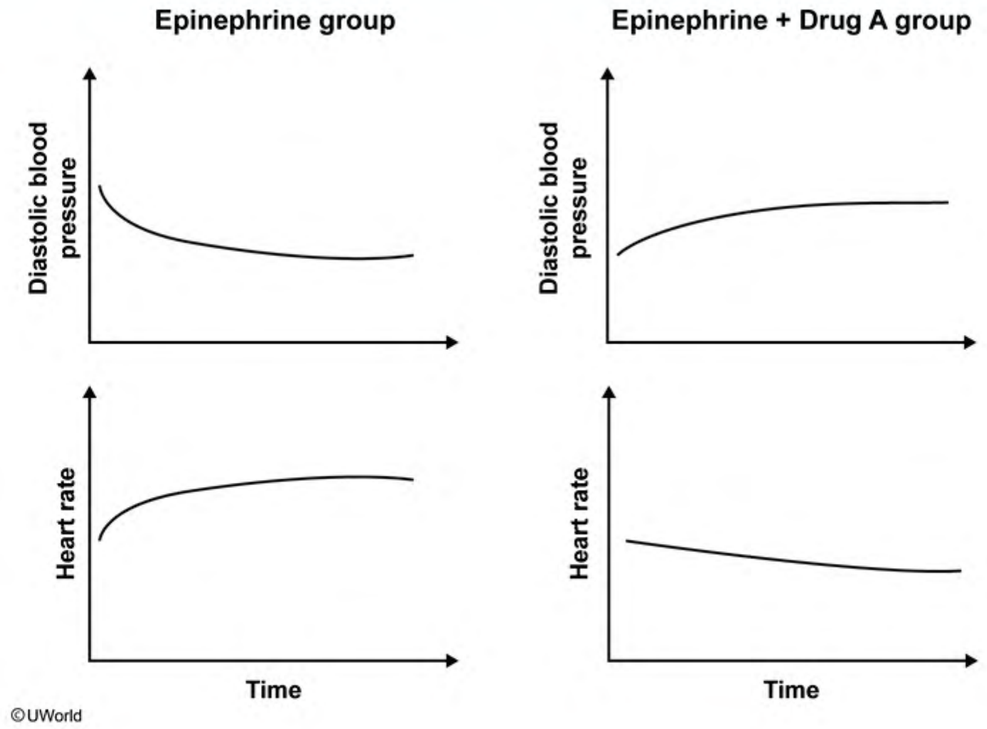

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Which

A. Atropine

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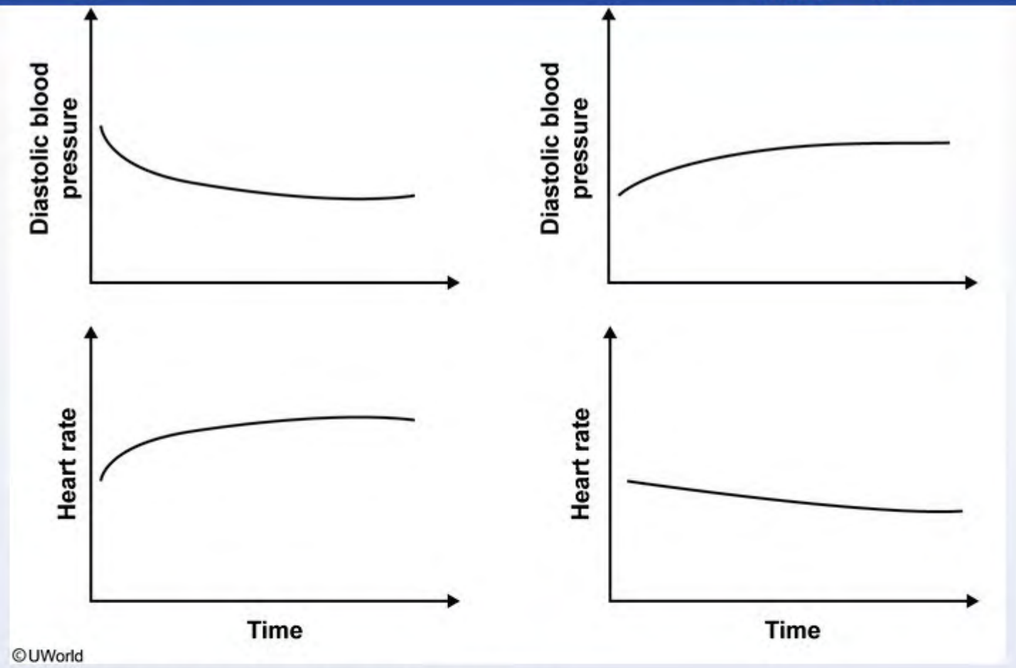

Which of the following medications is most likely to be drug A?

- A. Atropine
- B. Isoproterenol
- C. Phentolamine
- D. Phenylephrine
- E. Propranolol

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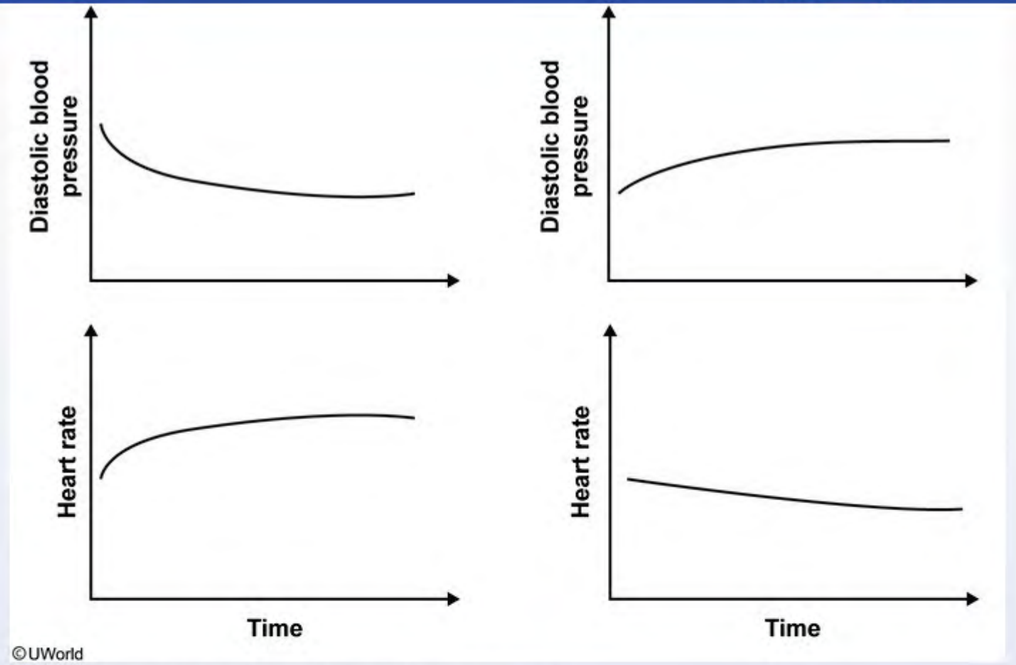

Which of the following medications is most likely to be drug A?

- A. Atropine (3%)
- B. Isoproterenol (15%)
- C. Phentolamine (11%)
- D. Phenylephrine (14%)
- ✓ E. Propranolol (55%)

Correct

55% Answered correctly

16 secs Time Spent

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Hemodynamic actions of epinephrine		
	Effect	**Involved receptors**
Heart rate & contractility	↑	β_1
Systolic blood pressure	↑↑	$\beta_1 + \alpha_1$
Diastolic blood pressure	↓ (low-dose)	$\beta_2 > \alpha_1$
	↑ (high-dose)	$\alpha_1 > \beta_2$

The hemodynamic actions of **epinephrine** occur secondary to agonistic effects at α1, β1, and β2 adrenoreceptors (relative strength: $\boldsymbol{\beta_1 = \beta_2 > \alpha_1}$). Stimulation of β1 receptors in the heart increases cardiac rate and contractility, which increases systolic blood pressure due to the rise in cardiac output. Stimulation of β2 receptors in the skeletal muscle vasculature produces vasodilation and decreased diastolic blood pressure, while activation of α1 receptors on vascular smooth muscle in the skin and viscera increases peripheral resistance and raises diastolic blood pressure. At **low doses**, β2-mediated **vasodilation predominates**, whereas at high doses α1-induced vasoconstriction becomes more pronounced.

In this experiment, low-dose epinephrine by itself caused **increased heart rate** (β1 response) and **decreased diastolic blood pressure** (β2 response). However, after pretreatment with drug A, the heart rate *fails* to increase and there is a marked *rise* in diastolic blood pressure. These effects are best explained by pretreatment with a **nonselective beta antagonist** such as propranolol. When administered alone, propranolol produces peripheral vasoconstriction (β2 antagonism) without significantly increasing blood pressure (due to concomitant β1 blockade). However, in combination with epinephrine, propranolol will counteract epinephrine's β2-mediated vasodilation and β1-mediated heart rate increase while leaving the α1 effect (vasoconstriction) intact. This will

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vasoconstriction (β2 antagonism) without significantly increasing blood pressure (due to concomitant β1 blockade). However, in combination with epinephrine, propranolol will counteract epinephrine's β2-mediated vasodilation and β1-mediated heart rate increase while leaving the α1 effect (vasoconstriction) intact. This will increase diastolic blood pressure and cause a slight reflexive reduction in heart rate.

(Choice A) Atropine is a competitive antagonist of cholinergic muscarinic receptors in the heart (M_2). Atropine increases the heart rate by blocking parasympathetic vagal influence; after pretreatment with atropine, epinephrine-mediated tachycardia would increase.

(Choice B) Isoproterenol is a nonselective β-adrenergic agonist that stimulates both β2 receptors on vascular smooth muscle (leading to vasodilatation) and cardiac β1 receptors (leading to increases in cardiac rate and contractility). A combination of isoproterenol and epinephrine would further decrease diastolic blood pressure and exacerbate the tachycardia.

(Choice C) Phentolamine is a nonspecific alpha-1 and alpha-2 blocker with no beta-blocking effects that cause a profound fall in diastolic blood pressure and reflex tachycardia. If this were drug A, blood pressure would have decreased further in the pretreatment group and the rise in heart rate would have been exacerbated.

(Choice D) Phenylephrine is a selective α-adrenergic agonist. Administration by itself may cause elevated systolic and diastolic blood pressures, which may be associated with reflex bradycardia. However, after pretreatment, epinephrine would still produce an *initial rise* in heart rate due to unopposed β1 stimulation.

Educational objective:
Epinephrine increases systolic blood pressure (α_1 + β_1) and heart rate (β1), and either increases or decreases diastolic blood pressure depending on the dose (either α1 or β2 predominates). Pretreatment with propranolol eliminates the β effects of epinephrine (vasodilatation and tachycardia), leaving only the α effect (vasoconstriction).

Pharmacology	Cardiovascular System	Sympathomimetic agents
Subject	System	Topic

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A 42-year-old woman comes to the emergency department for evaluation of chest pain. She was moving furniture in her summer house 2 days ago when she experienced sharp pain in the left side of the sternum that quickly subsided. Since then, the patient has had episodic pain with deep inspiration or trunk movement. She has no fever or cough. The patient has a history of hypertension. Her father died of myocardial infarction at age 67. She does not use tobacco or illicit drugs. Blood pressure is 146/85 mm Hg in the right arm and 142/80 mm Hg in the left arm, pulse is 86/min, and respirations are 12/min. She has localized tenderness to palpation at the left sternal border. Lungs are clear to auscultation, and cardiac examination reveals normal heart sounds without gallops or murmurs. The abdomen is soft and nontender. There is no peripheral edema. Which of the following is the most likely cause of this patient's symptoms?

- A. Acute pericarditis
- B. Aortic dissection
- C. Costochondritis
- D. Esophageal spasm
- E. Gastroesophageal reflux disease
- F. Panic disorder
- G. Pneumonia
- H. Pulmonary arterial hypertension
- I. Pulmonary embolism
- J. Unstable angina

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- A. Acute pericarditis (8%)
- B. Aortic dissection (8%)
- ✓ C. Costochondritis (73%)
- ✗ D. Esophageal spasm (0%)
- E. Gastroesophageal reflux disease (0%)
- F. Panic disorder (0%)
- G. Pneumonia (0%)
- H. Pulmonary arterial hypertension (1%)
- I. Pulmonary embolism (1%)
- J. Unstable angina (5%)

Incorrect
Correct answer
C

73%
Answered correctly

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Differential diagnosis & features of chest pain	
Coronary artery disease	• Substernal • Precipitated by exertion • Relieved by rest or nitroglycerin
Pulmonary/pleuritic (pleurisy, pneumonia, pericarditis, PE)	• Sharp/stabbing pain • Worse with inspiration • Pericarditis: worse when lying flat
Aortic (dissection, intramural hematoma)	• Abrupt (maximal at onset), severe "tearing" pain • May radiate to back • Hypertension and/or inherited aortopathy
Esophageal	• Substernal, may refer to neck • Associated with regurgitation • Provoked by recumbent position • Nonexertional, relieved by antacids
Chest wall/ musculoskeletal	• Persistent pain • Worse with movement or change in position • Often follows repetitive activity
PE = pulmonary embolism.	

This patient's chest pain is most likely due to **costosternal syndrome** (also known as costochondritis or anterior chest wall syndrome) involving the regional chest wall. It usually occurs after repetitive activity and involves the upper costal cartilage at the costochondral or costosternal junctions. The pain is typically **reproduced with palpation** and worsened with movement or changes in position (eg, horizontal arm flexion). Patients typically do not have palpable warmth, swelling, or erythema.

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(Choices A, G, and I) Conditions causing inflammation of the pleura or pericardium can cause sharp pain worsened with inspiration. Pericarditis often follows an upper respiratory viral syndrome. The pain of pericarditis is typically worse when lying flat and relieved by leaning forward. Examination may show a pericardial friction rub. Pulmonary embolism can cause pleural inflammation with chest pain. However, such cases are typically due to large emboli and are associated with significant hypoxia and shortness of breath. Pneumonia usually presents with fever, cough, and shortness of breath. None of these conditions would be associated with focal tenderness of the chest wall.

(Choice B) Aortic dissection causes sudden, tearing chest pain radiating to the back and blood pressure disparity between the arms. This patient's episodic pain makes this less likely. Blood pressure will normally vary mildly between arms; a difference in systolic or diastolic blood pressure <10 mm Hg is not considered significant.

(Choices D and E) Gastroesophageal reflux disease typically causes postprandial or nocturnal burning pain in the upper abdomen and chest. Esophageal spasm can cause chest pain with meals or swallowing.

(Choice F) Panic disorder is usually a diagnosis of exclusion and can present with episodic chest pain, shortness of breath, and palpitations. Patients usually have intense fear/anxiety, and chest wall tenderness is not seen.

(Choice H) Pulmonary arterial hypertension usually presents with exertional dyspnea, lethargy, and fatigue. Other symptoms, such as exertional chest pain, syncope, and peripheral edema, are generally seen in advanced disease.

(Choice J) Unstable angina is characterized by exertional chest pressure, often radiating to the arm or jaw. Local chest tenderness does not rule out a cardiac cause of chest pain but is unusual.

Educational objective:
Costosternal syndrome (costochondritis) usually occurs after repetitive activity and is characterized by pain that is

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(Choices A, G, and I) Conditions causing inflammation of the pleura or pericardium can cause sharp pain worsened with inspiration. Pericarditis often follows an upper respiratory viral syndrome. The pain of pericarditis is typically worse when lying flat and relieved by leaning forward. Examination may show a pericardial friction rub. Pulmonary embolism can cause pleural inflammation with chest pain. However, such cases are typically due to large emboli and are associated with significant hypoxia and shortness of breath. Pneumonia usually presents with fever, cough, and shortness of breath. None of these conditions would be associated with focal tenderness of the chest wall.

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Educational objective:
Costosternal syndrome (costochondritis) usually occurs after repetitive activity and is characterized by pain that is reproducible with palpation and worsened with movement or changes in position.

References

- Costochondritis: diagnosis and treatment.

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A 52-year-old woman comes to the emergency department due to increasing dyspnea and chest tightness over the past 2 weeks. She has a history of stage 4 chronic kidney disease due to chronic glomerulonephritis. Temperature is 36.6 C (97.8 F), blood pressure is 106/82 mm Hg, and pulse is 98/min. Systolic blood pressure decreases by 12 mm Hg during inspiration. Physical examination shows distant heart sounds and clear lungs. Echocardiography in this patient is most likely to reveal which of the following findings?

- A. Apical wall motion abnormality
- B. Dilation of all cardiac chambers
- C. Engorged inferior vena cava
- D. Severe tricuspid regurgitation
- E. Systolic anterior motion of the mitral valve

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- A. Apical wall motion abnormality (23%)
- ✕ B. Dilation of all cardiac chambers (17%)
- ✓ C. Engorged inferior vena cava (47%)
- D. Severe tricuspid regurgitation (6%)
- E. Systolic anterior motion of the mitral valve (5%)

Incorrect
Correct answer
C

47%
Answered correctly

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Explanation

Cardiac tamponade	
Etiology	• Blood in pericardial space (eg, LV rupture, cardiac surgery) • Pericardial effusion (eg, malignancy, infection, uremia)
Clinical signs	• Beck triad: hypotension, JVD, ↓ heart sounds • Pulsus paradoxus: SBP ↓10 mm Hg during inspiration
	• ECG: low-voltage QRS, electrical alternans

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Clinical signs	• Beck triad: hypotension, JVD, ↓ heart sounds • Pulsus paradoxus: SBP ↓10 mm Hg during inspiration
Diagnosis	• ECG: low-voltage QRS, electrical alternans • Chest x-ray: enlarged cardiac silhouette*, clear lungs • Echocardiogram: right atrial & ventricular collapse, IVC plethora
*Subacute but not acute tamponade. **IVC** = inferior vena cava; **JVD** = jugular venous distension; **LV** = left ventricular; **SBP** = systolic blood pressure.	

This patient with progressive dyspnea and chest tightness over a period of 2 weeks and a **drop** in systolic blood pressure **>10 mm Hg with inspiration** (ie, pulsus paradoxus) likely has **subacute cardiac tamponade**; uremia in the setting of **advanced renal failure** can lead to pericarditis with associated **pericardial effusion** and sometimes tamponade. Tamponade classically presents with **Beck's triad** of hypotension (often less prominent with subacute compared to acute tamponade), distant heart sounds, and jugular venous distension.

Normally, a pressure gradient exists between the vena cava and the right side of the heart, allowing blood to fill the right atrium during diastole. With inspiration, a reduction in intrathoracic pressure pulls additional blood into the right side of the heart and the pulmonary circulation, causing normal inspiratory collapse of the inferior vena cava (IVC).

With cardiac tamponade, increasing pericardial pressure begins to compress the relatively low-pressure, **right-sided heart chambers** and **restrict diastolic filling**. The increased right-sided pressures are transmitted

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With cardiac tamponade, increasing pericardial pressure begins to compress the relatively low-pressure, **right-sided heart chambers** and **restrict diastolic filling**. The increased right-sided pressures are transmitted backward to the vena cava, causing **engorgement of the IVC** with decreased inspiratory collapse that can be seen on echocardiography. In addition, because the right ventricle is compressed and unable to expand during diastole, the increase in right-sided blood flow during inspiration causes the ventricular septum to bow into the left ventricle; this reduces left ventricular stroke volume during inspiration and is responsible for the observed >10 mm Hg drop in systolic blood pressure.

As tamponade progresses, impaired right-sided filling causes the left ventricle to receive less blood, resulting in decreased cardiac output and **obstructive shock**. Because the obstruction to blood flow is primarily right sided, there is no pulmonary edema and the lungs remain clear to auscultation.

(Choices A, B, D, and E) A segmental (eg, apical) wall motion abnormality suggests myocardial ischemia or infarction, dilation of all 4 cardiac chambers is seen with dilated cardiomyopathy, severe tricuspid regurgitation can occur with carcinoid syndrome, and systolic anterior motion of the mitral valve is seen with hypertrophic cardiomyopathy (and often exacerbates obstruction of the left ventricular outflow tract). None of these conditions are expected to cause pulsus paradoxus or distant heart sounds.

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Educational objective:
Cardiac tamponade involves restriction of diastolic filling of the right-sided heart chambers, with engorgement and decreased inspiratory collapse of the inferior vena cava seen on echocardiography. Pulsus paradoxus (>10 mm Hg drop in systolic blood pressure during inspiration) is commonly present with tamponade.

Pathophysiology	Cardiovascular System	Cardiac tamponade
Subject	System	Topic

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• Echocardiogram: right atrial & ventricular collapse, IVC plethora

*Subacute but not acute tamponade

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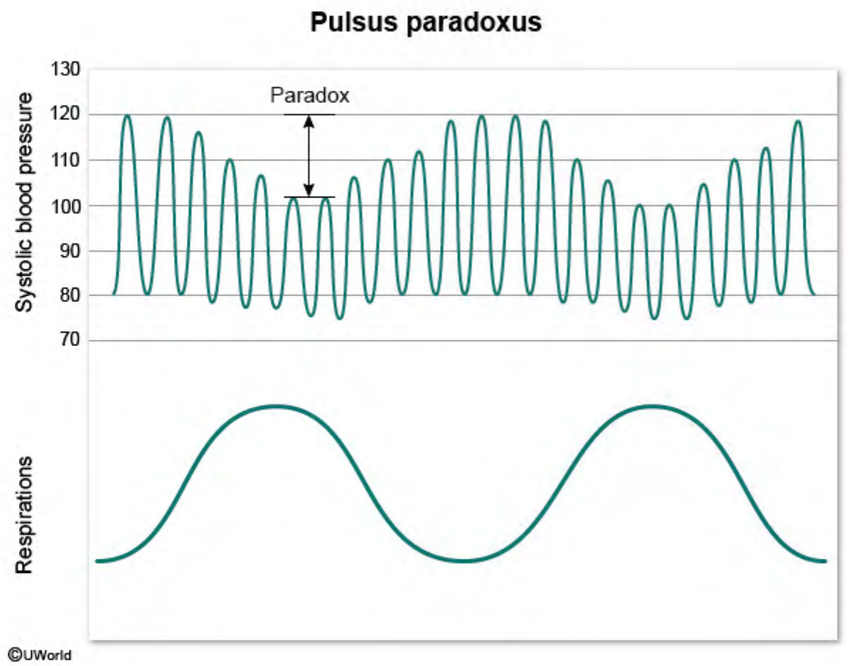

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A 44-year-old man comes to the office for a routine check-up. Past medical history is significant for hypercholesterolemia, which he has controlled through diet. His father died of a myocardial infarction at age 56, and his mother, who is still living, has a history of stroke. On physical examination, the patient's blood pressure is 160/100 mm Hg and heart rate is 70/min. He is started on enalapril. During the first 7 days of therapy, the patient's glomerular filtration rate (GFR) adjusts as shown in the graph below.

Which of the following structures was most likely affected by a downstream effect of enalapril and is therefore responsible for this patient's renal response?

- A. Afferent arterioles
- B. Distal tubules
- C. Efferent arterioles
- D. Proximal tubules
- E. Vasa recta

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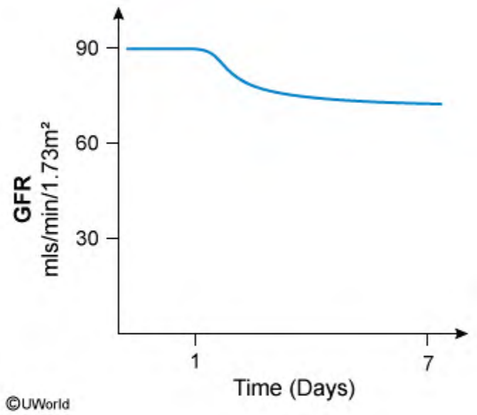

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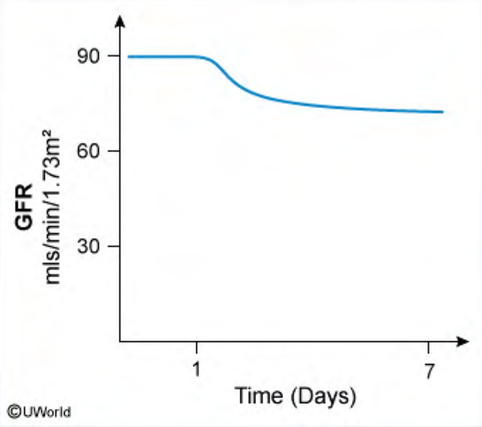

Which of the following structures was most likely affected by a downstream effect of enalapril and is therefore responsible for this patient's renal response?

- A. Afferent arterioles (12%)
- B. Distal tubules (2%)
- ✓ C. Efferent arterioles (83%)
- D. Proximal tubules (1%)
- E. Vasa recta (0%)

Correct

83% Answered correctly

09 secs Time Spent

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- ✓ C. Efferent arterioles (83%)
- D. Proximal tubules (1%)
- E. Vasa recta (0%)

Correct

83% Answered correctly

09 secs Time Spent

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Explanation

Enalapril is an angiotensin-converting-enzyme **(ACE) inhibitor**. All ACE inhibitors decrease circulating levels of angiotensin II, a substance that causes the following:

- Systemic vasoconstriction
- Preferential constriction of the glomerular efferent arteriole
- Enhancement of adrenal aldosterone secretion

As a result, **reduced angiotensin II** production in patients taking an ACE inhibitor would be expected to acutely **decrease efferent arteriolar resistance** and systemic vascular resistance. Selective efferent arteriolar dilation combined with the decreased renal perfusion pressure causes a **reduction in glomerular filtration rate**.

Educational objective:
In the kidney, angiotensin II preferentially constricts the efferent arteriole, thereby maintaining the glomerular filtration rate (GFR) within normal range. ACE-inhibitors promote efferent arteriolar dilation, causing GFR reduction.

Pharmacology	Cardiovascular System	ACE inhibitors
Subject	System	Topic

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A 52-year-old man comes to the office due to a 2-week history of progressive fatigue and exertional dyspnea. The patient is a postal delivery worker and reports that he gets short of breath after walking a few blocks. He has not had chest pain or palpitations. The patient does not use tobacco, alcohol, or illicit drugs. Cardiac auscultation reveals a murmur that is best heard when the patient sits up and leans forward. He brings to the office the report from a recent cardiac catheterization (graph shown below). This patient's murmur would most likely be loudest at which of the following time points on the graph?

- A. A
- B. B
- C. C
- D. D
- E. E

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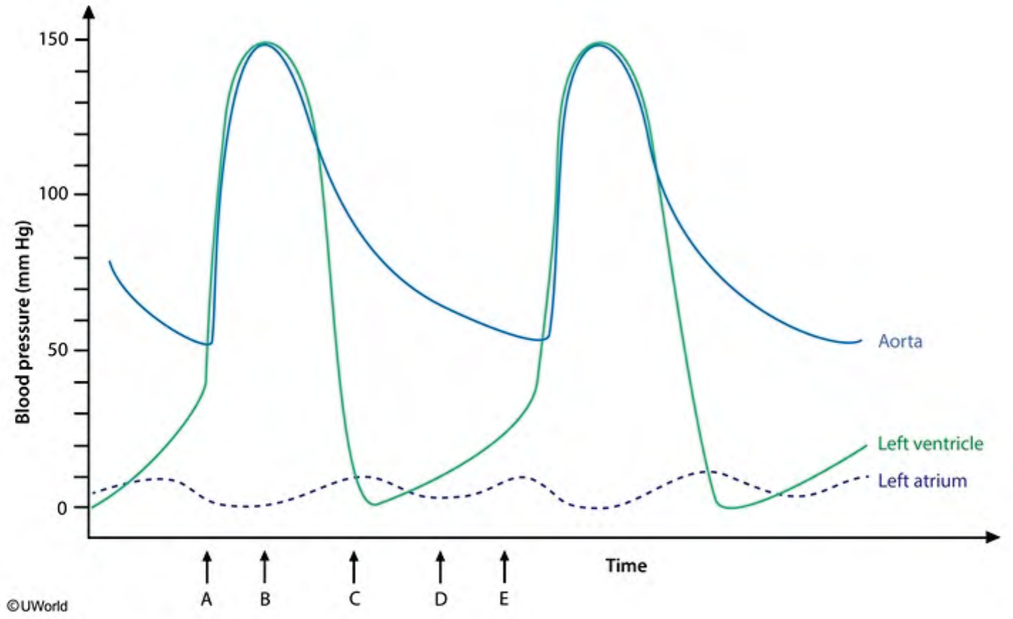

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D. D

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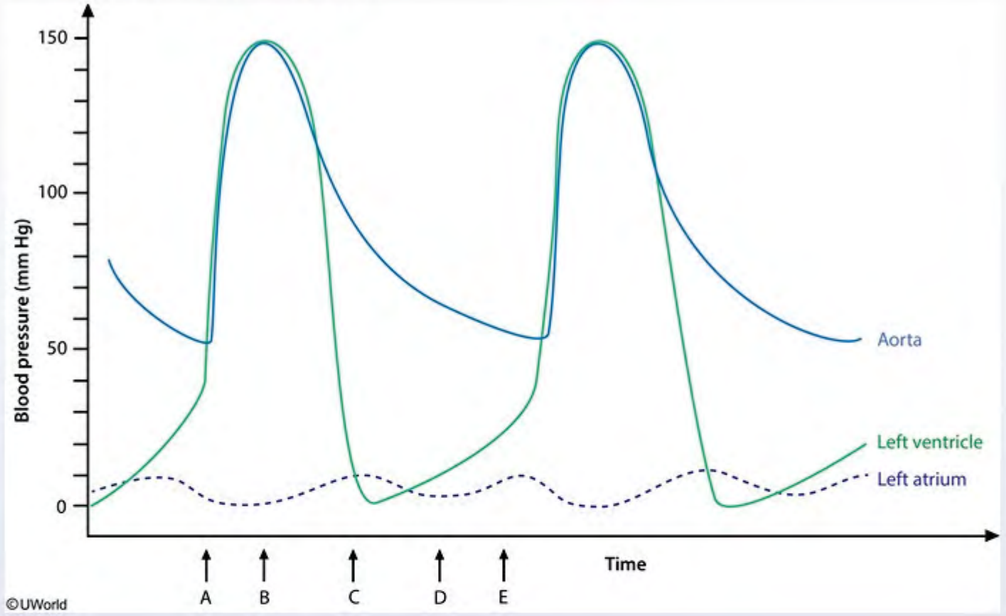

- A. A
- B. B
- C. C
- D. D
- E. E

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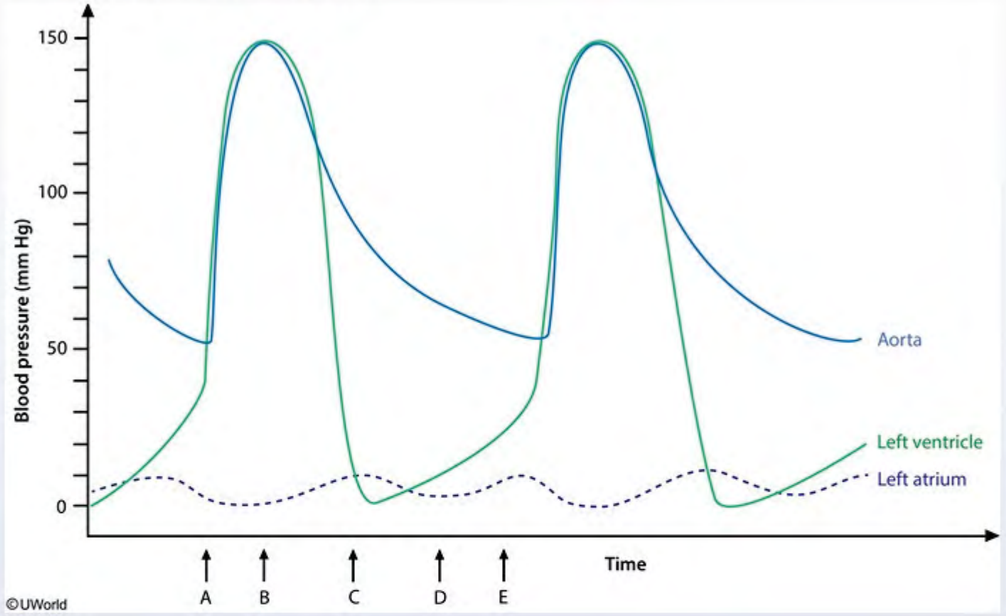

- A. A (14%)
- ✗ B. B (25%)
- ✓ C. C (47%)
- D. D (9%)
- E. E (3%)

Incorrect
Correct answer
C

47%
Answered correctly

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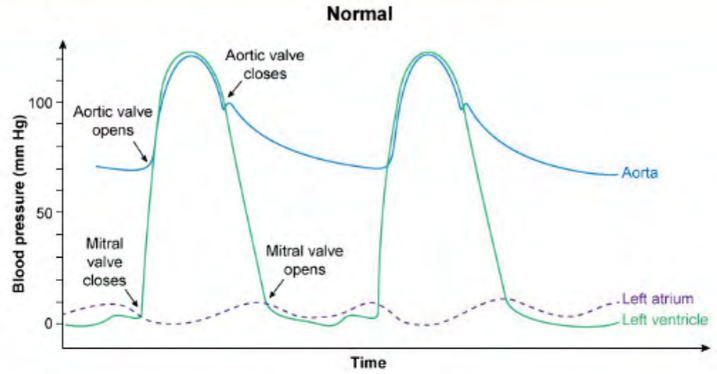

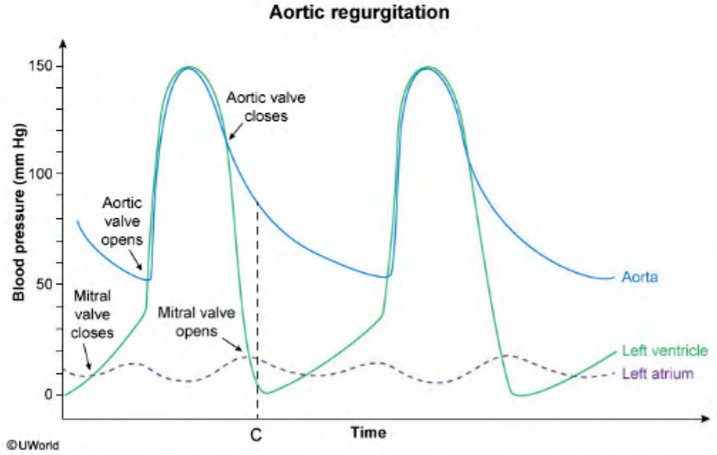

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This patient's cardiac catheterization shows a hemodynamic profile consistent with **aortic regurgitation** (AR); the characteristic decrescendo diastolic murmur is best heard with the patient leaning forward. The **peak intensity** of the murmur occurs **just after aortic valve closure** when the pressure gradient between the aorta and the left ventricle is maximal (point C). This pressure gradient falls progressively during diastole as blood regurgitates into the left ventricle, accounting for the **decrescendo** nature of the murmur.

Notable changes to the pressure tracings that occur in AR include:

- **Loss of the aortic dicrotic notch**: On the normal aortic pressure tracing the dicrotic notch is created by a small and abrupt increase in aortic pressure as blood is secured in the aorta by closure of the aortic valve. However, with an incompetent aortic valve there is immediate backflow of blood into the left ventricle, resulting in loss of pressure in the aorta and disappearance of the dicrotic notch.
- **Steep diastolic decline of aortic pressure**: Aortic diastolic pressure declines more rapidly than normal as blood regurgitates into the left ventricle and nadirs at a lower than normal level.
- **High-peaking left ventricular and aortic systolic pressures**: The left ventricle attempts to compensate for regurgitant backflow by increasing stroke volume, resulting in higher systolic blood pressures, which combined with low aortic diastolic pressure leads to a **wide pulse pressure**.

(Choice A) This point corresponds to the start of left ventricular contraction, just after mitral valve closure and just prior to the opening of the aortic valve; it would mark the onset of the holosystolic murmur of mitral regurgitation.

(Choice B) This is the peak systolic pressure produced by the left ventricle during systole; the peak intensity of the crescendo-decrescendo murmur of aortic stenosis is heard here.

(Choice D) This point corresponds to mid-diastole. The murmur of AR is likely heard here as well, but at a lower intensity given the relatively lower pressure gradient between the aorta and left ventricle. The low-pitch rumble of mitral stenosis would also be heard at this time.

(Choice E) This point marks the onset of left atrial contraction (atrial kick) just before closure of the mitral valve.

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However, with an incompetent aortic valve there is immediate backflow of blood into the left ventricle, resulting in loss of pressure in the aorta and disappearance of the dicrotic notch.

- **Steep diastolic decline of aortic pressure**: Aortic diastolic pressure declines more rapidly than normal as blood regurgitates into the left ventricle and nadirs at a lower than normal level.
- **High-peaking left ventricular and aortic systolic pressures**: The left ventricle attempts to compensate for regurgitant backflow by increasing stroke volume, resulting in higher systolic blood pressures, which combined with low aortic diastolic pressure leads to a **wide pulse pressure**.

(Choice A) This point corresponds to the start of left ventricular contraction, just after mitral valve closure and just prior to the opening of the aortic valve; it would mark the onset of the holosystolic murmur of mitral regurgitation.

(Choice B) This is the peak systolic pressure produced by the left ventricle during systole; the peak intensity of the crescendo-decrescendo murmur of aortic stenosis is heard here.

(Choice D) This point corresponds to mid-diastole. The murmur of AR is likely heard here as well, but at a lower intensity given the relatively lower pressure gradient between the aorta and left ventricle. The low-pitch rumble of mitral stenosis would also be heard at this time.

(Choice E) This point marks the onset of left atrial contraction (atrial kick) just before closure of the mitral valve. Accentuation of a mitral stenosis murmur may be heard at this time (presystolic accentuation).

Educational objective:

Aortic regurgitation causes a decrescendo diastolic murmur with maximal intensity occurring just after closure of the aortic valve, when the pressure gradient between the aorta and left ventricle is the highest. The pressure tracing for aortic regurgitation is characterized by loss of the aortic dicrotic notch, steep diastolic decline in aortic pressure, and high-peaking systolic pressures.

References

- The rational clinical examination. Does this patient have aortic regurgitation?

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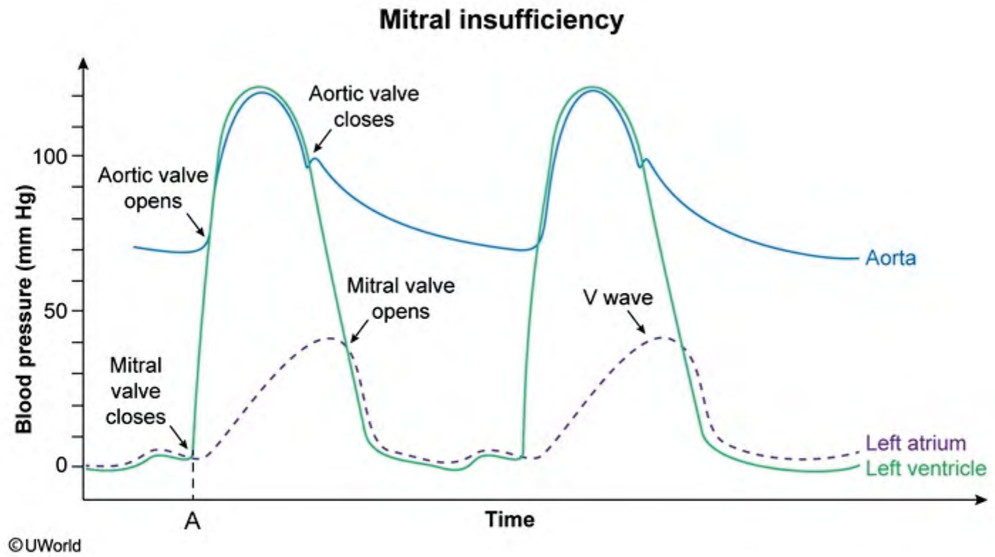

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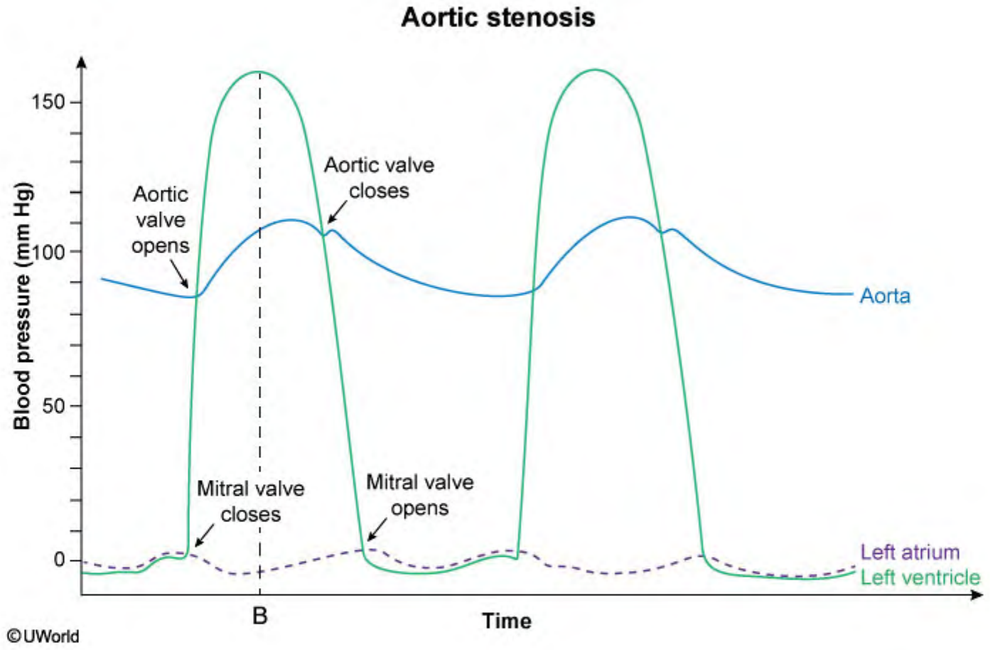

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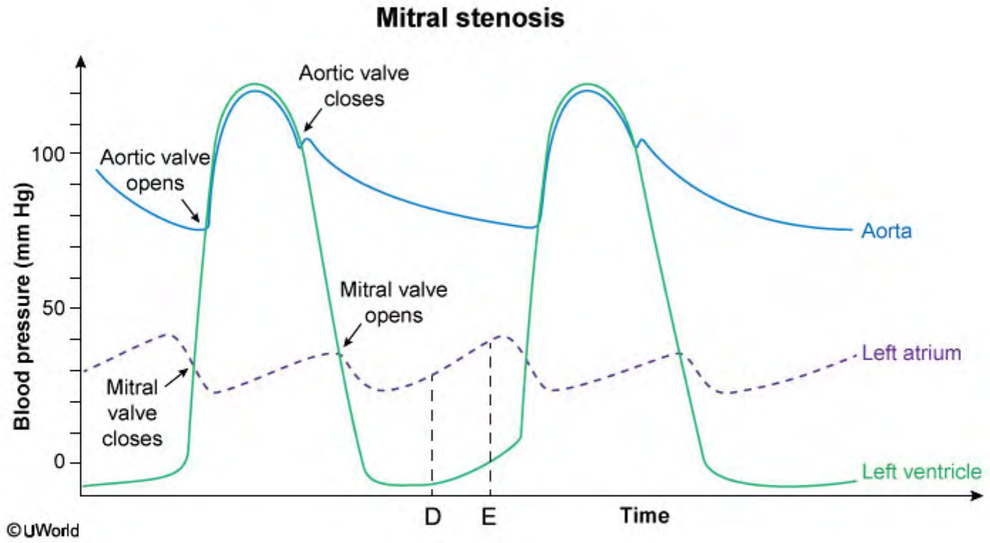

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A 64-year-old man with type 2 diabetes mellitus is evaluated in the clinic due to occasional dizziness. His symptoms usually occur while playing table tennis. The patient has not had any falls or loss of consciousness. He has no known history of coronary artery disease or stroke. The patient has smoked a pack of cigarettes daily for the past 40 years and occasionally drinks alcohol. Orthostatic vital signs are normal. ECG shows normal sinus rhythm. Doppler ultrasound evaluation of the left vertebral artery reveals retrograde (caudal) flow instead of normal antegrade flow. Which of the following arteries is most likely to be occluded based on these ultrasound findings?

- A. Innominate artery
- B. Left internal carotid
- C. Left internal mammary
- D. Left subclavian
- E. Right vertebral

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- A. Innominate artery (10%)
- ✗ B. Left internal carotid (33%)
- C. Left internal mammary (6%)
- ✓ D. Left subclavian (40%)
- E. Right vertebral (8%)

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Correct answer
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40%
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Explanation

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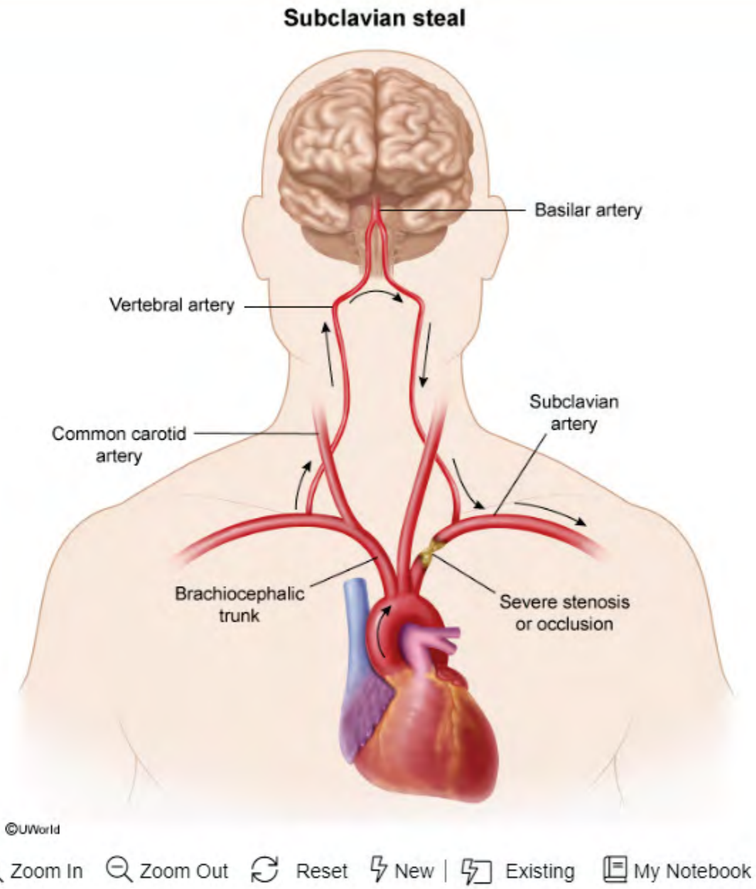

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Basilar artery

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This patient's presentation is consistent with **subclavian steal syndrome**, which typically occurs due to hemodynamically significant stenosis of the subclavian artery proximal to the origin of the vertebral artery. **Subclavian stenosis** is typically caused by atherosclerosis, although less common etiologies include Takayasu arteritis and complications from heart surgery (eg, aortic coarctation repair). The lowered distal subclavian arterial pressure leads to reversal in blood flow ("steal") from the contralateral vertebral artery to the ipsilateral vertebral artery, away from the brainstem.

Most patients with subclavian artery stenosis are asymptomatic. When symptoms occur, they are typically related to **arm ischemia** in the affected extremity (eg, exercise-induced fatigue, pain, paresthesias) or **vertebrobasilar insufficiency** (eg, dizziness, vertigo, drop attacks). Physical examination can show a significant difference (>15 mm Hg) in brachial systolic blood pressure between the affected arm and normal arm. Doppler ultrasound of the cerebrovascular and upper extremity arterial circulation establishes the diagnosis in most patients.

(Choice A) Although innominate (brachiocephalic) artery stenosis or occlusion can cause subclavian steal syndrome, patients with significant occlusion would develop retrograde flow through the right vertebral artery rather than the left.

(Choice B) Internal carotid artery occlusion may occur due to thrombosis or embolism, leading to a transient ischemic attack (TIA) or ischemic stroke. Patients typically have profound neurologic deficits (eg, contralateral homonymous hemianopsia, hemiparesis, hemisensory loss), including cortical signs (eg, aphasia, agnosia, neglect). Blood flow reversal in the vertebral artery is not characteristic.

(Choice C) Coronary-subclavian steal phenomenon occurs in patients with prior coronary artery bypass surgery using the internal mammary artery (IMA). Similar to that of subclavian steal syndrome, blood flow through the IMA can reverse and steal flow from the coronary circulation during increased demand (eg, upper extremity exercise). However, symptoms are typically related to coronary ischemia (eg, angina pectoris).

(Choice E) Right vertebral artery occlusion usually leads to ischemic stroke or TIA and can cause similar symptoms as seen in this patient. However, occlusion of the right vertebral artery would cause retrograde flow on the right versus the left, and symptoms are not typically precipitated by arm exertion.

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Educational objective:
Subclavian steal syndrome occurs due to severe stenosis of the proximal subclavian artery, which leads to reversal in blood flow from the contralateral vertebral artery to the ipsilateral vertebral artery. Patients may have symptoms related to arm ischemia in the affected extremity (eg, exercise-induced fatigue, pain, paresthesias) or vertebrobasilar insufficiency (eg, dizziness, vertigo).

References

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A 20-year-old college student dies suddenly in his sleep. His friends say he appeared fatigued and described palpitations and dyspnea with exertion in the weeks leading up to the event. He had no known medical history and did not use tobacco, alcohol, or illicit drugs. He has one brother who is healthy. His mother has a history of heart failure and underwent heart transplantation at age 40. Genetic studies reveal a defect in the *TTN* gene encoding for the titin protein. Autopsy findings are most likely to show which of the following phenotypes?

- A. Arrhythmogenic right ventricular cardiomyopathy
- B. Bicuspid aortic valve
- C. Dilated cardiomyopathy
- D. Hypertrophic cardiomyopathy
- E. Restrictive cardiomyopathy

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- D. Hypertrophic cardiomyopathy
- E. Restrictive cardiomyopathy

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Correct answer
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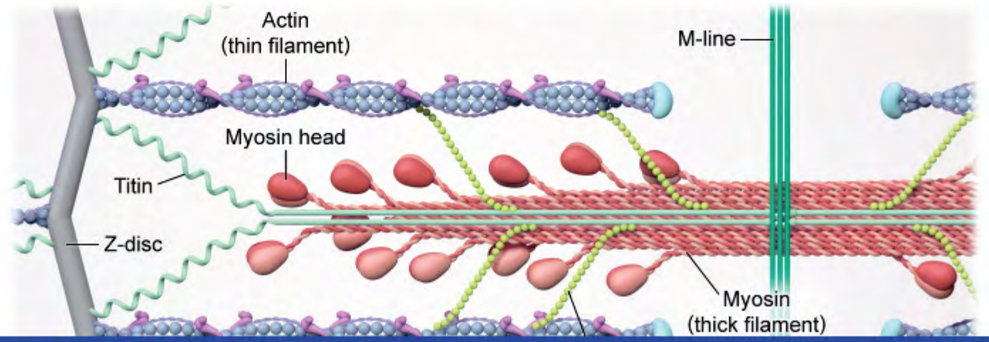
Explanation

Cardiac sarcomere protein structure



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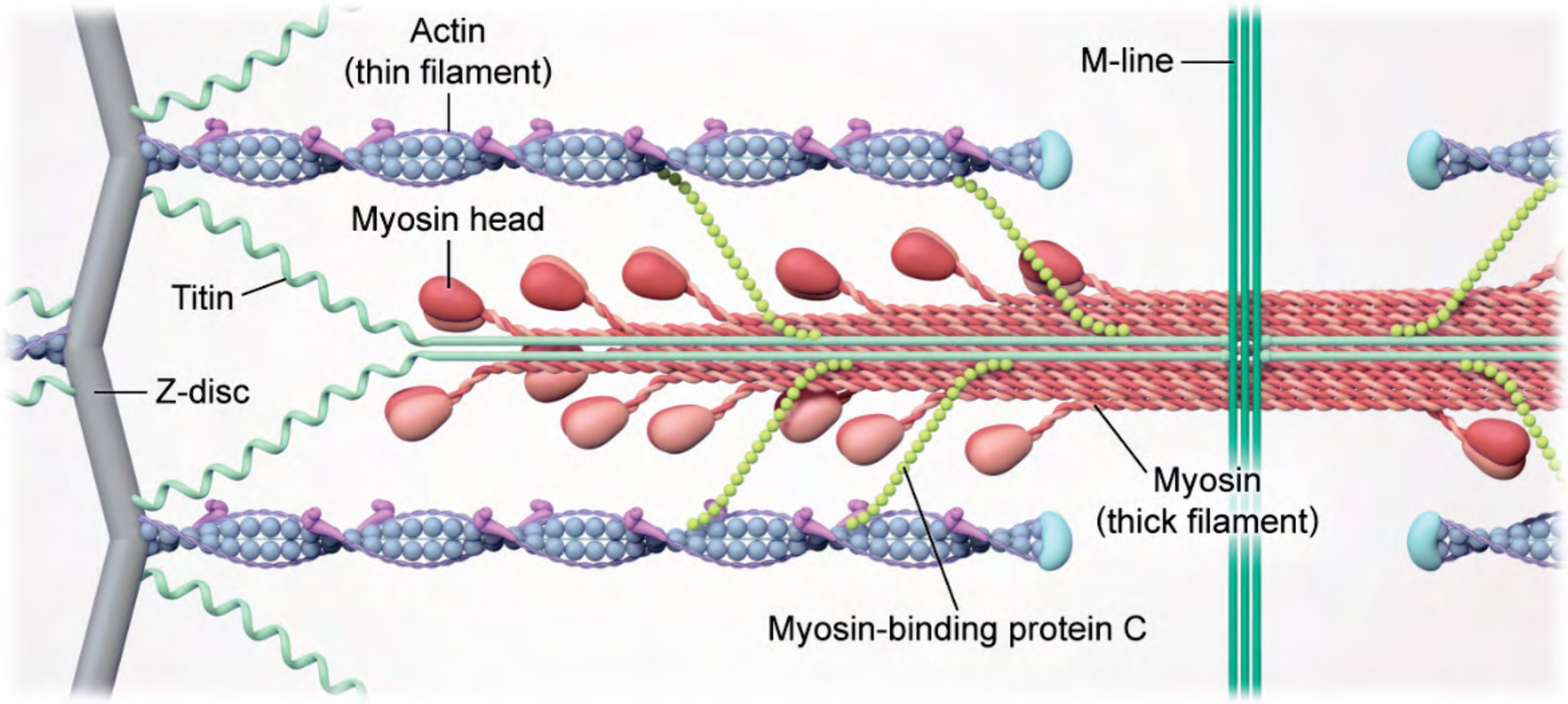

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Dilated cardiomyopathy (DCM) occurs due to a direct insult to the myocardium that leads to a decrease in contractile function of one or both ventricles with a consequent increase in ventricular cavity size. It can result from a variety of causes, including viral infection, chemical toxicity (eg, doxorubicin, alcohol, cocaine), and infiltrative disease (late manifestation). Patients typically have symptoms of decompensated heart failure (eg, fatigue, dyspnea, orthopnea). In addition, the left ventricular structural changes place these patients at risk for sudden cardiac death due to **ventricular arrhythmia**.

Patients are classified as having idiopathic DCM when no apparent cause of DCM is identified on routine clinical evaluation. A large percentage of these patients likely have a familial (inherited) cause. **Familial DCM** can result from a variety of genetic mutations affecting either sarcomere (ie, the contractile apparatus) or nonsarcomere proteins. Truncating mutations (usually nonsense mutations) affecting the ***TTN* gene**, which encodes for the sarcomere protein **titin**, are the most common cause of familial DCM. Titin is an elastic protein that anchors the beta-myosin heavy chain to the Z-discs and likely contributes to passive myocardial tension; absence of the full titin protein leads to myocardial dysfunction.

TTN gene mutations follow **autosomal dominant** inheritance; however, they have incomplete penetrance, leading to delayed or absent clinical manifestations in some family members.

(Choice A) Arrhythmogenic right ventricular cardiomyopathy is characterized by fibrosis and scarring of right ventricular myocardium, which predisposes to ventricular arrhythmias and sudden cardiac death. Familial disease most commonly results from impaired desmosome function due to mutations in genes encoding desmosomal proteins (eg, plakoglobin, desmoplakin).

(Choice B) Familial bicuspid aortic valve disease has been associated with a mutation affecting the *NOTCH1* gene that encodes transcription regulatory proteins.

(Choice D) Hypertrophic cardiomyopathy is a common cause of sudden cardiac death in young patients. It is inherited in an autosomal dominant pattern and most commonly involves mutations in the genes encoding beta-myosin heavy chain or myosin-binding protein C.

(Choice E) Restrictive cardiomyopathy most commonly results from infiltrative disease (eg, amyloidosis,

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(Choice E) Restrictive cardiomyopathy most commonly results from infiltrative disease (eg, amyloidosis, hemochromatosis, sarcoidosis); however, familial disease does occur. Familial restrictive cardiomyopathy typically involves mutations in genes encoding one of several sarcomere or cytoskeletal proteins, but mutations of the *TTN* gene are not a well-established cause.

Educational objective:
Autosomal dominant mutations in the *TTN* gene, which encodes for the sarcomere protein titin, are the most common cause of familial dilated cardiomyopathy.

References

- Truncations of titin causing dilated cardiomyopathy.

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A 5-year-old boy is brought to the office by his parents for evaluation of cyanosis with minimal exertion. The boy has had occasional episodes of "turning blue" that first began during infancy and are now occurring more frequently. During the episodes, the boy assumes a squatting position as it makes him "feel better." The family recently immigrated to the United States; the boy has never had a medical checkup prior to this visit. Physical examination reveals a prominent right ventricular impulse and a harsh systolic murmur. Which of the following embryological events is the most likely mechanism that caused this patient's condition?

- A. Anomalous pulmonary venous return
- B. Aortic arch constriction
- C. Deviation of the infundibular septum
- D. Endocardial cushion defect
- E. Linear development of the aorticopulmonary septum

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- ✗ A. Anomalous pulmonary venous return (5%)
- B. Aortic arch constriction (4%)
- ✓ C. Deviation of the infundibular septum (42%)
- D. Endocardial cushion defect (34%)
- E. Linear development of the aorticopulmonary septum (12%)

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Correct answer
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42%
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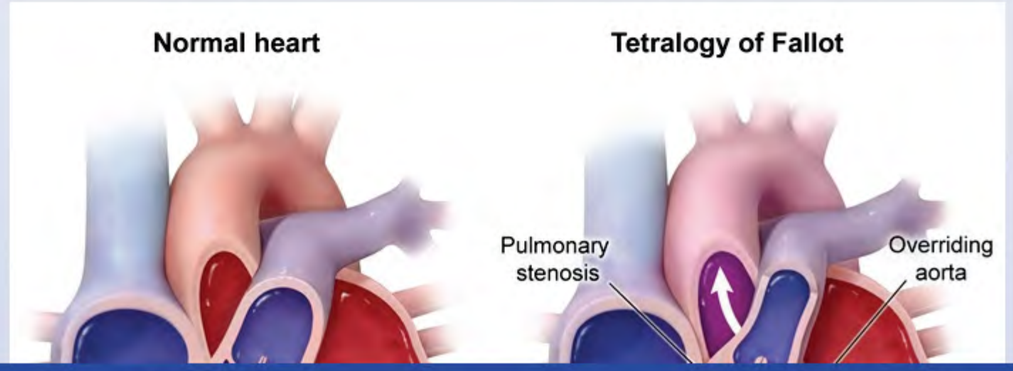

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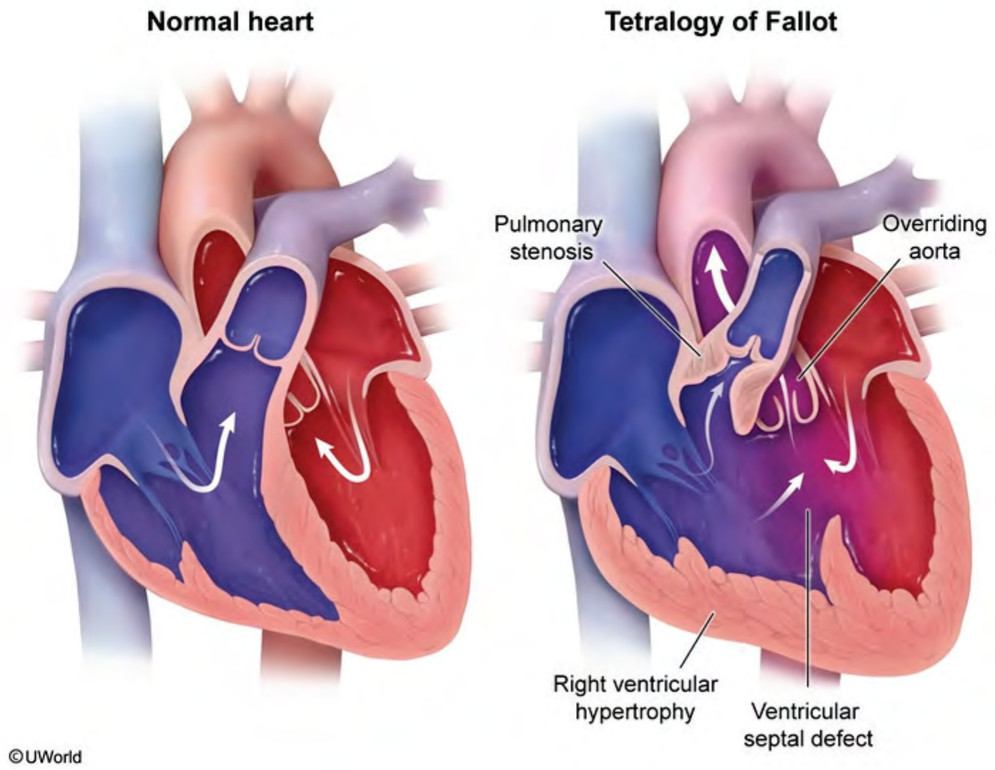

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This patient's clinical presentation (**cyanotic spells** that improve with **squatting**, prominent right ventricular impulse, **systolic murmur**) is consistent with tetralogy of Fallot (TOF). Abnormal neural crest cell migration leads to anterior and cephalad deviation of the infundibular septum during embryologic development, resulting in a malaligned VSD and an overriding aorta. TOF is characterized by 4 distinct anatomic abnormalities:

- Ventricular septal defect (VSD)
- Overriding aorta over the right and left ventricles
- Right ventricular outflow tract (RVOT) obstruction
- Right ventricular hypertrophy

Cyanosis occurs due to the presence of right-to-left shunt in patients with severe or worsening **RVOT obstruction**. The typical harsh, systolic ejection murmur over the mid-to-left upper sternal border is due to the presence of RVOT obstruction (subvalvular, pulmonary valve stenosis or supravalvular narrowing in the main pulmonary artery). Squatting increases the peripheral systemic vascular resistance (afterload) and decreases the degree of right-to-left shunting across the VSD, thereby improving cyanosis.

(Choice A) In anomalous pulmonary venous return, blood from both the pulmonary (oxygenated) and systemic (deoxygenated) venous systems flow into the right atrium, leading to right atrial and ventricular dilation. Patients also have obligatory right-to-left atrial shunting.

(Choice B) Aortic arch constriction results in coarctation of the aorta and is most commonly located just distal to the left subclavian artery (juxtaductal). Common manifestations include brachial-femoral pulse delay and blood pressure discrepancy between the upper and lower extremities.

(Choice D) Failed fusion of the superior and inferior endocardial cushions (eg, endocardial cushion defect) results in defects of the atrioventricular septum and valves (mitral and tricuspid valves). These typically manifest initially as atrial defects and/or VSDs with left-to-right shunting of blood. Over time, the increased right-sided blood flow leads to pulmonary hypertension with reversal of blood flow through the shunt and development of cyanosis (Eisenmenger syndrome). This patient has had cyanotic symptoms since birth that would not be expected with an isolated endocardial cushion defect.

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- Right ventricular hypertrophy

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(Choice E) Transposition of the great arteries results from linear, rather than spiral, development of the aorticopulmonary septum in utero.

Educational objective:
Tetralogy of Fallot results from anterior and cephalad deviation of the infundibular septum during embryologic development, resulting in a malaligned ventricular septal defect (VSD) with an overriding aorta. As a result, the patient has right ventricular outflow obstruction (resulting in a systolic murmur) and squats to increase the peripheral systemic vascular resistance (afterload) and decrease right-to-left shunting across the VSD.

References

- Evaluation and management of heart murmurs in children

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- Right ventricular hypertrophy

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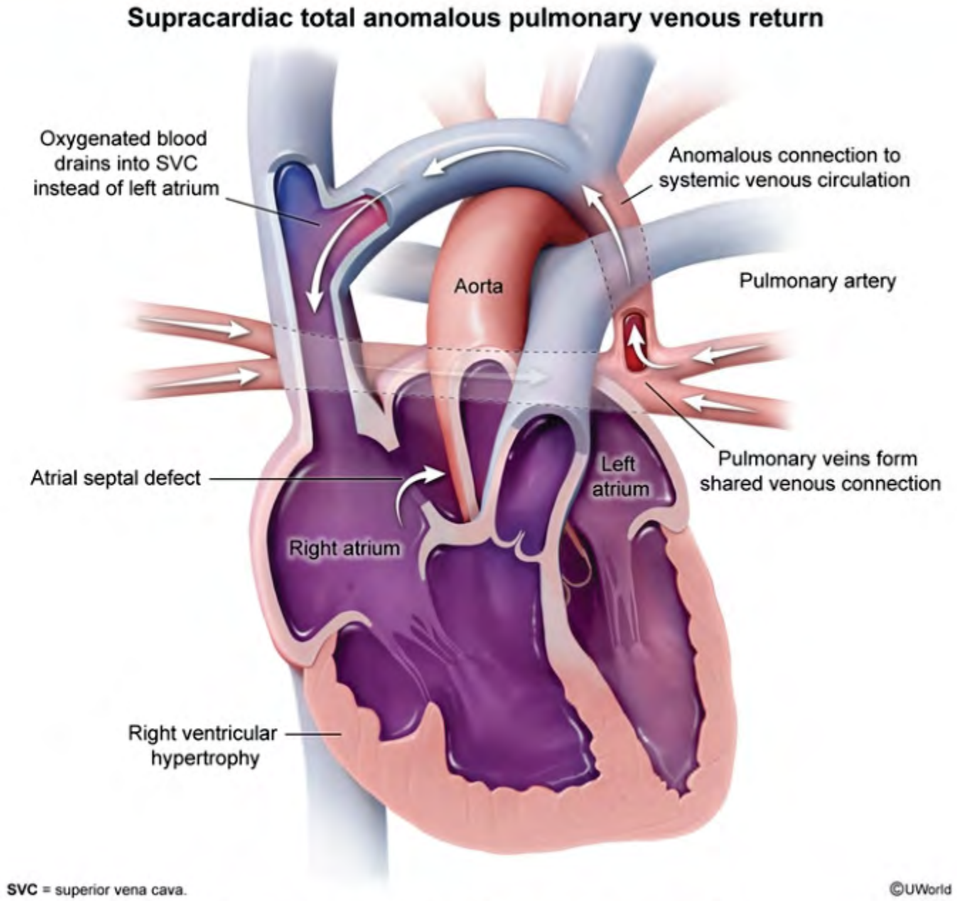

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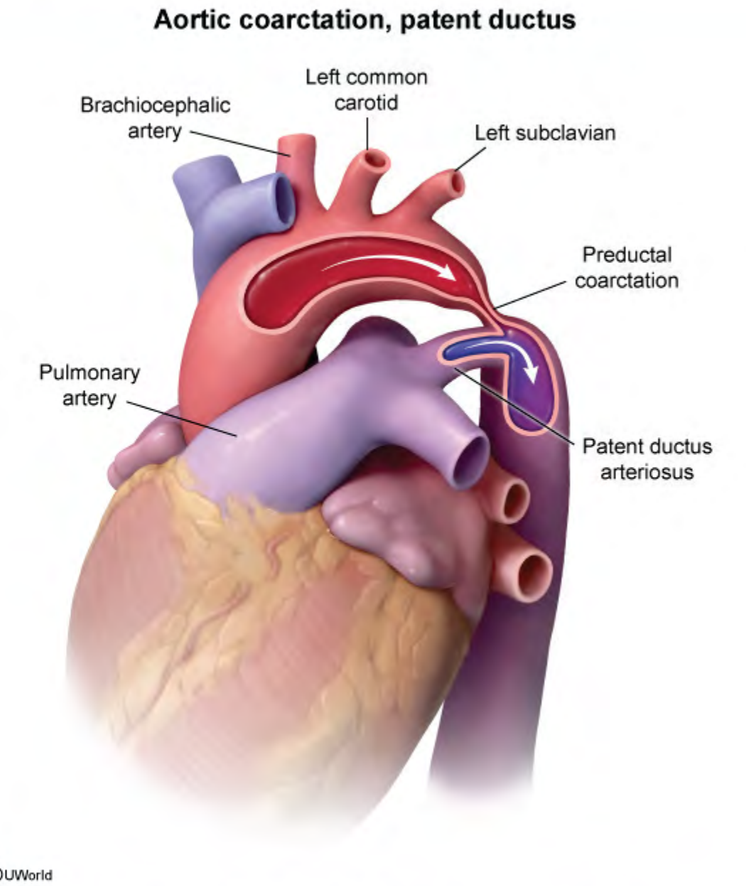

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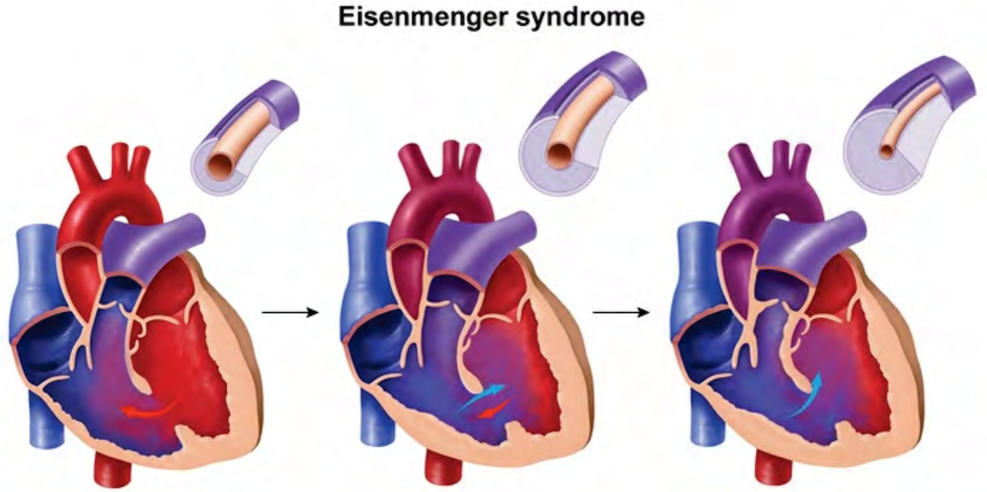

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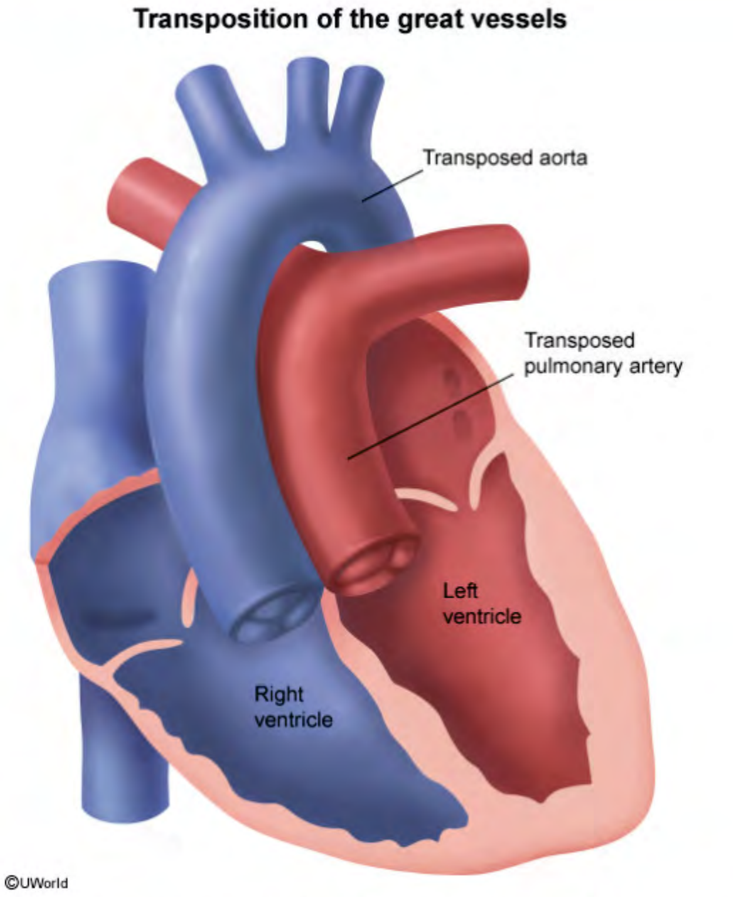

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A 35-year-old woman comes to the office due to solid food dysphagia for the past several months. The patient has a sensation of food getting stuck in the mid-thorax, but has no pain or reflux. She also has palpitations and dyspnea on exertion. The patient immigrated to the United States from India 1 year ago, and was treated in her home country for rheumatic heart disease. Barium esophagram demonstrates extrinsic compression on the mid-esophagus. Enlargement of which of the following is most likely causing this patient's dysphagia?

- A. Left atrium
- B. Left ventricle
- C. Pulmonary artery
- D. Right atrium
- E. Right ventricle

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- ✓ A. Left atrium (86%)
- B. Left ventricle (3%)
- C. Pulmonary artery (1%)
- D. Right atrium (7%)
- E. Right ventricle (1%)

Correct

86% Answered correctly

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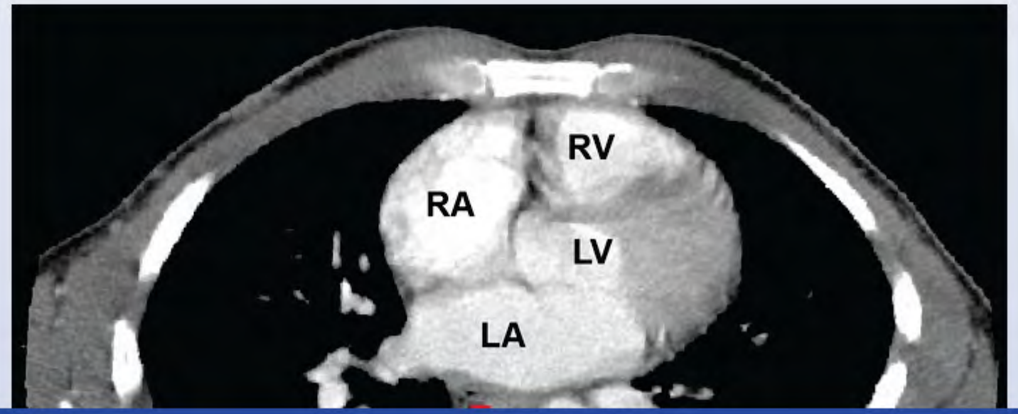
Explanation



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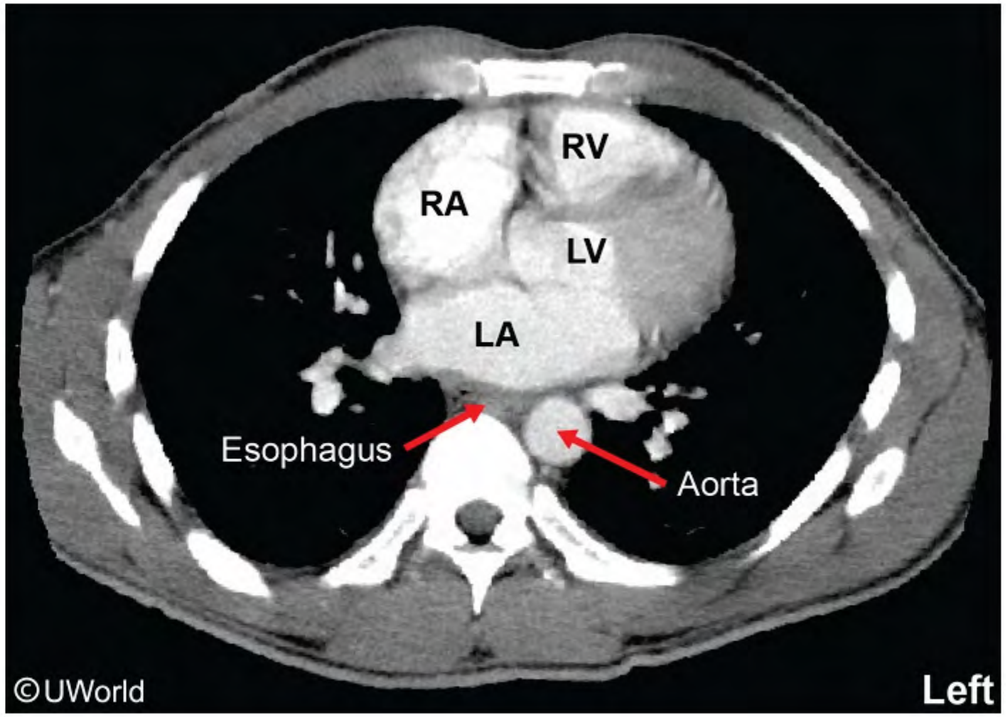

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This patient with a history of **rheumatic heart disease** has likely developed mitral stenosis/regurgitation (eg, palpitations, exertional dyspnea), leading to left atrial enlargement and extrinsic compression of the esophagus (eg, solid food dysphagia).

The **posterior surface** of the heart is mostly formed by the left atrium, which lies directly over the **esophagus**. Longstanding **mitral stenosis/regurgitation** can lead to progressive **left atrial enlargement**, further displacing the left atrium posteriorly and causing external compression of the mid-esophagus with dysphagia (**cardiovascular dysphagia**). On rare occasions, left atrial enlargement can also cause compression of the left recurrent laryngeal nerve, leading to voice hoarseness and chronic cough.

(Choice B) The left ventricle makes up most of the left surface of the heart, which abuts the left lung.

(Choice C) The pulmonary artery emerges from the right ventricle and branches into the right and left pulmonary arteries below the aortic arch.

(Choices D and E) The anterior (sternocostal) surface of the heart is mostly formed by the right atrium superiorly and right ventricle inferiorly. Blood from the coronary sinus and inferior and superior venae cavae drain into the right atrium, which then drains blood into the right ventricle through the tricuspid valve.

Educational objective:
Cardiovascular dysphagia can result from external compression of the esophagus by a dilated and posteriorly displaced left atrium in patients with rheumatic heart disease and mitral stenosis/regurgitation.

References

- An unusual case of dysphonia and dysphagia.

Anatomy	Cardiovascular System	Left atrial enlargement
Subject	System	Topic

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A 6-month-old boy is brought to the emergency department due to lethargy for several hours. His mother says he is usually a "very active baby," and she is afraid that he is "coming down with something." The patient's birth history is unremarkable, and he was born at 38 weeks gestation. He is afebrile. Blood pressure is 70/44 mm Hg, pulse is 240 beats/min, and respirations are 52/min. The patient appears pale and is perspiring. Capillary refill time is less than 2 seconds. The lungs are clear to auscultation. No murmurs are heard. An ECG shows a regular heart rate of 240/min with narrow QRS complexes and no identifiable P waves. Which of the following is the most likely cause of this patient's hypotension?

- A. Decreased effective intravascular volume
- B. Decreased myocardial contractility
- C. Decreased peripheral vascular resistance
- D. Left-to-right intracardiac shunting
- E. Shortening of ventricular diastole

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- ✗ A. Decreased effective intravascular volume (9%)
- B. Decreased myocardial contractility (7%)
- C. Decreased peripheral vascular resistance (10%)
- D. Left-to-right intracardiac shunting (6%)
- ✓ E. Shortening of ventricular diastole (65%)

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Correct answer
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65%
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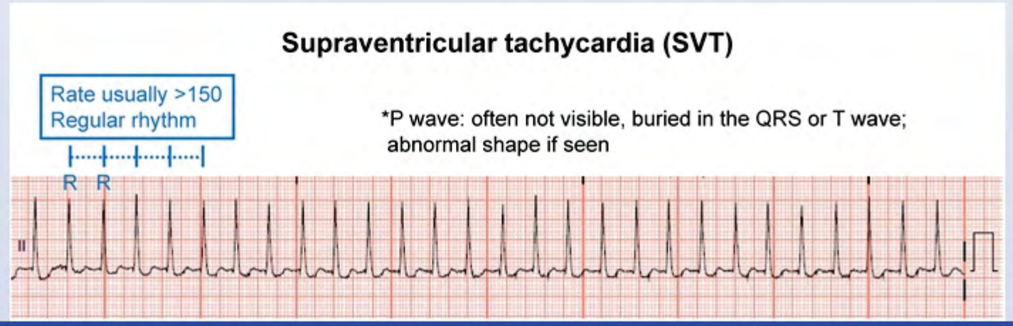

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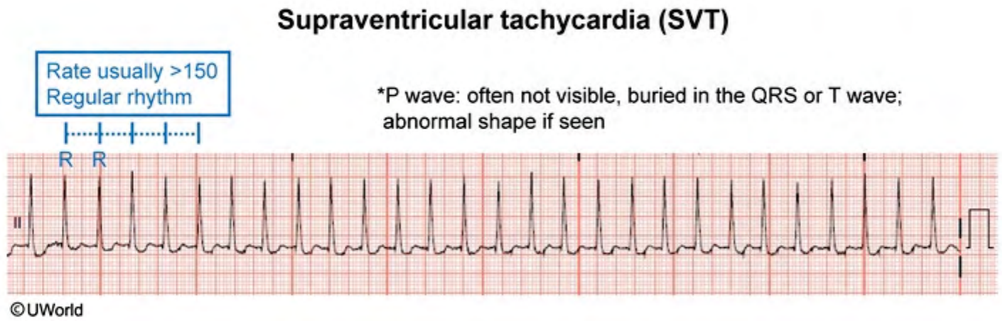

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This patient is pale, lethargic, and diaphoretic with ECG findings consistent with **supraventricular tachycardia** (SVT), the most common pediatric arrhythmia.

Normally, depolarization impulses originate at the sinoatrial node and travel through the atrial myocardium to the atrioventricular (AV) node, where the signal pauses to allow ventricular filling following atrial contraction. The impulse then continues through the His-Purkinje system to the ventricular myocardium.

SVT occurs when an impulse originates at an abnormal focus above the ventricles, most commonly a reentrant circuit within the AV node (ie, AV nodal reentry tachycardia). **Heart rate** is **elevated** (usually >150/min in adults or >220/min in infants), but rhythm is usually regular. **QRS complexes are narrow**, and **P waves** are generally **buried** within the QRS complex due to retrograde atrial depolarization occurring in conjunction with anterograde ventricular depolarization.

With persistent tachycardia, **ventricular diastole shortens**, allowing less time for ventricular relaxation and filling. Stroke volume and cardiac output decrease, leading to **hypotension** and poor peripheral perfusion. Infants may become altered (eg, lethargic, poor feeding) and develop signs of heart failure (eg, tachypnea, crackles, hepatomegaly).

(Choice A) Decreased effective intravascular volume is the mechanism for hypotension associated with hypovolemic shock. This patient has normal capillary refill without a history of volume depletion (eg, diarrhea, vomiting, blood loss).

(Choice B) Hypotension due to decreased myocardial contractility is seen with a myocardial infarction, which is extremely rare in infancy, or a viral-induced cardiomyopathy, which is unlikely in the absence of infectious symptoms (eg, fever, rhinorrhea).

(Choice C) Low cardiac output associated with SVT would lead to a compensatory increase, not a decrease, in peripheral vascular resistance.

(Choice D) A large left-to-right intracardiac shunt (eg, ventricular septal defect [VSD]) can cause hypotension and tachycardia, but signs of heart failure (eg, crackles, hepatomegaly) would be expected. In addition, depending on

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(Choice D) A large left-to-right intracardiac shunt (eg, ventricular septal defect [VSD]) can cause hypotension and tachycardia, but signs of heart failure (eg, crackles, hepatomegaly) would be expected. In addition, depending on the size of VSD, ECG may show left or biventricular hypertrophy, not narrow QRS complexes with absent P waves.

Educational objective:
Supraventricular tachycardia is characterized by an elevated heart rate with a regular rhythm and narrow QRS complexes due to an abnormal electrical impulse originating above the ventricles. Sustained tachycardia shortens ventricular diastole, leading to decreased stroke volume and cardiac output and subsequent hypotension.

References

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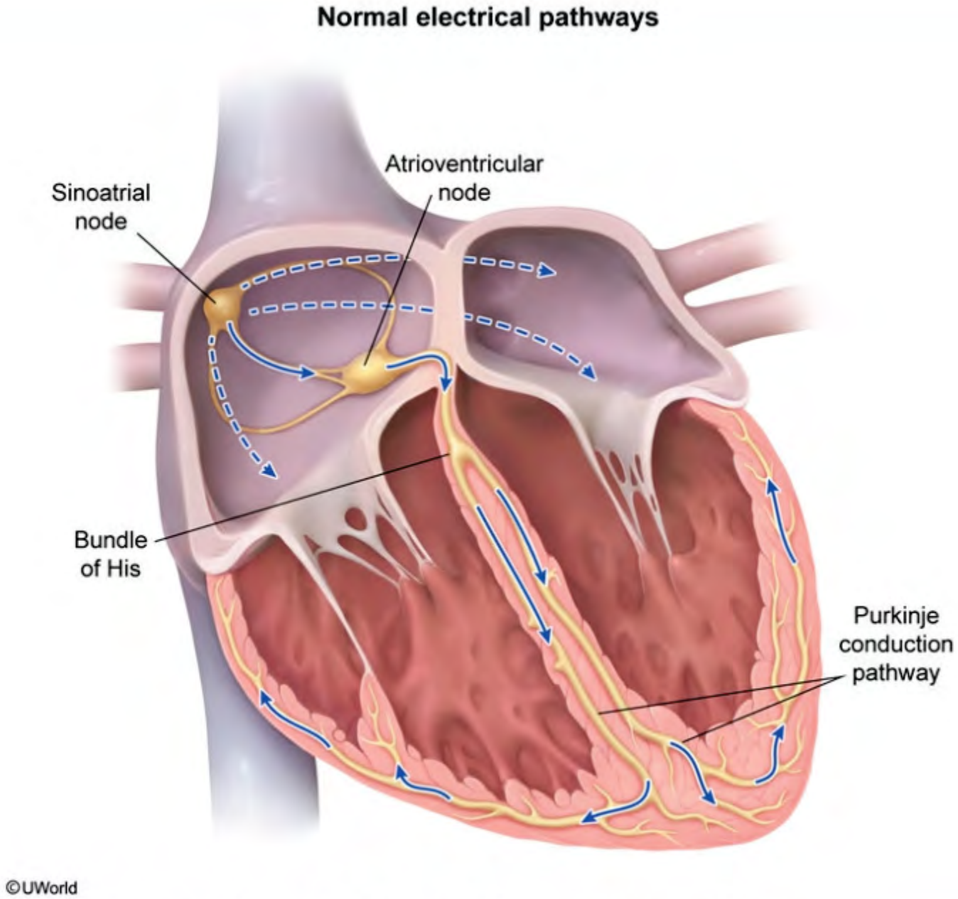

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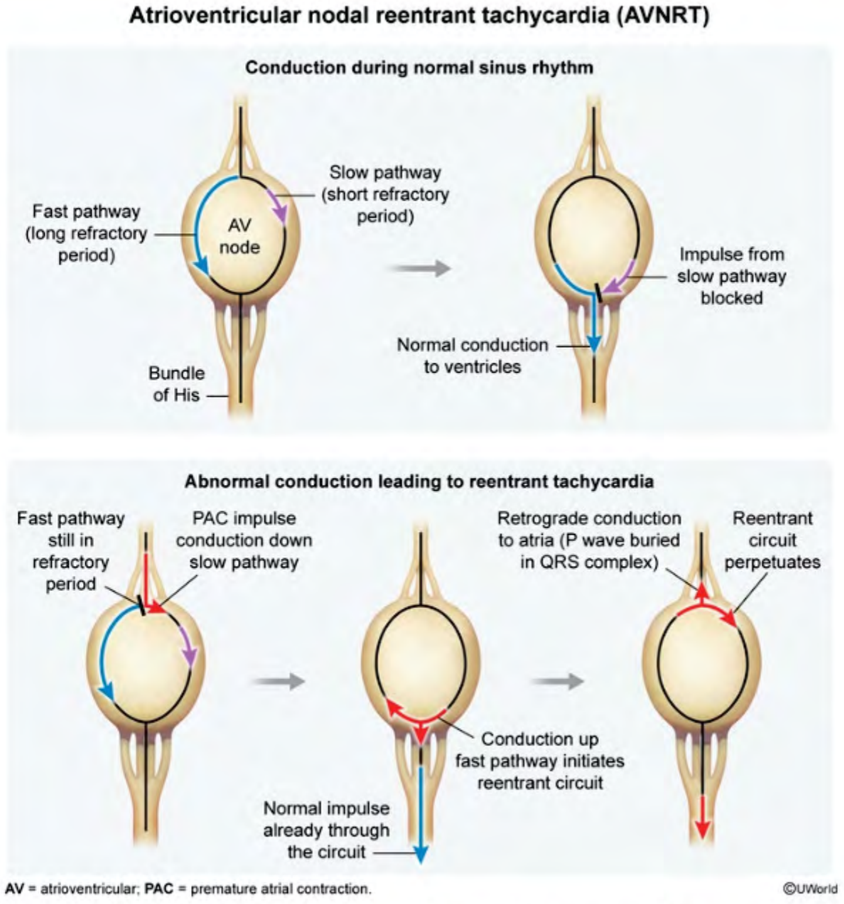

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A 54-year-old man is evaluated for paroxysmal atrial fibrillation. During the episodes, the patient experiences palpitations, chest tightness, light-headedness, and shortness of breath. Medical history is significant for asthma, hypertension, and diet-controlled diabetes mellitus. Prior testing showed normal left ventricular ejection fraction, no evidence of valvular disease, and no obstructive coronary artery disease. It is decided to proceed with dofetilide therapy. Initiation of this therapy increases this patient's risk for which of the following?

- A. Bronchospasm
- B. Complete atrioventricular block
- C. Polymorphic ventricular tachycardia
- D. Thyroid dysfunction
- E. Venous thromboembolism
- F. Visual disturbance

Submit

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- A. Bronchospasm (4%)
- ✗ B. Complete atrioventricular block (20%)
- ✓ C. Polymorphic ventricular tachycardia (63%)
- D. Thyroid dysfunction (7%)
- E. Venous thromboembolism (1%)
- F. Visual disturbance (3%)

Incorrect
Correct answer
C

63%
Answered correctly

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Explanation

Pharmacotherapy for supraventricular arrhythmias	
Medication	**Short-term adverse effects**
Flecainide/propafenone (class IC)	• Monomorphic VT*

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Pharmacotherapy for supraventricular arrhythmias	
Medication	**Short-term adverse effects**
Flecainide/propafenone (class IC)	• Monomorphic VT*
Metoprolol (class II)	• Bradycardia & advanced AV block
Amiodarone (class III)	• Bradycardia • Hepatic toxicity, thyroid dysfunction
Ibutilide/dofetilide/sotalol (class III)	• Torsade de pointes
Verapamil/diltiazem (class IV)	• Bradycardia & advanced AV block • Decreased ventricular contractility • Constipation (verapamil)
Adenosine	• Flushing & hypotension • Bronchospasm • High-grade AV block
Digoxin	• Bradycardia & other arrhythmias • Nausea & vomiting ±

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Metoprolol (class II)	• Bradycardia & advanced AV block
Amiodarone (class III)	• Bradycardia • Hepatic toxicity, thyroid dysfunction
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Verapamil/diltiazem (class IV)	• Bradycardia & advanced AV block • Decreased ventricular contractility • Constipation (verapamil)
Adenosine	• Flushing & hypotension • Bronchospasm • High-grade AV block
Digoxin	• Bradycardia & other arrhythmias • Nausea & vomiting ± visual disturbances
*Especially a concern with ischemic or structural heart disease. **AV** = atrioventricular; **VT** = ventricular tachycardia.	

Relatively young patients with **symptomatic paroxysmal atrial fibrillation** are often treated with a **rhythm-control** strategy to restore and maintain normal sinus rhythm and to eliminate symptoms. Antiarrhythmic drugs commonly used for rhythm control of atrial fibrillation include class IC (eg, flecainide, propafenone) and class III

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Dofetilide and ibutilide selectively block the rapid component of the delayed-rectifier potassium current. This **slows repolarization** and prolongs action potential duration and the **effective refractory period** in cardiomyocytes, suppressing the electrical foci that lead to atrial fibrillation. As a result, the **QT interval is prolonged**, leading to an increased risk of **torsade de pointes**, a form of **polymorphic ventricular tachycardia** that can cause syncope and sudden cardiac death. To minimize this risk, dofetilide or ibutilide therapy is started only in the hospital with temporary cardiac monitoring.

(Choice A) Adenosine can trigger bronchospasm, likely by stimulating the release of histamine and leukotrienes from mast cells. Class III antiarrhythmics are not associated with bronchospasm.

(Choice B) Beta blockers (via inhibition of beta-1 receptor–driven chronotropy) and nondihydropyridine calcium channel blockers (via blockade of L-type atrioventricular [AV] node calcium channels) slow AV node conduction and in overdose can cause complete AV block. Digoxin (via enhanced vagal tone) and adenosine can also cause AV block. Although some class III antiarrhythmics (ie, sotalol, amiodarone) have negative chronotropic effects and may slow AV node conduction, dofetilide and ibutilide have no significant effect on AV node conduction.

(Choice D) Amiodarone can disrupt thyroid function and lead to hyper- or hypothyroidism, but dofetilide and other class III antiarrhythmics do not affect thyroid function. Compared with other class III antiarrhythmics, amiodarone is associated with a lower risk of torsade de pointes.

(Choice E) Most antiarrhythmic drugs, including class III, are not associated with an increased risk of venous thromboembolism.

(Choice F) Digoxin toxicity can cause visual disturbances consisting of blurred or yellow vision, but visual disturbance is not an adverse effect of class III antiarrhythmics.

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Educational objective:
Dofetilide and ibutilide are class III antiarrhythmics that treat atrial fibrillation by blocking the rapid component of the delayed-rectifier potassium current to slow repolarization and increase the effective refractory period. As a result, the QT interval is prolonged, creating an increased risk of polymorphic ventricular tachycardia (torsade de pointes).

References

- 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American

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- Bradycardia & other

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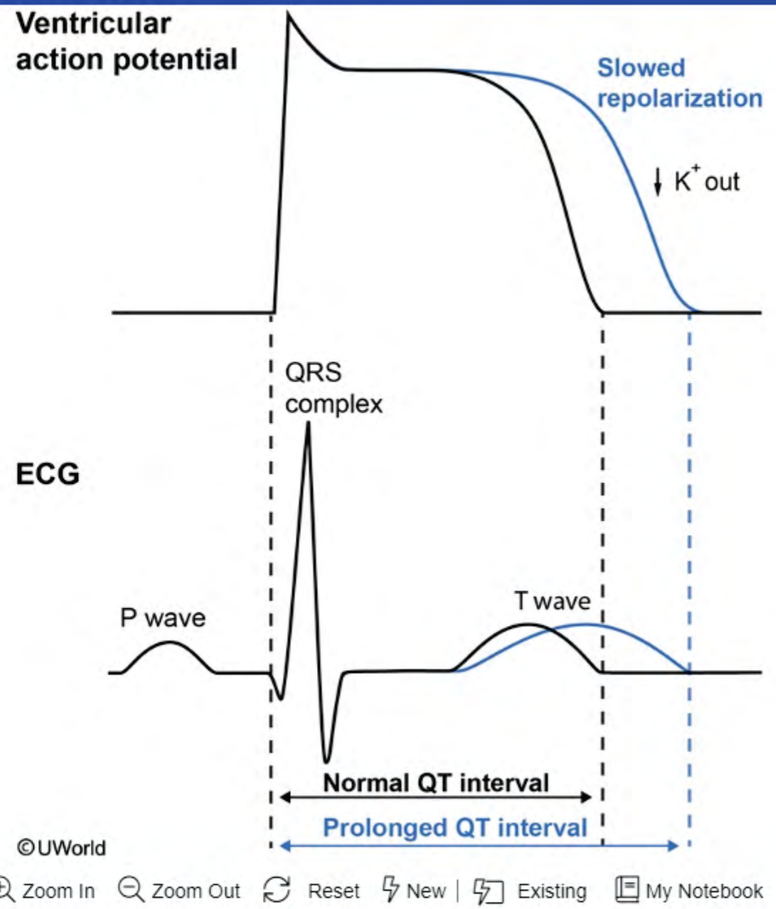

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(Choice D) Amiodarone can disrupt thyroid function and lead to hyper- or hypothyroidism, but dofetilide and other

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A 20-year-old man comes to the office due to occasional palpitations that he describes as skipped beats. He has no chest pain or shortness of breath. Medical history is unremarkable. On physical examination, the patient has wide, fixed splitting of S2. No murmurs are heard. Chest x-ray reveals enlargement of the right heart contour. Echocardiographic findings are consistent with a congenital heart defect. Repair of this patient's congenital disease is aimed primarily at preventing irreversible changes to which of the following?

- A. Coronary vessels
- B. Left atrium
- C. Left ventricle
- D. Pulmonary vessels
- E. Right atrium

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- ✗ A. Coronary vessels (1%)
- B. Left atrium (6%)
- C. Left ventricle (7%)
- ✓ D. Pulmonary vessels (40%)
- E. Right atrium (44%)

Incorrect
Correct answer
D

40%
Answered correctly

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Explanation

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A 20-year-old man comes to the office due to occasional palpitations that he describes as skipped beats. He has no che
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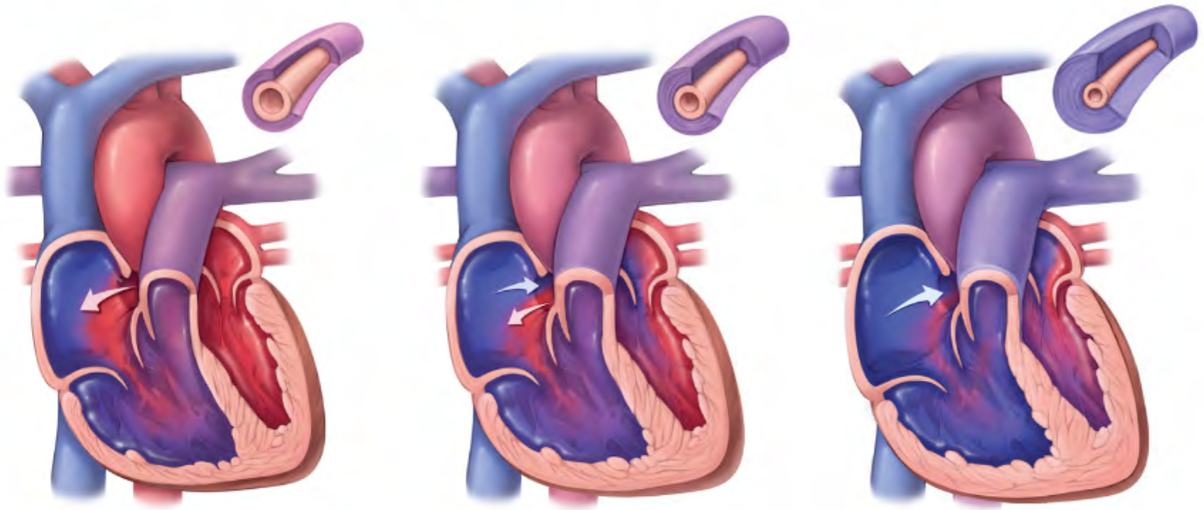
Eisenmenger syndrome



Congenital defect (eg, ASD) allows left-to-right shunt

Over time, ↑ PVR leads to bidirectional flow

PVR continues to increase with eventual continuous right-to-left shunt (Eisenmenger syndrome)

ASD = atrial septal defect; **PVR** = pulmonary vascular resistance.

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ASD = atrial septal defect; **PVR** = pulmonary vascular resistance.

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This patient has **wide, fixed splitting of S2**, a characteristic auscultatory finding of an **atrial septal defect** (ASD). ASD permits left-to-right shunting due to higher pressure in the left atrium compared to the right. The increase in right atrial blood flow causes **right-sided volume overload**, leading to delayed closure of the pulmonic valve (wide S2) that does not vary with respirations (fixed S2). The low-velocity blood flow through the ASD does not typically produce a murmur; the characteristic murmur of ASD, present in some patients, is a midsystolic ejection murmur resulting from increased flow across the pulmonic valve.

Right-sided volume overload eventually leads to right atrial and ventricular **enlargement**, which may be seen as an enlarged right heart contour on chest x-ray. **Atrial fibrillation**, which is the likely cause of this patient's palpitations, is a potential complication resulting from chronic right atrial stretching.

With long-standing ASD, the chronic increase in blood flow through the pulmonary circulation induces laminated **medial hypertrophy** of the pulmonary arteries. This increases pulmonary vascular resistance (PVR), causing **pulmonary hypertension**. Eventually, PVR can exceed systemic vascular resistance, reversing the direction of the intracardiac shunt (Eisenmenger syndrome); this allows blood to bypass the pulmonary circulation and can cause adult-onset cyanosis, clubbing, and polycythemia.

Pulmonary **vascular remodeling** is **irreversible**. Therefore, early closure of a hemodynamically significant ASD is needed to avoid irreversible pulmonary vascular changes.

(Choices A, B, and C) ASD does not typically cause volume overload or enlargement of the left heart chambers because excess blood flow is shunted to the right atrium before reaching the left ventricle. The coronary vessels are not typically affected.

(Choice E) ASD causes right atrial dilation, but the consequence of this change is far less than that of the pulmonary vascular changes. In addition, right atrial dilation is mostly reversible because, unlike in the pulmonary arteries and often the right ventricle, significant muscular hypertrophy does not take place.

Educational objective:

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Educational objective:
Atrial septal defects cause wide, fixed splitting of S2 due to right-sided volume overload from left-to-right shunting. Uncorrected defects can lead to irreversible medial hypertrophy of the pulmonary arteries with pulmonary hypertension and reversal to right-to-left shunting (ie, Eisenmenger syndrome).

References

- Atrial septal defect (ASD).

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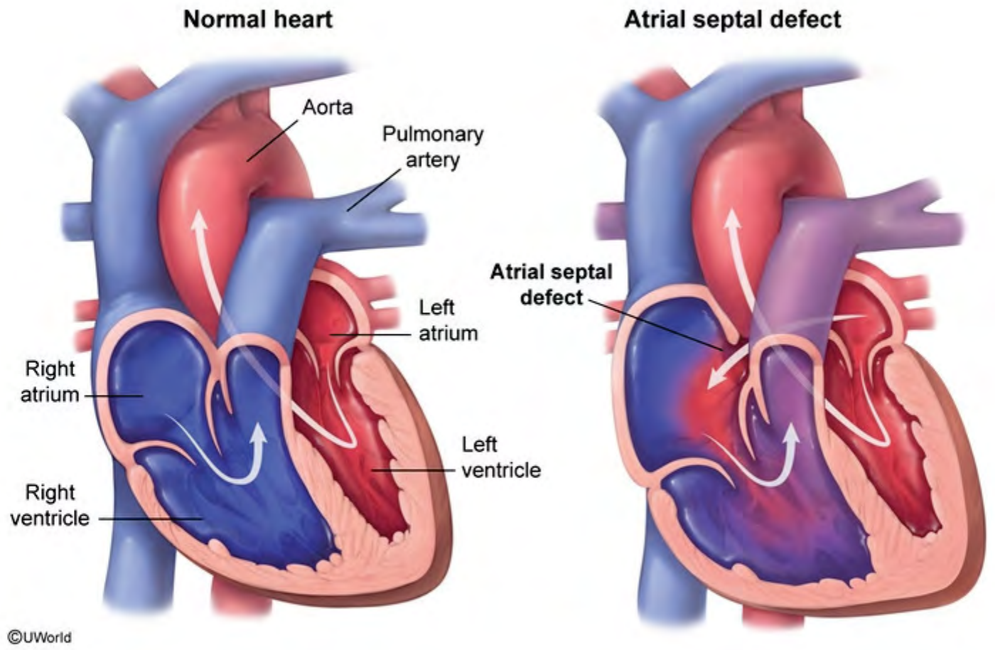

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Physiologists conducting research on the electrical properties of the heart measure action potential conduction velocity at 4 different points within normal cardiac tissue. The results, expressed in terms of speed of conduction (meters per second), are as follows:

Point 1 – 0.05 m/sec
Point 2 – 0.3 m/sec
Point 3 – 1.1 m/sec
Point 4 – 2.2 m/sec

From the following list of locations, which most likely corresponds to the order of points 1-2-3-4 (AV = atrioventricular)?

- A. Atrial muscle, ventricular muscle, Purkinje system, AV node
- B. AV node, Purkinje system, ventricular muscle, atrial muscle
- C. AV node, ventricular muscle, atrial muscle, Purkinje system
- D. Purkinje system, AV node, ventricular muscle, atrial muscle
- E. Ventricular muscle, AV node, Purkinje system, atrial muscle

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Physiologists conducting research on the electrical properties of the heart measure action potential conduction velocity at 4 different points within normal cardiac tissue. The results, expressed in terms of speed of conduction (meters per second), are as follows:

Point 1 – 0.05 m/sec
Point 2 – 0.3 m/sec
Point 3 – 1.1 m/sec
Point 4 – 2.2 m/sec

From the following list of locations, which most likely corresponds to the order of points 1-2-3-4 (AV = atrioventricular)?

- ✗ A. Atrial muscle, ventricular muscle, Purkinje system, AV node (15%)
- B. AV node, Purkinje system, ventricular muscle, atrial muscle (15%)
- ✓ C. AV node, ventricular muscle, atrial muscle, Purkinje system (49%)
- D. Purkinje system, AV node, ventricular muscle, atrial muscle (14%)
- E. Ventricular muscle, AV node, Purkinje system, atrial muscle (3%)

Incorrect
Correct answer
C

49%
Answered correctly

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Explanation

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Physiologists conducting research on the electrical properties of the heart measure action potential conduction velocit
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Point 1
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Cardiac tissue conduction velocity

Fastest

Purkinje system

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Atrial muscle

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Ventricular muscle

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AV node

Slowest

Mnemonic: "**Park At Venture Avenue**"

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Cardiac tissue conduction velocity

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Cardiac impulses normally originate in the sinoatrial (SA) node. SA node depolarization delivers an electrical impulse to the surrounding atrial myocardium, which carries the action potential to the atrioventricular (AV) node at a rate of 1.1 m/sec. Speed of conduction in the **AV node** is the **slowest** (0.05 m/sec), and this delay allows the ventricles to completely fill with blood during diastole. From the AV node, the action potential enters the His-Purkinje system. Impulses travel the **fastest** through the **Purkinje fibers** (2.2 m/sec), which ensures that the ventricles contract in a bottom-up fashion (necessary for efficient propulsion of blood into the pulmonary artery and aorta). From the Purkinje fibers, the action potential is transmitted to the ventricular myocardium, where it travels at a rate of 0.3 m/sec.

Points 1-2-3-4 are arranged in order of increasing **conduction speed** (not conduction time), as follows:

Point 1 – AV node (0.05 m/sec)
Point 2 – Ventricular muscle (0.3 m/sec)
Point 3 – Atrial muscle (1.1 m/sec)
Point 4 – Purkinje system (2.2 m/sec)

Educational objective:
The cardiac action potential conduction speed is slowest in the atrioventricular node and fastest in the Purkinje system. Conduction speed of the atrial muscle is faster than that of the ventricular muscle.

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

An 11-year-old boy is brought to the office for a routine checkup prior to enrollment at summer camp. The patient and his mother report no health concerns. Family history is significant for his father passing away suddenly from a heart condition 2 years ago. The patient is at the 75th and 80th percentiles for height and weight, respectively. Vital signs are normal. Physical examination shows a well-appearing child. The lungs are clear to auscultation. Cardiac auscultation findings at the left lower sternal border are given below. The auscultatory abnormality intensifies with the handgrip exercise. Which of the following is the most likely diagnosis?

Play Media

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Atrial septal defect
- D. Hypertrophic cardiomyopathy
- E. Patent ductus arteriosus
- F. Ventricular septal defect

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A 44-year-old previously healthy woman comes to the emergency department due to a 2-day history of left flank pain, dysuria, fever, and chills. Temperature is 38.9 C (102 F), blood pressure is 80/40 mm Hg, and pulse is 140/min and regular. Physical examination shows left costovertebral angle tenderness. Laboratory studies reveal leukocytosis, pyuria, and bacteriuria. Empiric broad-spectrum antibiotics and intravenous fluids are administered. The patient remains persistently hypotensive, and an intravenous phenylephrine infusion is begun. Several minutes later, her heart rate decreases to 100/min. Compared to just prior to the infusion, which of the following intracellular changes are expected in this patient?

	Cyclic AMP level in ventricular myocytes	Inositol trisphosphate level in vascular smooth muscle	Inward calcium current in sinoatrial nodal cells
○ A.	Decreased	Increased	Decreased
○ B.	Decreased	Decreased	No change
○ C.	Increased	Increased	Increased
○ D.	Increased	No change	Increased
○ E.	No change	Decreased	Decreased

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

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Play Media

- A. Aortic regurgitation (8%)
- ✗ B. Aortic stenosis (7%)
- C. Atrial septal defect (4%)
- D. Hypertrophic cardiomyopathy (24%)
- E. Patent ductus arteriosus (7%)
- ✓ F. Ventricular septal defect (46%)

Incorrect
Correct answer
F

46%
Answered correctly

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Time Spent

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Explanation

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	Cyclic AMP level in ventricular myocytes	Inositol trisphosphate level in vascular smooth muscle	Inward calcium current in sinoatrial nodal cells	
✓ A.	Decreased	Increased	Decreased	(59%)
B.	Decreased	Decreased	No change	(5%)
C.	Increased	Increased	Increased	(18%)
D.	Increased	No change	Increased	(8%)
E.	No change	Decreased	Decreased	(8%)

Correct

59% Answered correctly

06 secs Time Spent

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Explanation

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Explanation

Variation in intensity of cardiac murmurs			
Maneuver	**Change in cardiac parameters**	**Murmurs that increase**	**Murmurs that decrease**
Inspiration	↑ RV preload, ↓ LV preload	Most right-sided murmurs (↑ RV blood volume)	Most left-sided murmurs (↓ LV blood volume)
Valsalva strain phase	↓ RV & LV preload	MVP & HCM (↓ LV blood volume)	Most valvular murmurs (↓ flow through valve)
Abrupt standing			
Squatting	↑ RV & LV preload, ↑ LV afterload	Most valvular murmurs (↑ flow through valve)	MVP & HCM (↑ LV blood volume)
Passive leg raise	↑ RV & LV preload		
Hand grip	↑ LV afterload	AR, MR & VSD (↑ aortic & LV pressure)	MVP & HCM (↑ LV blood volume) AS (↓ transvalvular pressure gradient)
AR = aortic regurgitation; **AS** = aortic stenosis; **HCM** = hypertrophic cardiomyopathy; **LV** = left ventricular; **MR** = mitral regurgitation; **MVP** = mitral valve prolapse; **RV** = right ventricular; **VSD** = ventricular septal defect.			

The presentation of a **ventricular septal defect** (VSD) depends on its size. Large defects result in high-volume shunting of blood across the VSD, leading to a low interventricular pressure gradient and a soft or absent murmur; the large degree of shunting may lead to heart failure in infancy (eg, difficulty feeding, tachypnea, failure to thrive)

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The presentation of a **ventricular septal defect** (VSD) depends on its size. Large defects result in high-volume shunting of blood across the VSD, leading to a low interventricular pressure gradient and a soft or absent murmur; the large degree of shunting may lead to heart failure in infancy (eg, difficulty feeding, tachypnea, failure to thrive). In contrast, **small defects** allow only a small amount of blood to flow across the VSD; most patients are **asymptomatic** and have **loud murmurs** that are detected incidentally on cardiac auscultation.

This patient's harsh holosystolic murmur best heard at the left lower sternal border is indicative of blood flow through a small, hemodynamically insignificant VSD. The **handgrip maneuver** increases systemic vascular resistance, resulting in increased aortic and left ventricular (LV) pressure that **intensifies the murmur**.

(Choice A) Aortic regurgitation causes a decrescendo early *diastolic* murmur that is best heard at the left sternal border when due to valvular disease. Like a VSD murmur, an aortic regurgitation murmur is also accentuated by maneuvers that increase afterload (eg, handgrip).

(Choice B) Aortic stenosis causes a crescendo-decrescendo systolic murmur best heard at the right upper sternal border with radiation to the carotids. The murmur *decreases* with the handgrip maneuver due to increased afterload decreasing the transvalvular pressure gradient.

(Choice C) The typical murmur of an atrial septal defect is not caused by flow through the defect itself; instead, it results from increased right-sided blood volume with increased flow across the pulmonic valve (ie, pulmonic flow murmur). It is recognized as a faint systolic murmur best heard at the left upper sternal border and with wide and fixed splitting of S2.

(Choice D) Hypertrophic cardiomyopathy causes a systolic ejection murmur best heard at the left midsternal border. However, handgrip maneuver *decreases* the murmur intensity due to increased LV volume and consequently reduced LV outflow tract obstruction.

(Choice E) A patent ductus arteriosus produces a continuous flow murmur best heard at the left infraclavicular or posterior interscapular region. Affected patients usually develop symptoms (eg, poor feeding, respiratory difficulty) during early childhood.

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This patient's harsh holosystolic murmur best heard at the left lower sternal border is indicative of blood flow through a small, hemodynamically insignificant VSD. The **handgrip maneuver** increases systemic vascular resistance, resulting in increased aortic and left ventricular (LV) pressure that **intensifies the murmur**.

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(Choice E) A patent ductus arteriosus produces a continuous flow murmur best heard at the left infraclavicular or posterior interscapular region. Affected patients usually develop symptoms (eg, poor feeding, respiratory difficulty) during early childhood.

Educational objective:
A ventricular septal defect (VSD) commonly causes a harsh, holosystolic murmur at the left lower sternal border; the murmur increases in intensity during maneuvers that increase left ventricular afterload (eg, handgrip maneuver). A small VSD is usually asymptomatic and produces a relatively loud murmur due to a high interventricular pressure gradient.

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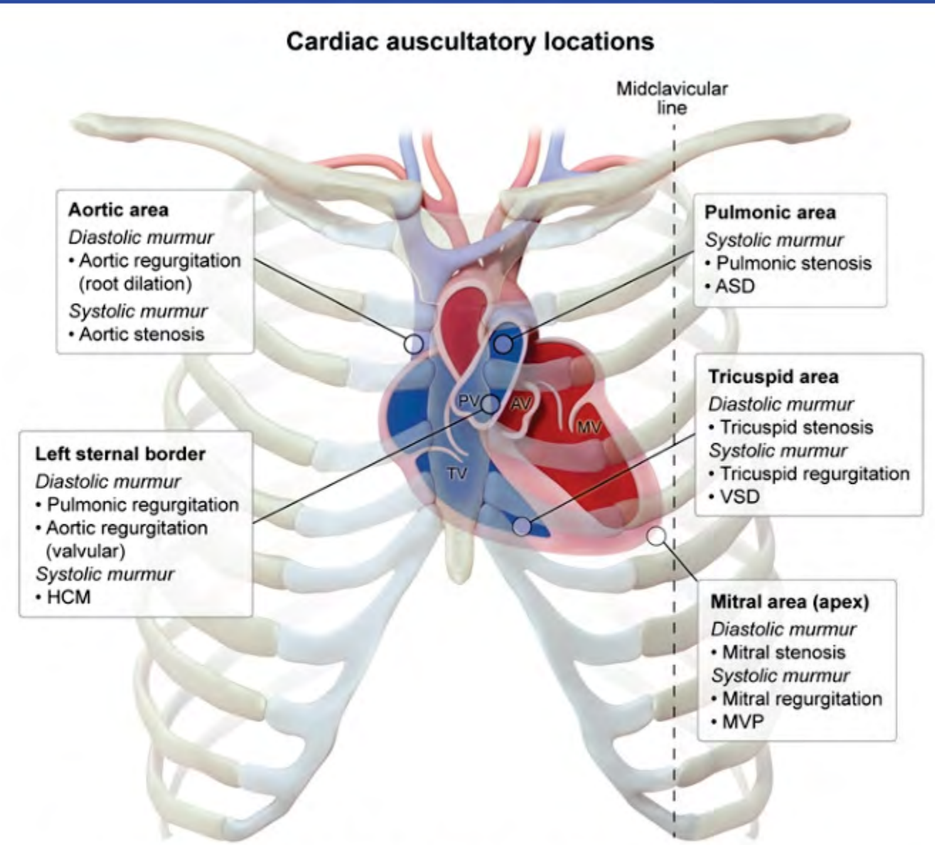

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Receptor stimulated	Change in second messenger	Primary effects
Alpha-1	↑ IP_3	• Peripheral vasoconstriction • Urethral constriction • Pupillary dilation
Alpha-2	↓ cAMP	• CNS sympatholytic • ↓ insulin release & intestinal motility
Beta-1	↑ cAMP	• ↑ Cardiac contractility & heart rate • ↑ Renin release by JG cells of kidney
Beta-2	↑ cAMP	• Peripheral vasodilation • Bronchodilation
Muscarinic-2	↓ cAMP	• ↓ Cardiac contractility & heart rate
Muscarinic-3	↑ IP_3	• Bronchoconstriction • ↑ insulin release & intestinal motility • Bladder contraction • Pupillary constriction • Peripheral vasodilation*

*Via release of nitric oxide.
cAMP = cyclic AMP; **IP_3** = inositol 1,4,5-triphosphate; **JG** = juxtaglomerular.

Phenylephrine is a selective alpha-1 adrenergic receptor agonist that causes vasoconstriction and increased blood pressure by **increasing inositol trisphosphate** (IP_3) levels in **vascular smooth muscle cells**. The abrupt increase in blood pressure triggers a **baroreceptor reflex**, resulting in increased parasympathetic and decreased sympathetic outflow to the heart and vasculature.

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Phenylephrine is a selective alpha-1 adrenergic receptor agonist that causes vasoconstriction and increased blood pressure by **increasing inositol trisphosphate** (IP_3) levels in **vascular smooth muscle cells**. The abrupt increase in blood pressure triggers a **baroreceptor reflex**, resulting in increased parasympathetic and decreased sympathetic outflow to the heart and vasculature.

The baroreceptor reflex–driven changes in the **heart** are mediated by **beta-1 adrenergic receptors** and **muscarinic-2 receptors**. Both reduced sympathetic stimulation of beta-1 receptors and increased parasympathetic stimulation of muscarinic-2 receptors trigger a **decrease in cyclic AMP** (cAMP) levels, which leads to a decrease in heart rate and contractility to reduce cardiac output and lessen the increase in blood pressure. The reduction in heart rate is driven in part by **decreased inward calcium current** during phases 4 and 0 of the sinoatrial node action potential.

The above baroreceptor response also affects vascular smooth muscle, which is mostly under sympathetic control. Although this response causes reduced sympathetic stimulation of vascular alpha-1 receptors, the infusion of phenylephrine still maintains IP_3 activity above baseline, resulting in a persistent vasoconstrictive effect.

In contrast, an abrupt decrease in blood pressure triggers the opposite regulatory response via the baroreceptor reflex.

Educational objective:
Phenylephrine increases blood pressure via an alpha-1 receptor-mediated increase in inositol triphosphate in vascular smooth muscle cells. The abrupt increase in blood pressure triggers a reflexive increase in parasympathetic activity while decreasing sympathetic outflow. This leads to decreased cyclic AMP activity in the heart (mediated by beta-1 adrenergic and muscarinic-2 receptors), which decreases heart rate and myocardial contractility. The reduction in heart rate is driven in part by decreased inward calcium current in sinoatrial cells.

Physiology	Cardiovascular System	Sympathomimetic agents
Subject	System	Topic

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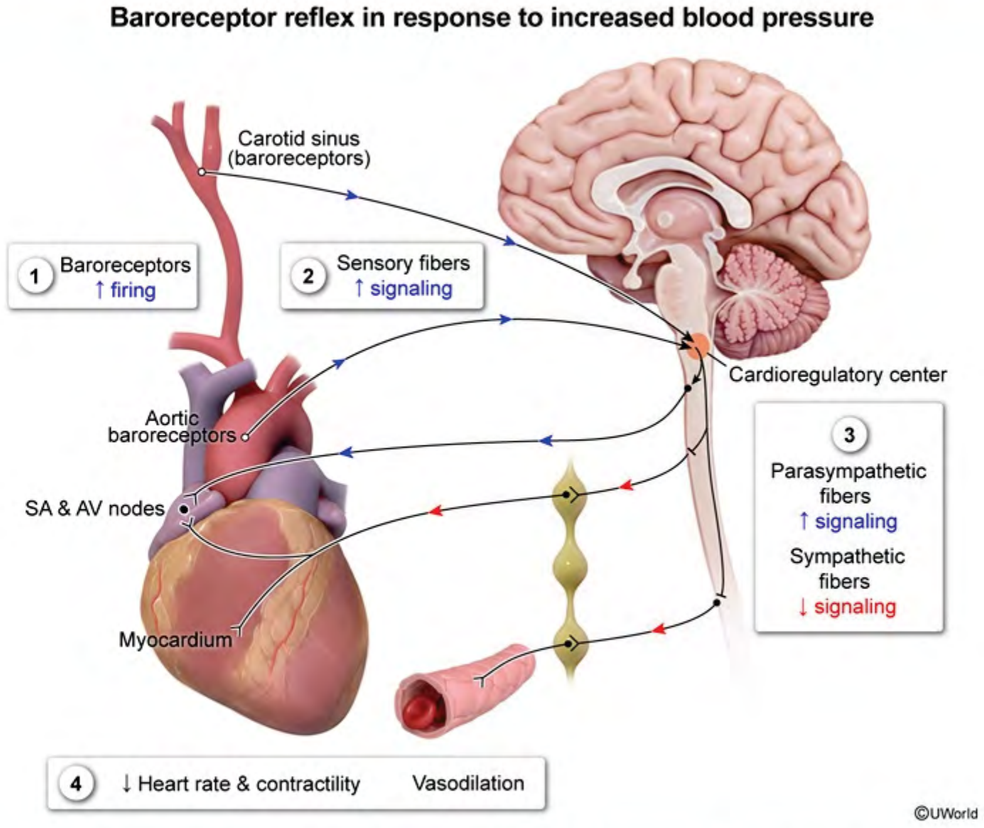

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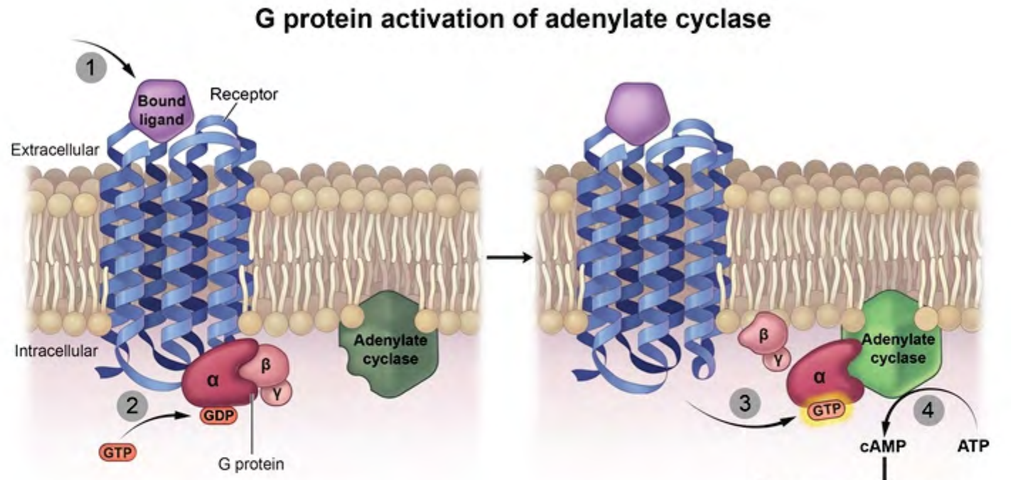

cAMP = cyclic AMP; **GDP** = guanosine 5'-diphosphate; **GTP** = guanosine 5'-triphosphate.
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Physiology | Subject
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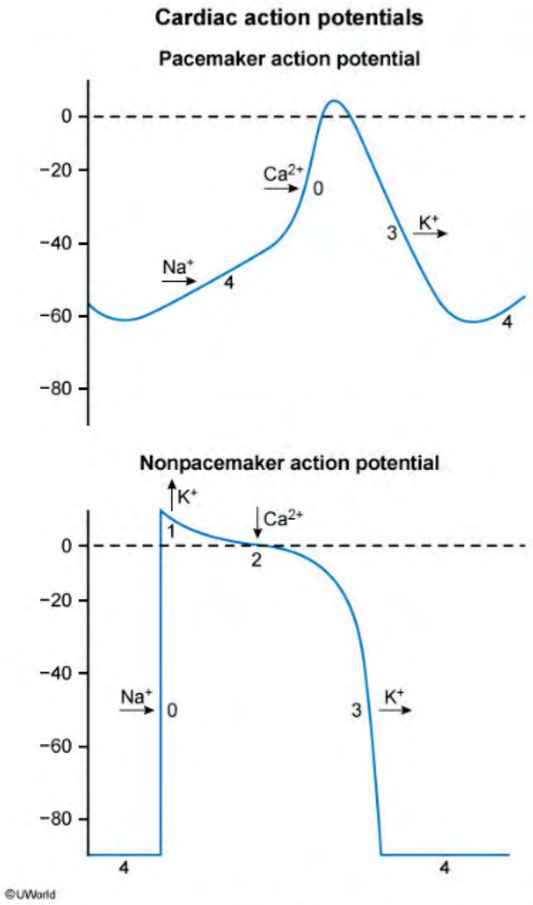

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Phenylephrine is a selective alpha-1 adrenergic receptor agonist that causes vasoconstriction and increased blood

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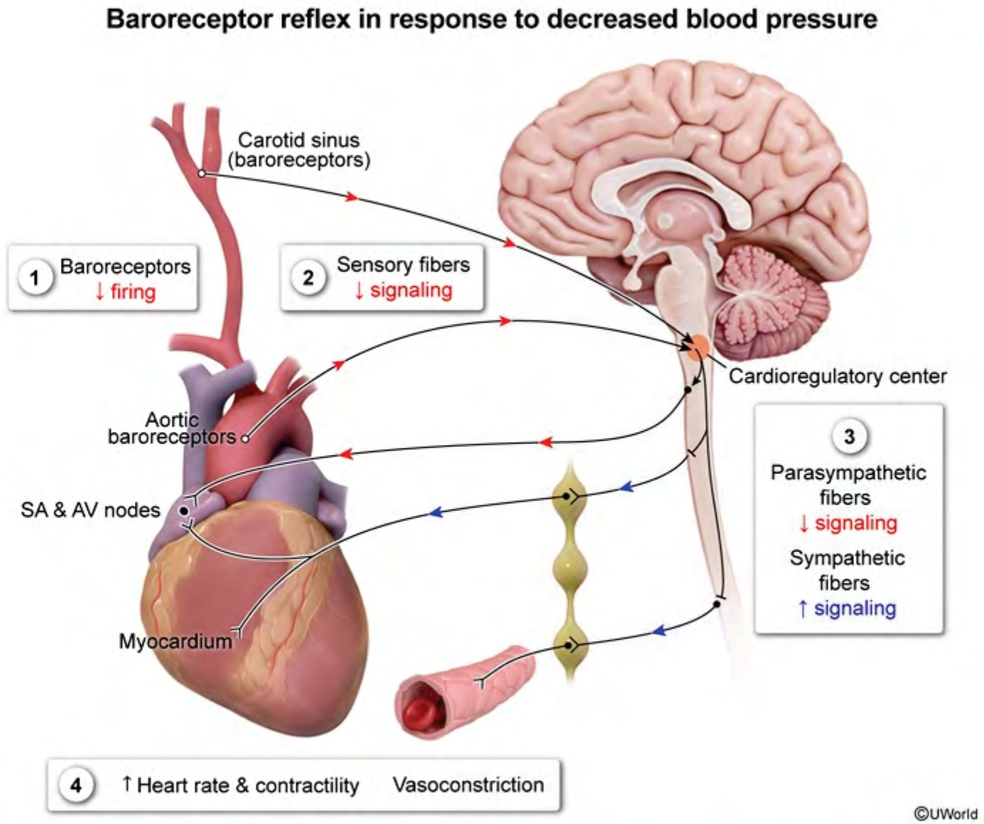

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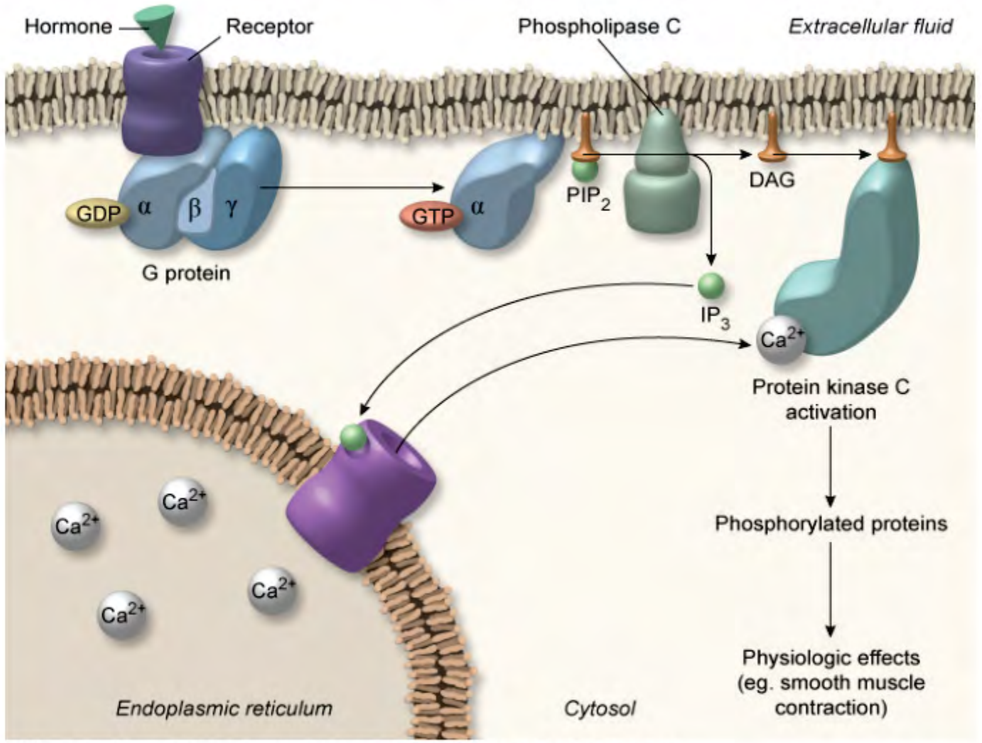
Phosphatidylinositol 2nd messenger system



DAG = diacylglycerol; **GDP** = guanosine 5′-diphosphate; **GTP** = guanosine 5′-triphosphate; **IP_3** = inositol 1,4,5-trisphosphate; **PIP_2** = phosphatidylinositol 4,5-bisphosphate.

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A 50-year-old man has recurrent episodes of paroxysmal atrial fibrillation accompanied by uncomfortable palpitations and chest pressure. Echocardiogram reveals normal biventricular function and no significant valvular disease. Coronary angiography reveals no obstructive coronary artery disease. The patient is started on medication to reduce his symptoms. Two weeks later, he is seen in the emergency department for lightheadedness, weakness, and presyncope. ECG reveals sinus bradycardia at a rate of 53/min with QTc prolongation (508 msec). Telemetry monitoring reveals a short episode of self-resolved torsades de pointes. Which of the following medications was most likely used to treat this patient's palpitations?

- A. Diltiazem
- B. Metoprolol
- C. Mexiletine
- D. Ranolazine
- E. Sotalol

Submit

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A 50-year-old man has recurrent episodes of paroxysmal atrial fibrillation accompanied by uncomfortable palpitations and chest pressure. Echocardiogram reveals normal biventricular function and no significant valvular disease. Coronary angiography reveals no obstructive coronary artery disease. The patient is started on medication to reduce his symptoms. Two weeks later, he is seen in the emergency department for lightheadedness, weakness, and presyncope. ECG reveals sinus bradycardia at a rate of 53/min with QTc prolongation (508 msec). Telemetry monitoring reveals a short episode of self-resolved torsades de pointes. Which of the following medications was most likely used to treat this patient's palpitations?

- A. Diltiazem (12%)
- ✗ B. Metoprolol (4%)
- C. Mexiletine (7%)
- D. Ranolazine (3%)
- ✓ E. Sotalol (71%)

Incorrect
Correct answer
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71%
Answered correctly

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Time Spent

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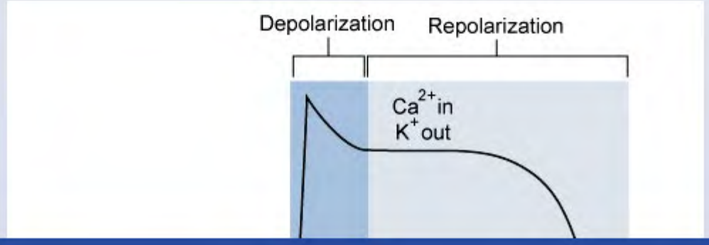
Explanation



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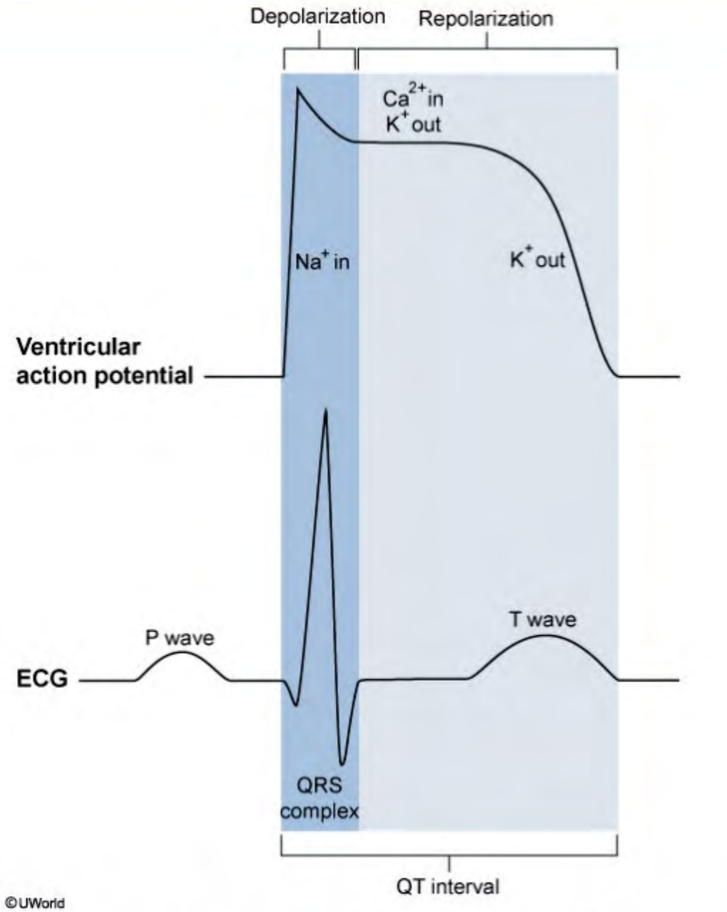

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Paroxysmal atrial fibrillation is usually treated with rate or rhythm control strategies. Rate control uses AV nodal-blocking drugs (beta blockers, calcium channel blockers) to prevent rapid ventricular response, whereas **rhythm control** attempts to maintain sinus rhythm with antiarrhythmic drugs (eg, sotalol, flecainide, amiodarone).

The QT interval represents both ventricular depolarization and repolarization (total duration of ventricular action potential). However, ventricular depolarization normally contributes only a small amount of time to the interval, and in practice, the QT interval is primarily used as a measure of ventricular repolarization. **Class III antiarrhythmics** (eg, sotalol, amiodarone, dofetilide) predominantly **block potassium channels** and inhibit outward repolarizing currents during phase 3 of the cardiomyocyte action potential. This leads to increased action potential duration and **QT interval prolongation**, which predisposes to the development of polymorphic ventricular tachycardia (**torsades de pointes** [TdP]). Amiodarone has a lesser proarrhythmic effect than other class III drugs; therefore, TdP is less frequently observed with amiodarone than with sotalol.

(Choice A) Diltiazem is a class IV antiarrhythmic drug (calcium channel blocker) that inhibits L-type calcium channels during phase 2 (cardiomyocyte action potential) and phase 0 (nodal action potential) depolarization. It slows the sinus rate, prolongs conduction through the AV node, and depresses myocardial contractility (negative inotropic effect) but does not prolong the QT interval.

(Choice B) Metoprolol is a class II antiarrhythmic drug that is a selective beta-1 receptor antagonist. It acts as a negative inotropic and chronotropic agent by decreasing myocardial contractility and heart rate. Unlike sotalol, beta blockers do not have any potassium channel-blocking properties and do not prolong the QT interval.

(Choice C) Mexiletine is a class IB antiarrhythmic drug that blocks sodium channels, inhibiting the initial depolarization phase (phase 0) of the cardiomyocyte action potential. It is occasionally used to maintain sinus rhythm in patients with paroxysmal symptomatic atrial fibrillation, but it does not prolong the QT interval.

(Choice D) Ranolazine is an antianginal agent and exerts its effect by inhibiting the late phase of inward sodium channels in ischemic cardiac myocytes. It also blocks potassium channels and causes a dose-dependent increase in QT interval; however, TdP has not been reported in ranolazine use.

Educational objective:

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(Choice A) Diltiazem is a class IV antiarrhythmic drug (calcium channel blocker) that inhibits L-type calcium channels during phase 2 (cardiomyocyte action potential) and phase 0 (nodal action potential) depolarization. It slows the sinus rate, prolongs conduction through the AV node, and depresses myocardial contractility (negative inotropic effect) but does not prolong the QT interval.

(Choice B) Metoprolol is a class II antiarrhythmic drug that is a selective beta-1 receptor antagonist. It acts as a negative inotropic and chronotropic agent by decreasing myocardial contractility and heart rate. Unlike sotalol, beta blockers do not have any potassium channel-blocking properties and do not prolong the QT interval.

(Choice C) Mexiletine is a class IB antiarrhythmic drug that blocks sodium channels, inhibiting the initial depolarization phase (phase 0) of the cardiomyocyte action potential. It is occasionally used to maintain sinus rhythm in patients with paroxysmal symptomatic atrial fibrillation, but it does not prolong the QT interval.

(Choice D) Ranolazine is an antianginal agent and exerts its effect by inhibiting the late phase of inward sodium channels in ischemic cardiac myocytes. It also blocks potassium channels and causes a dose-dependent increase in QT interval; however, TdP has not been reported in ranolazine use.

Educational objective:
Sotalol has both beta adrenergic-blocking and class III antiarrhythmic (K+ channel-blocking) properties and is occasionally used in treatment of atrial fibrillation. Major side effects of sotalol include bradycardia, proarrhythmia, and most commonly torsades de pointes due to QT interval prolongation.

References

- Drug-induced arrhythmia: pharmacogenomic prescribing?

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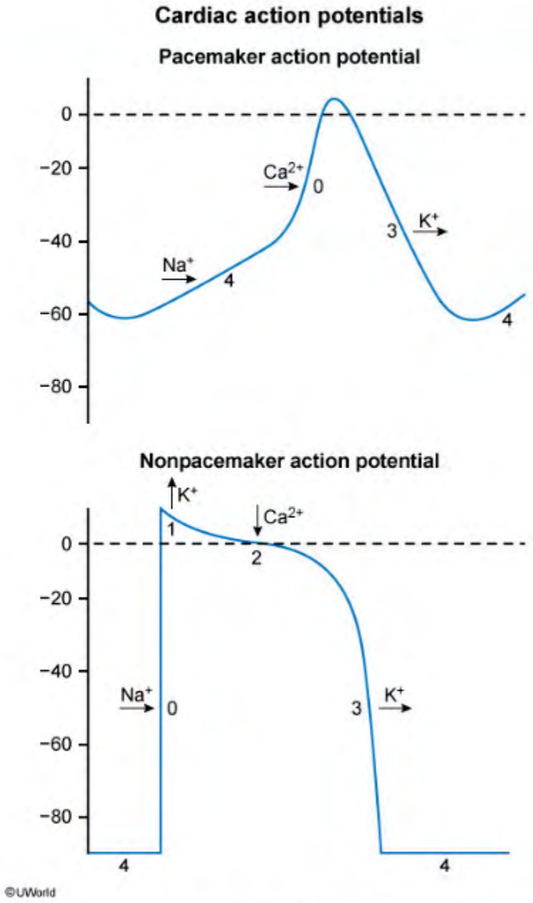

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 34-year-old Russian immigrant comes to the office with a 2-month history of exertional dyspnea and progressive lower extremity swelling. He also reports abdominal distension and decreased appetite. The patient has gained 4.5 kg (9.9 lb) over the last 2 months. Medical history is significant for hypertension and recurrent lung infections requiring prolonged antibiotic therapy. The patient has no history of coronary artery disease. He has smoked a pack of cigarettes daily for the past 15 years. The patient undergoes noninvasive cardiac testing, followed by cardiac catheterization. A jugular venous pressure tracing is shown in the image below:

Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

- A. Atrial contraction
- B. Carotid pulse upstroke
- C. Isovolumetric ventricular contraction
- D. Tricuspid valve closure
- E. Tricuspid valve opening

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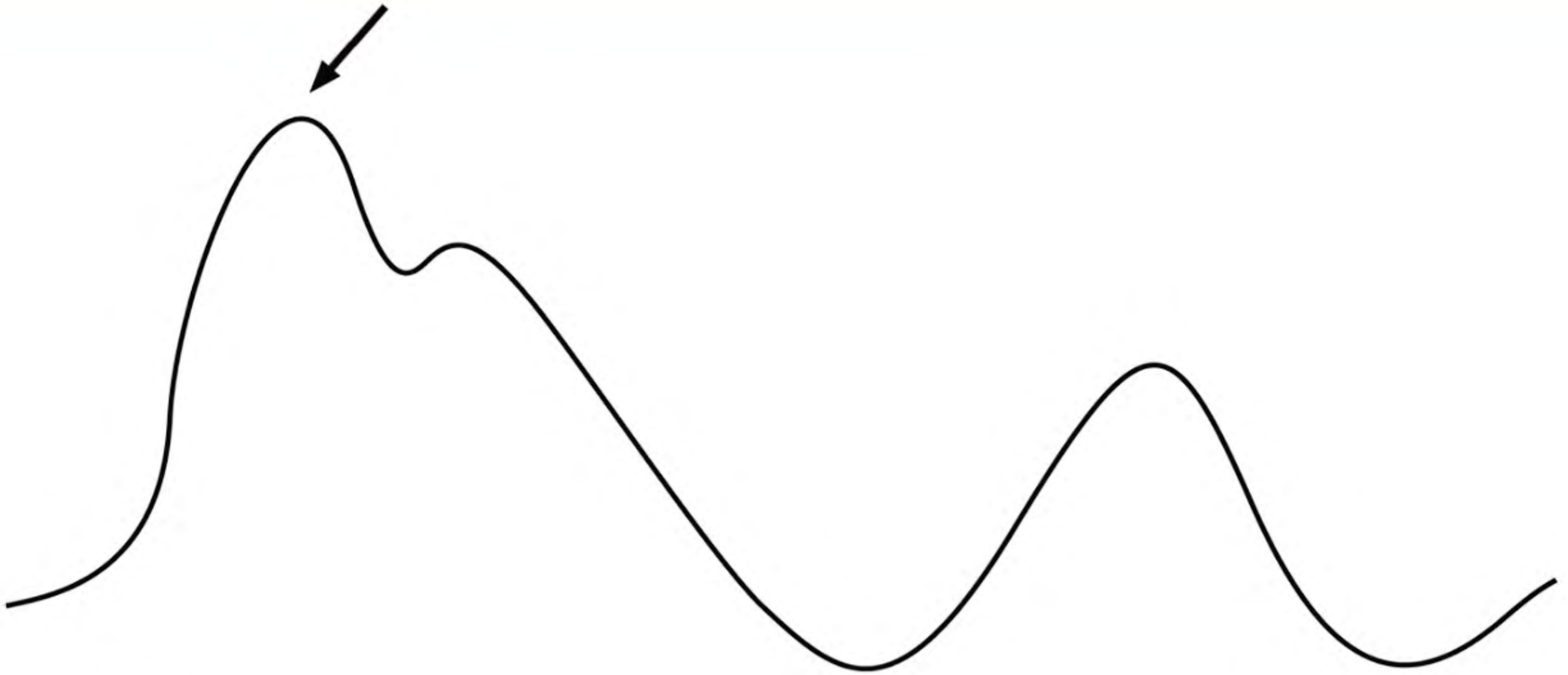

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Item 1
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B. Carotid pulse upstroke

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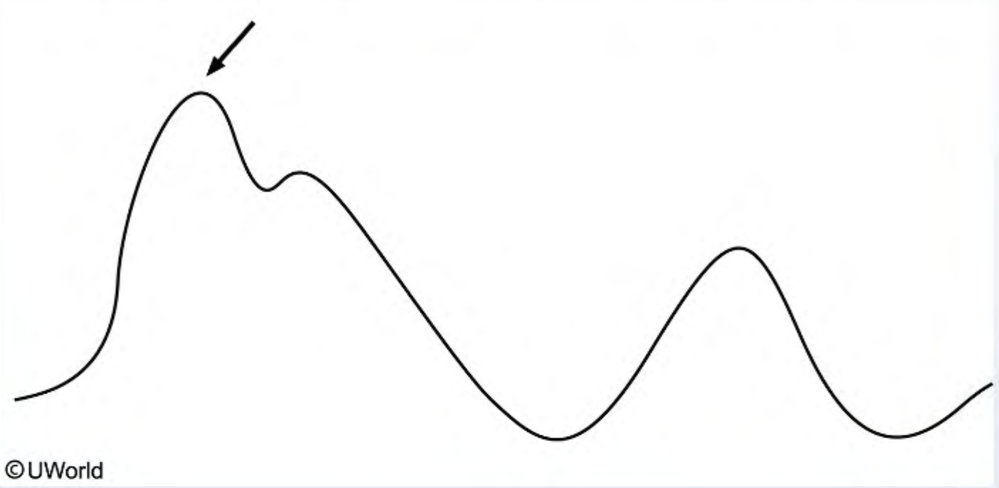

Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

- A. Atrial contraction
- B. Carotid pulse upstroke
- C. Isovolumetric ventricular contraction
- D. Tricuspid valve closure
- E. Tricuspid valve opening

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Item 2 of 2

The patient is admitted to the hospital for further work-up and treatment. After initial evaluation, CT scan of the chest is obtained and is shown in the image below:

Which of the following is the most likely diagnosis?

- A. Aortic dissection
- B. Constrictive pericarditis

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Item 2 of 2

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B. Constrictive pericarditis

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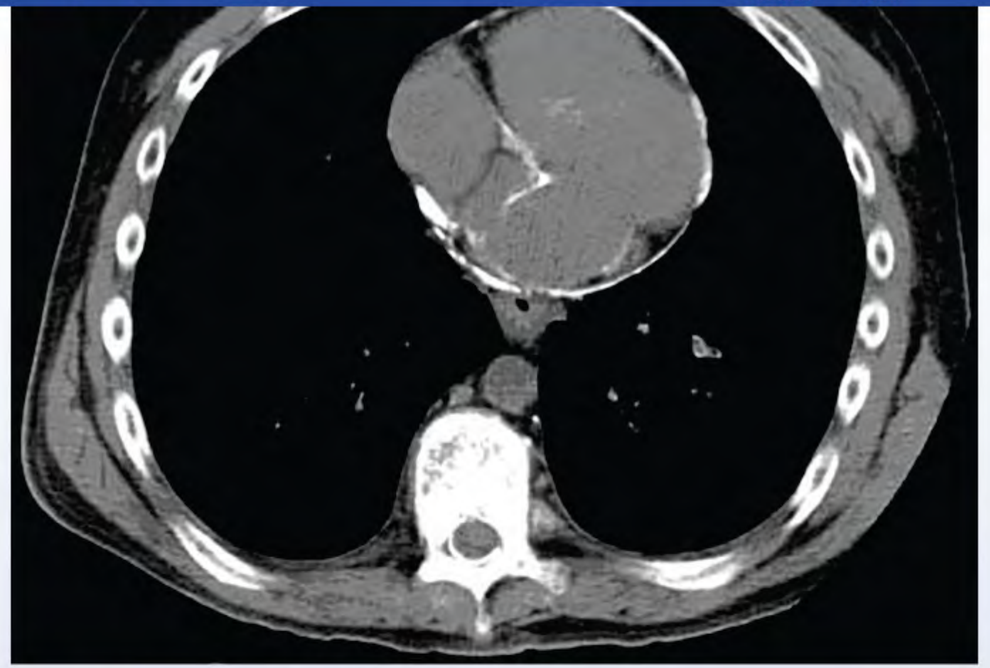

Which of the following is the most likely diagnosis?

- A. Aortic dissection
- B. Constrictive pericarditis
- C. Hypertrophic cardiomyopathy
- D. Ischemic heart disease
- E. Viral myocarditis

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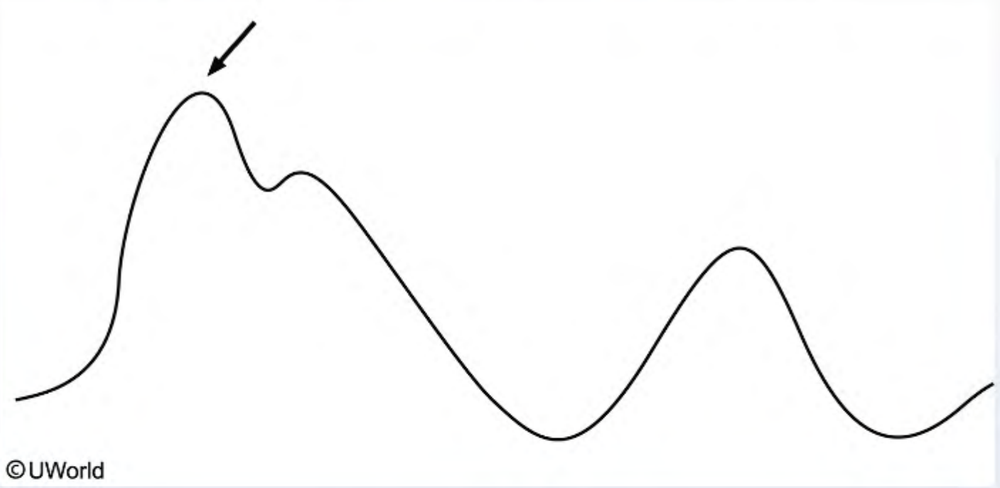
requiring prolonged antibiotic therapy. The patient has no history of coronary artery disease. He has smoked a pack of cigarettes daily for the past 15 years. The patient undergoes noninvasive cardiac testing, followed by cardiac catheterization. A jugular venous pressure tracing is shown in the image below:



Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

- ✓ A. Atrial contraction (60%)
- B. Carotid pulse upstroke (7%)
- C. Isovolumetric ventricular contraction (7%)
- D. Tricuspid valve closure (11%)
- E. Tricuspid valve opening (12%)

Correct

60% Answered correctly

25 secs Time Spent

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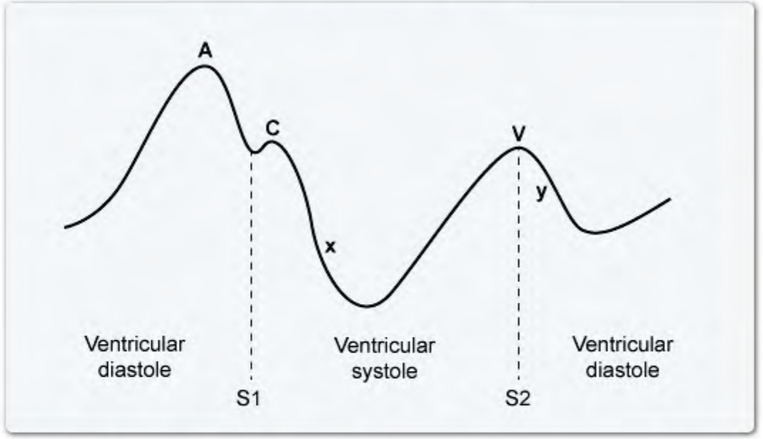
Normal JVP waveform



JVP = jugular venous pulsation.
A = atrial contraction; **C** = ventricular contraction; **x** = atrial relaxation;
V = maximal atrial filling; **y** = atrial emptying.

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(Choice C) against a closed tricuspid valve, which bulges into the right atrium. There is an ensuing decrease in

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JVP = jugular venous pulsation.
A = atrial contraction; **C** = ventricular contraction; **x** = atrial relaxation;
V = maximal atrial filling; **y** = atrial emptying.

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The **jugular venous pulsation (JVP) waveform** is representative of **pressure changes in the right atrium** throughout the cardiac cycle; the normal waveform consists of 3 peaks (A, C, and V) and 2 descents (x and y).

The **A wave** is caused by **atrial contraction** at the end of right ventricular diastole, which occurs just before tricuspid valve closure (indicated by S1) **(Choice D)**. The C wave then occurs due to right ventricular contraction **(Choice C)** against a closed tricuspid valve, which bulges into the right atrium. There is an ensuing decrease in pressure (x descent) as the emptied right atrium relaxes, followed by an uptrend as the right atrium refills with blood from the vena cava. This uptrend culminates in the V wave at the peak of right atrial filling. The pulmonic valve closes (indicated by S2) and the tricuspid valve opens **(Choice E)** to mark the beginning of passive right atrial emptying into the right ventricle (y descent).

The A wave is absent in patients with atrial fibrillation because no organized atrial contraction takes place.

(Choice B) Pressure changes in the systemic arterial circulation (eg, carotid pulse upstroke) are not represented on the JVP waveform. However, because both the right ventricle and the left ventricle contract simultaneously, the carotid pulse upstroke should occur right around the time of the C wave on the JVP waveform.

Educational objective:
On the jugular venous pulsation waveform, the A wave is generated by atrial contraction at the end of right ventricular diastole just before tricuspid valve closure.

References

- Physiology, jugular venous pulsation.

Pathophysiology	Cardiovascular System	Hemodynamic monitoring
Subject	System	Topic

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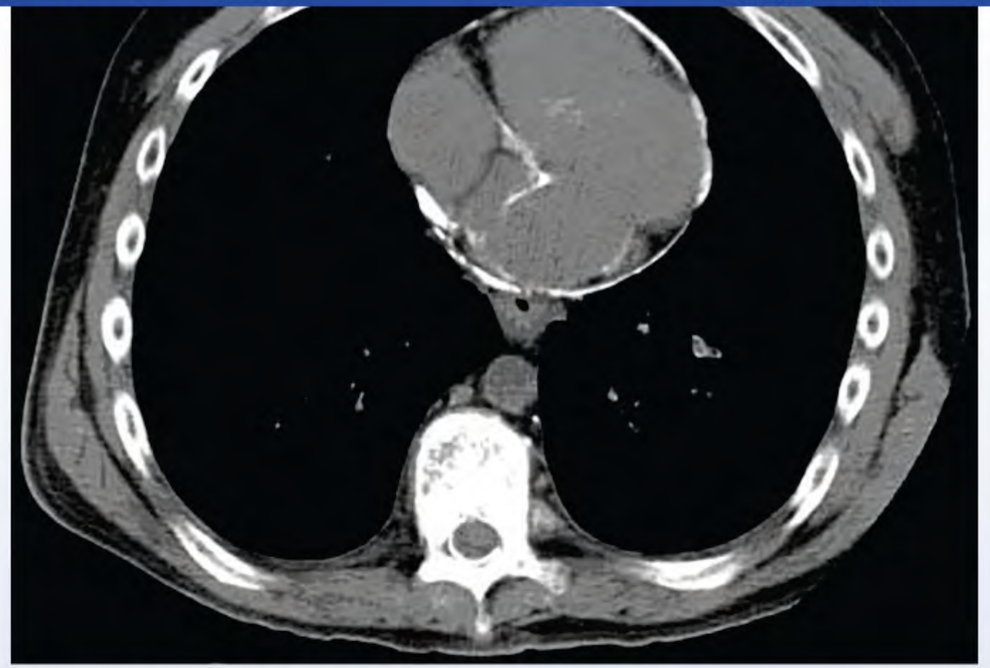

Which of the following is the most likely diagnosis?

- ✗ A. Aortic dissection (6%)
- ✓ B. Constrictive pericarditis (62%)
- C. Hypertrophic cardiomyopathy (22%)
- D. Ischemic heart disease (2%)
- E. Viral myocarditis (6%)

Incorrect
Correct answer
B

62%
Answered correctly

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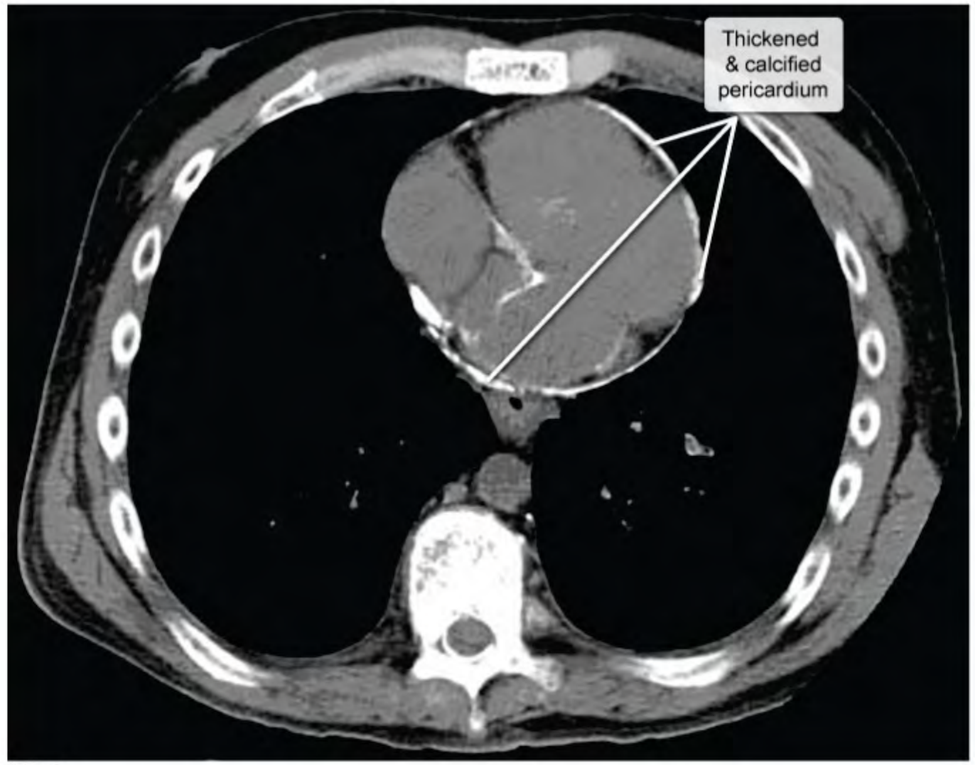
Constrictive pericarditis



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pericarditis. Although the pericardium is normally only 1-2 mm thick, its thickness can increase to up to 20 mm in patients with constrictive pericarditis. The symptomatic presentation **mimics** that of **right-sided heart failure**, with

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This patient's CT scan shows thickening and calcification of the pericardium, which are suggestive of **constrictive pericarditis**. Although the pericardium is normally only 1-2 mm thick, its thickness can increase to up to 20 mm in patients with constrictive pericarditis. The symptomatic presentation **mimics** that of **right-sided heart failure**, with patients experiencing slowly progressive dyspnea, lower extremity edema, ascites, and weight gain.

A **rapid y descent** (Friedreich sign) on the jugular venous pulsation waveform (as seen in this patient) is a characteristic finding of constrictive pericarditis; the constrictive pressure on the right atrium accentuates early filling of the right ventricle while the stiff pericardial wall restricts late diastolic filling.

Possible causes of constrictive pericarditis include radiation therapy to the chest, cardiac surgery, and **tuberculosis**, which may be the cause in this patient, who emigrated from an endemic region.

(Choice A) Aortic dissection causes sudden chest pain that radiates to the back. Chest radiographs often reveal widening of the mediastinum, and a double aortic lumen would be seen on CT scan of the chest.

(Choice C) Hypertrophic cardiomyopathy also presents with dyspnea and changes in the jugular venous pressure, specifically a prominent A wave (due to decreased right ventricular compliance). CT scan may reveal thickening of the interventricular septum but not thickening or calcification of the pericardium.

(Choice D) Ischemic heart disease classically causes pressure-like substernal chest pain that radiates to the left shoulder; calcifications in the coronary arteries and aorta are often seen on CT scan.

(Choice E) Patients with viral myocarditis have a variable clinical presentation ranging from asymptomatic to severe chest pain and cardiogenic shock. Pericardial thickening is not a feature of this condition.

Educational objective:
The symptomatic presentation of constrictive pericarditis mimics that of right-sided heart failure, with patients experiencing slowly progressive dyspnea, lower extremity edema, ascites, and weight gain. Calcification and thickening of the pericardium are typical features of constrictive pericarditis on CT scan.

Pathophysiology Cardiovascular System Constrictive pericarditis

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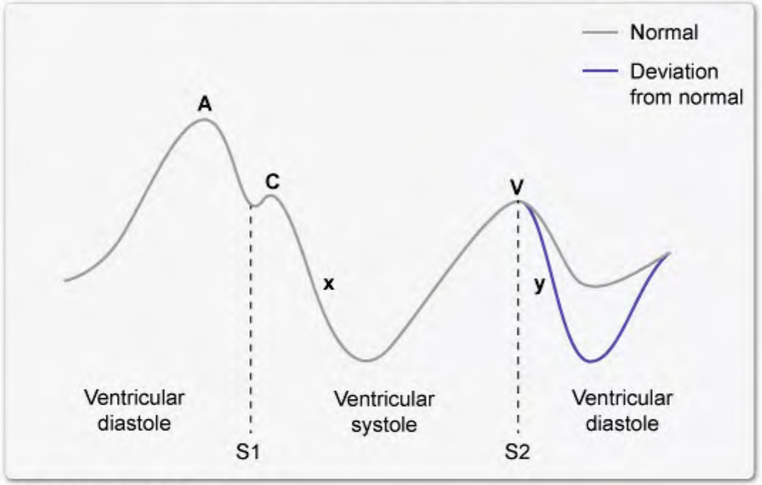
Constrictive pericarditis JVP waveform



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Pathophysiology | Cardiovascular System | Constrictive pericarditis

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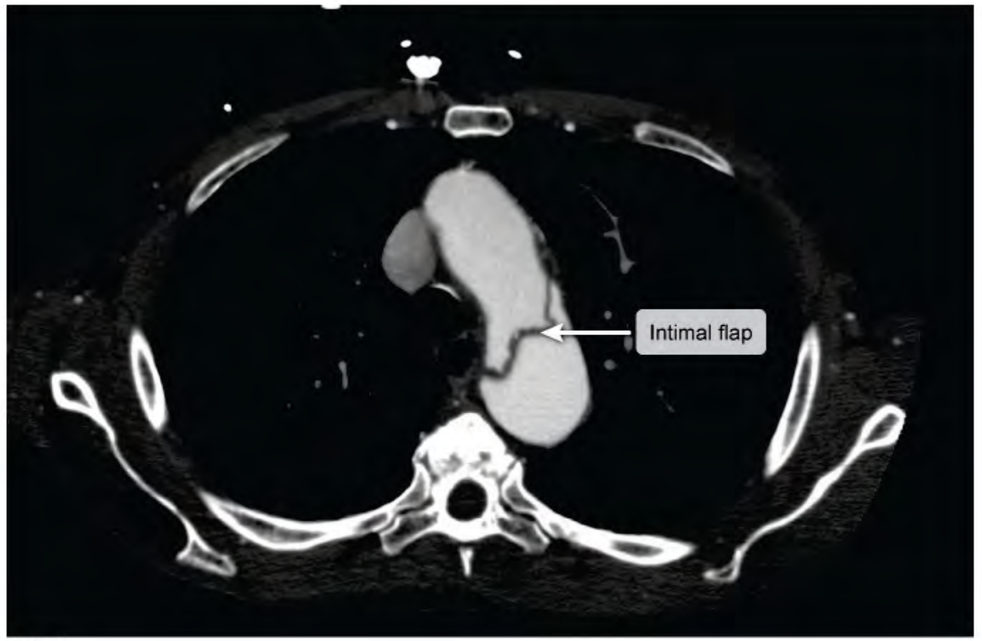
Aortic dissection



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Pathophysiology | Cardiovascular System | Constrictive pericarditis

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A 64-year-old man dies suddenly while playing tennis. In the preceding months, he experienced fatigue and some exertional dyspnea. Autopsy is performed. The heart examination shows left ventricular septal thickness of 1.6 cm (normal: <1.1), posterior wall thickness of 1.6 cm (normal: <1.1), and an internal left ventricular diameter of 3.2 cm (normal: 3.5-5.9). No focal myocardial scarring is seen. Which of the following is the most likely cause of the cardiac findings seen in this individual?

- A. Aortic stenosis
- B. Dilated cardiomyopathy
- C. Mitral regurgitation
- D. Obstructive coronary artery disease
- E. Rheumatic mitral stenosis

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- ✓ A. Aortic stenosis (87%)
- ✗ B. Dilated cardiomyopathy (4%)
- C. Mitral regurgitation (3%)
- D. Obstructive coronary artery disease (3%)
- E. Rheumatic mitral stenosis (0%)

Incorrect
Correct answer
A

87%
Answered correctly

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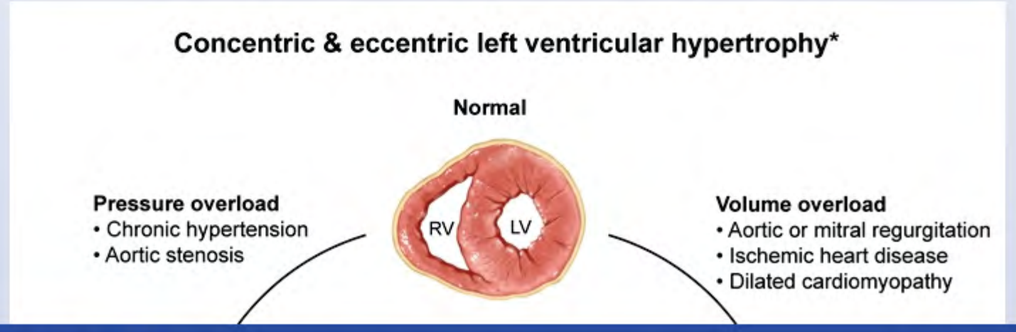

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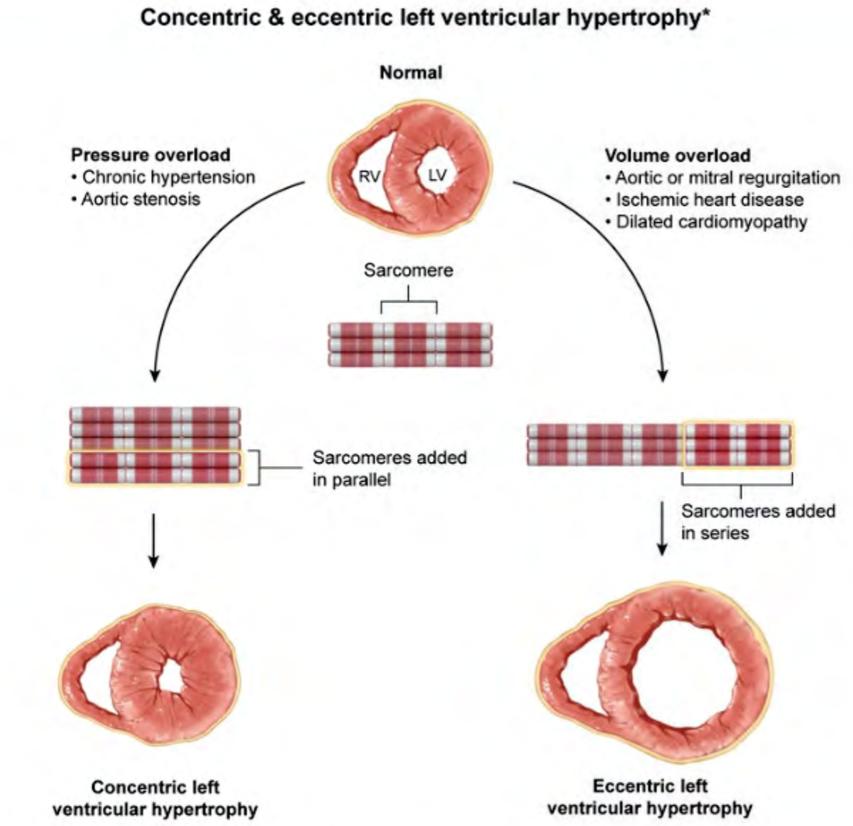

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The right ventricle undergoes similar adaptation when exposed to chronic pressure or volume overload.
LV = left ventricle; **RV** = right ventricle.

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This individual's autopsy reveals **thickened left ventricular walls** and a small left ventricular cavity consistent with **concentric hypertrophy**. Concentric hypertrophy results from the addition of myocardial contractile fibers in parallel in response to chronic **pressure overload**. The adaptation allows for increased contractile force but causes impaired diastolic filling and can lead to heart failure with preserved ejection fraction (diastolic failure). Common causes of concentric hypertrophy include prolonged systemic hypertension and **aortic stenosis**.

Patients with severe aortic stenosis typically experience exertional symptoms, including fatigue, shortness of breath, angina, and presyncope or syncope. **Sudden cardiac death** can result from the inability to increase cardiac output with exertion due to left ventricular outflow obstruction.

(Choice B) Dilated cardiomyopathy results from a primary insult to cardiomyocytes (eg, viral infection, chemical toxicity, inherited disease) that causes myocardial contractile dysfunction, left ventricular volume overload, and eventual eccentric hypertrophy.

(Choice C) Severe mitral regurgitation causes left ventricular volume overload and eccentric hypertrophy.

(Choice D) Obstructive coronary artery disease can lead to eccentric hypertrophy via myocardial contractile dysfunction and consequent left ventricular volume overload (ie, ischemic heart disease). The development of concentric hypertrophy is not typical.

(Choice E) Rheumatic mitral stenosis causes impaired diastolic filling of the left ventricle. It does not increase the pressure or volume load on the left ventricle and does not cause ventricular hypertrophy.

Educational objective:
Concentric left ventricular hypertrophy involves thickening of the ventricular walls and reduction in the ventricular cavity size. It occurs via the addition of myocardial contractile fibers in parallel in response to chronic pressure overload. Aortic stenosis and prolonged systemic hypertension are common causes of concentric left ventricular hypertrophy.

Pathology Cardiovascular System Aortic stenosis

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*The right ventricle undergoes similar adaptation when exposed to chronic pressure or volume overload.
LV = left ventricle; **RV** = right ventricle.

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Patients with severe aortic stenosis typically experience exertional symptoms, including fatigue, shortness of breath, angina, and presyncope or syncope. **Sudden cardiac death** can result from the inability to increase cardiac output with exertion due to left ventricular outflow obstruction.

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(Choice D) Obstructive coronary artery disease can lead to eccentric hypertrophy via myocardial contractile dysfunction and consequent left ventricular volume overload (ie, ischemic heart disease). The development of concentric hypertrophy is not typical.

(Choice E) Rheumatic mitral stenosis causes impaired diastolic filling of the left ventricle. It does not increase the pressure or volume load on the left ventricle and does not cause ventricular hypertrophy.

Educational objective:
Concentric left ventricular hypertrophy involves thickening of the ventricular walls and reduction in the ventricular cavity size. It occurs via the addition of myocardial contractile fibers in parallel in response to chronic pressure overload. Aortic stenosis and prolonged systemic hypertension are common causes of concentric left ventricular hypertrophy.

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The following vignette applies to the next **2** items.

A 52-year-old man is brought to the emergency department for evaluation of fever, chills, and malaise. Cardiopulmonary examination reveals a new holosystolic heart murmur that radiates toward the axilla. Blood cultures are obtained, and the patient undergoes transesophageal echocardiography. The ultrasound probe is placed in the midesophagus facing anteriorly, and the cardiac chambers are interrogated.

Item 1 of 2

Which of the following chambers is closest to the probe?

- A. Left atrium
- B. Left ventricle
- C. Right atrium
- D. Right ventricle

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Item 2 of 2

In the same midesophageal position, the probe is rotated so that it now faces posteriorly. Which of the following structures will be best visualized with the probe's new orientation?

- A. Descending aorta
- B. Pulmonary artery
- C. Pulmonary veins
- D. Superior vena cava
- E. Tricuspid valve

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A 52-year-old man is brought to the emergency department for evaluation of fever, chills, and malaise. Cardiopulmonary examination reveals a new holosystolic heart murmur that radiates toward the axilla. Blood cultures are obtained, and the patient undergoes transesophageal echocardiography. The ultrasound probe is placed in the midesophagus facing anteriorly, and the cardiac chambers are interrogated.

Item 1 of 2

Which of the following chambers is closest to the probe?

- ✓ A. Left atrium (79%)
- B. Left ventricle (4%)
- C. Right atrium (11%)
- D. Right ventricle (4%)

Correct

79% Answered correctly

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Explanation

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A. Left atrium (79%)

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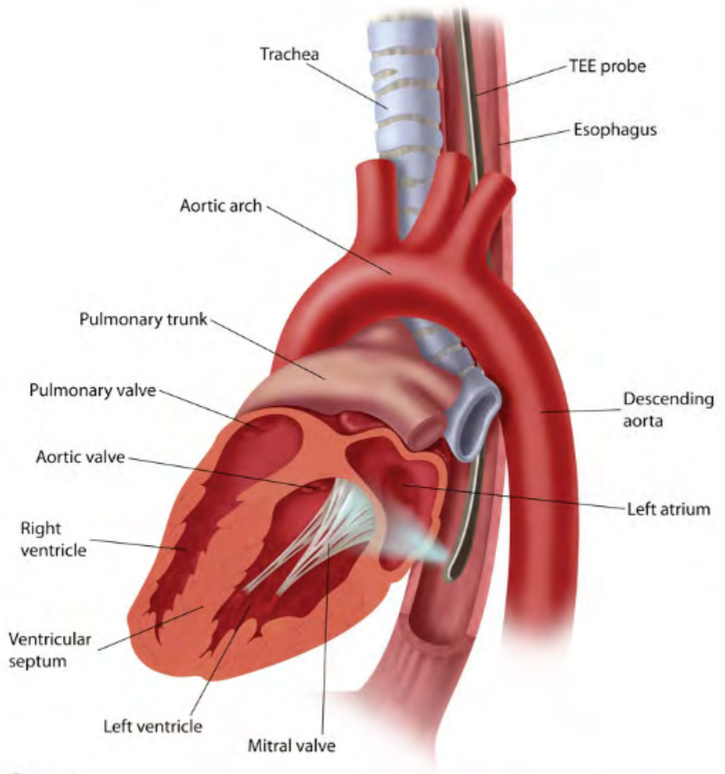
Transesophageal echocardiography (TEE)



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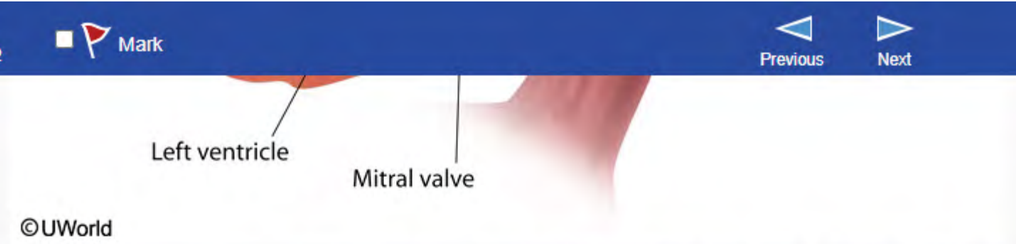

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This patient's presentation suggests acute endocarditis, with confirmation pending the results of the blood cultures and cardiac imaging. **Transesophageal echocardiography** (TEE) uses ultrasound waves generated from within the **esophagus** to produce clear images of the neighboring cardiac structures. The left atrium makes up the majority of the heart's posterior surface, with the esophagus passing immediately posterior to the heart. Therefore, the esophagus lies within closest proximity to the **left atrium**. This allows the left atrium, atrial septum, and mitral valve to be particularly well visualized on TEE.

Due to its proximity, conditions that result in left atrial enlargement (eg, mitral stenosis or regurgitation) can cause dysphagia through external compression of the esophagus.

(Choice B) The left ventricle forms the majority of the inferior (diaphragmatic) surface of the heart and the left border of the heart on frontal chest x-ray.

(Choice C) The right atrium, along with the superior vena cava, forms the right lateral cardiac border on frontal chest x-ray.

(Choice D) The right ventricle forms the anterior (sternal) surface of the heart and the majority of its inferior border on frontal chest x-ray.

Educational objective:
The left atrium forms the majority of the posterior surface of the heart and resides adjacent to the esophagus. Enlargement of the left atrium can compress the esophagus and cause dysphagia.

Anatomy	Cardiovascular System	Cardiovascular anatomy
Subject	System	Topic

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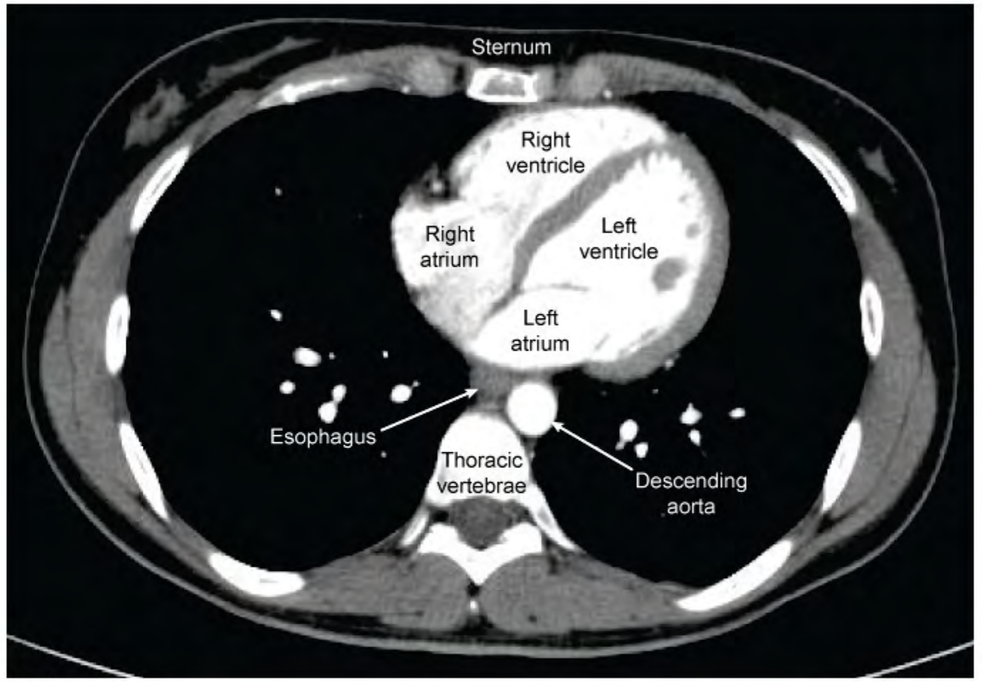
Normal chest CT



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Item 2 of 2

In the same midesophageal position, the probe is rotated so that it now faces posteriorly. Which of the following structures will be best visualized with the probe's new orientation?

- ✓ A. Descending aorta (74%)
- ✗ B. Pulmonary artery (4%)
- C. Pulmonary veins (8%)
- D. Superior vena cava (11%)
- E. Tricuspid valve (1%)

Incorrect
Correct answer
A

74%
Answered correctly

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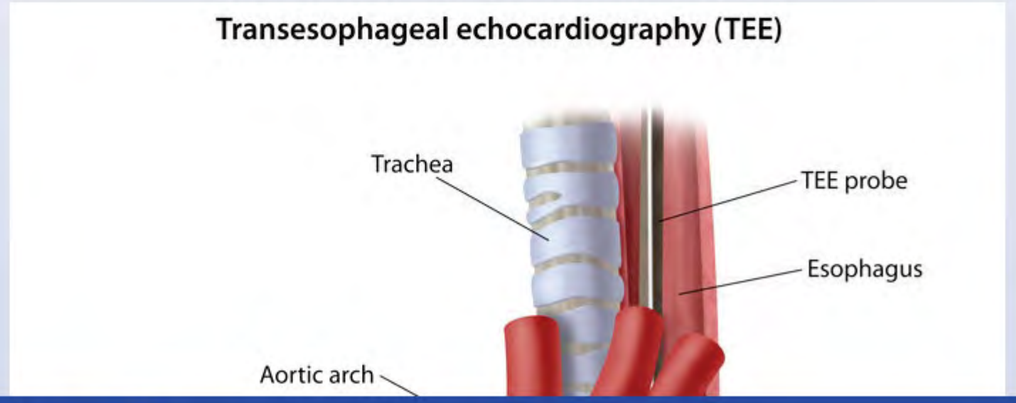
Explanation



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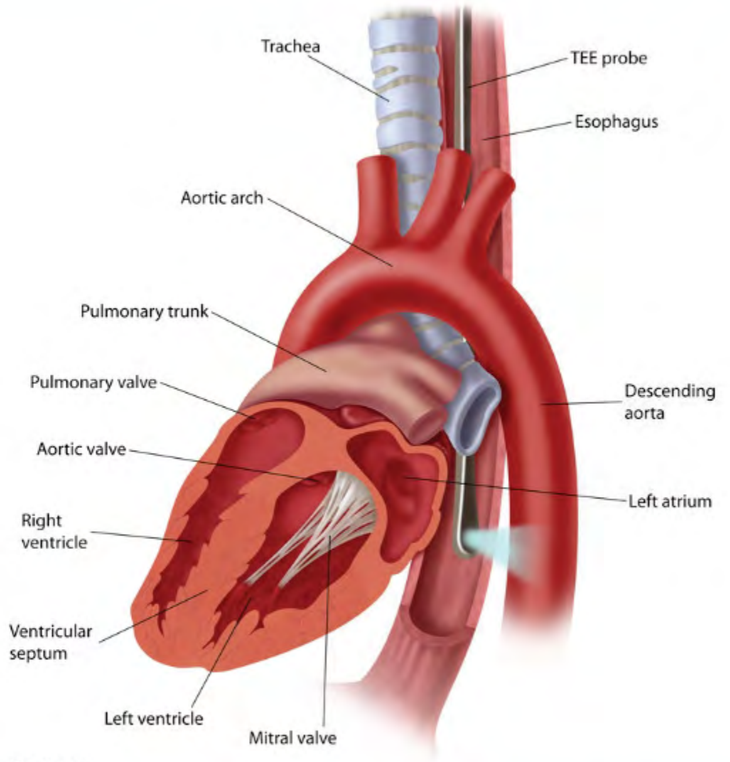
E. Tricuspid valve (1%)

Exhibit Display

Transesophageal echocardiography (TEE)



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Mitral valve

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The aorta has 4 major divisions: the ascending aorta, the aortic arch, the descending thoracic aorta, and the abdominal aorta. The ascending aorta lies posterior and to the right of the main pulmonary artery. The aortic arch travels above the right pulmonary artery and the left bronchus. The brachiocephalic, left common carotid, and left subclavian arteries (in that order) originate from its superior aspect.

The **descending thoracic aorta** travels down the anterior surface of the vertebral column, becoming the abdominal aorta as it crosses the diaphragm. As it descends, the aorta moves from the left side of the vertebral column toward the midline; at the level of the cardiac chambers, the descending aorta lies posterior to the esophagus and the left atrium. This permits clear visualization of the descending aorta during transesophageal echocardiography (TEE), allowing for the detection of abnormalities such as dissection or aneurysm.

The other answer choices describe structures located anterior to the esophagus.

(Choice B) The main pulmonary artery ascends anteriorly and to the left of the ascending aorta and is directed toward the left shoulder. After the pulmonary artery bifurcates, the right pulmonary artery travels horizontally under the aortic arch posterior to the superior vena cava, and the left pulmonary artery courses superiorly over the left main bronchus.

(Choice C) The superior and inferior pulmonary veins arise bilaterally from each lung and enter the left atrium. The proximal 2-3 cm of the pulmonary veins contain cardiac muscle within the media and function like sphincters during atrial systole.

(Choice D) The superior vena cava (SVC) is formed behind the right first costal cartilage by the merger of the right and left brachiocephalic veins. It returns blood from the head, neck, and upper extremities to the right atrium of the heart. Mediastinal neoplasms can compress the SVC and result in SVC syndrome.

(Choice E) The tricuspid valve is located between the right atrium and right ventricle and is composed of 3 valve leaflets, the annulus, supporting chordae tendineae, and the papillary muscles. It is commonly infected (endocarditis) in intravenous drug users.

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The **descending thoracic aorta** travels down the anterior surface of the vertebral column, becoming the abdominal aorta as it crosses the diaphragm. As it descends, the aorta moves from the left side of the vertebral column toward the midline; at the level of the cardiac chambers, the descending aorta lies posterior to the esophagus and the left atrium. This permits clear visualization of the descending aorta during transesophageal echocardiography (TEE), allowing for the detection of abnormalities such as dissection or aneurysm.

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Educational objective:
The descending thoracic aorta lies posterior to the esophagus and the left atrium. This position permits clear visualization of the descending aorta by transesophageal echocardiography, allowing for the detection of abnormalities such as dissection or aneurysm.

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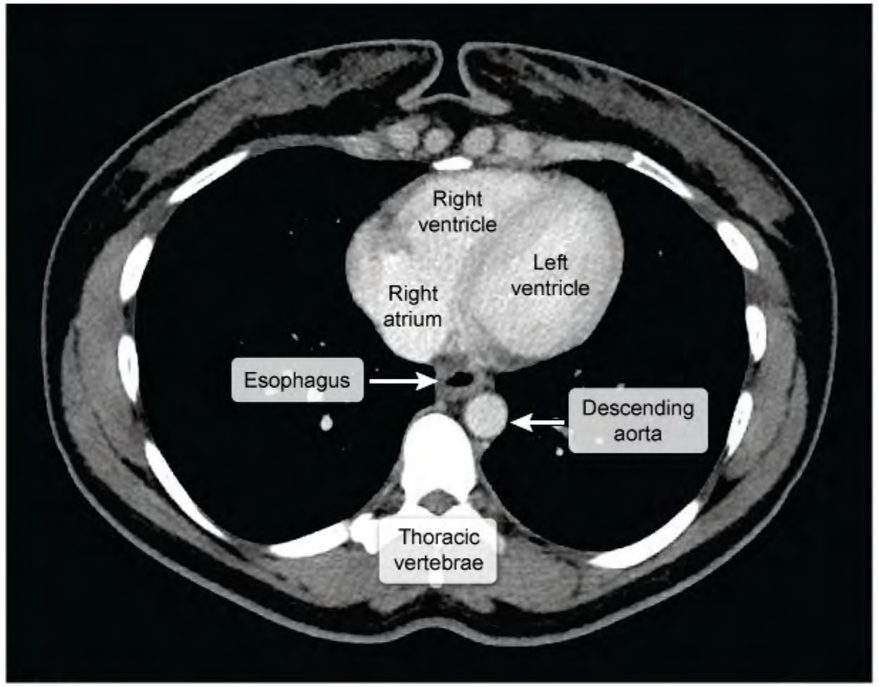
Mitral valve

Exhibit Display

Normal chest CT



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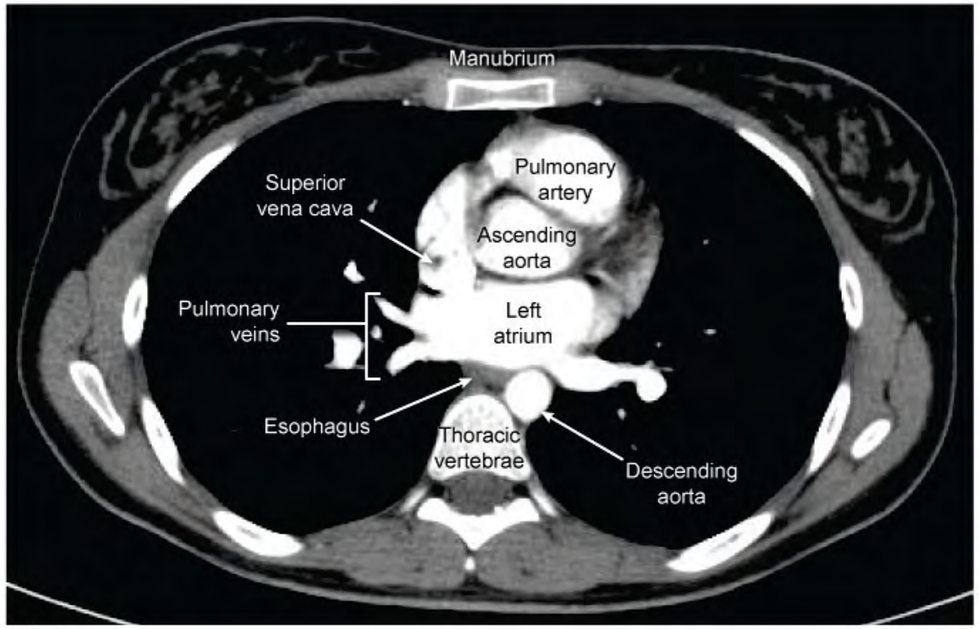
Mitral valve

Exhibit Display

Normal chest CT



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Autopsy of a 78-year-old man demonstrates decreased left ventricular cavity size and a sigmoid-shaped ventricular septum. Light microscopy shows increased collagen content within the ventricular wall. Some myocardial cells also have brownish perinuclear cytoplasmic inclusions. The changes described are most consistent with which of the following conditions?

- A. Dilated cardiomyopathy
- B. Hemolytic anemia
- C. Hypertrophic cardiomyopathy
- D. Ischemic heart disease
- E. Long-standing hypertension
- F. Normal aging

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Autopsy of a 78-year-old man demonstrates decreased left ventricular cavity size and a sigmoid-shaped ventricular septum. Light microscopy shows increased collagen content within the ventricular wall. Some myocardial cells also have brownish perinuclear cytoplasmic inclusions. The changes described are most consistent with which of the following conditions?

- A. Dilated cardiomyopathy (2%)
- ✗ B. Hemolytic anemia (1%)
- C. Hypertrophic cardiomyopathy (18%)
- D. Ischemic heart disease (11%)
- E. Long-standing hypertension (13%)
- ✓ F. Normal aging (53%)

Incorrect
Correct answer
F

53%
Answered correctly

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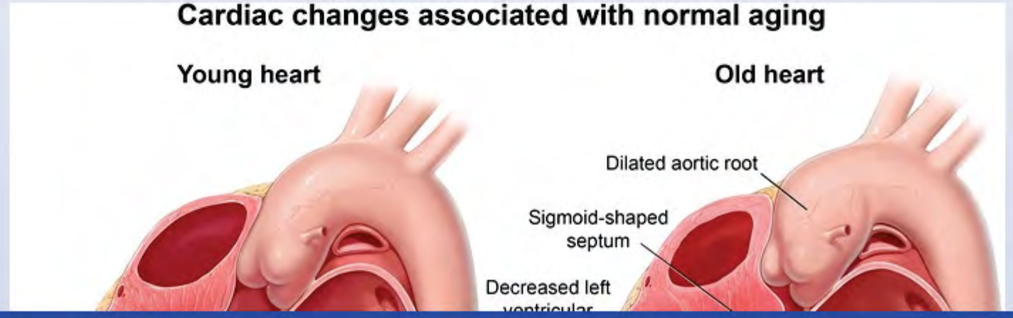

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Autopsy of a 78-year-old man demonstrates decreased left ventricular cavity size and a sigmoid-shaped ventricular septum. The patient also had the following

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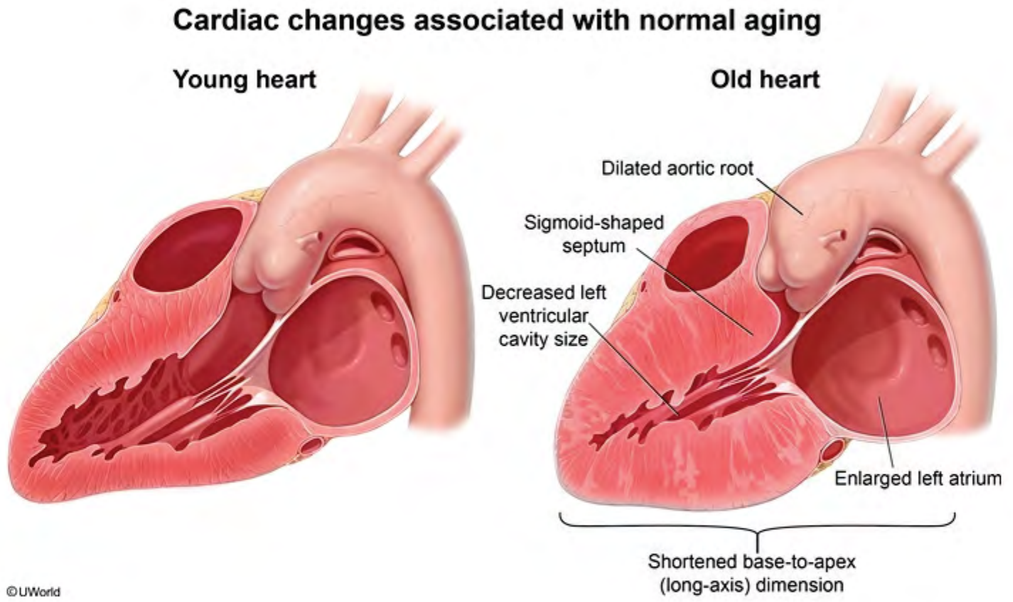

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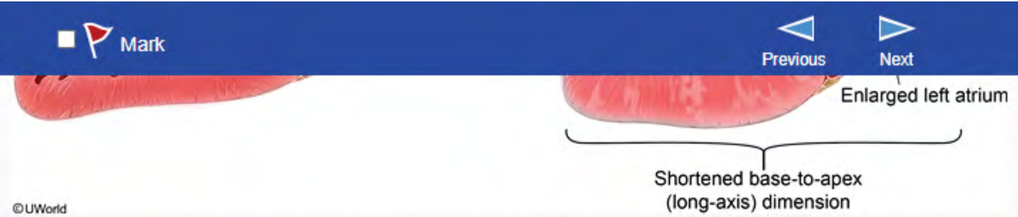

The morphologic changes described are consistent with **normal aging** of the heart. Aging is associated with **decreased left ventricular chamber size**, particularly in the apex-to-base dimension. This decrease in chamber length causes the ventricular septum to acquire a **sigmoid shape**, with the basilar portion bulging into the left ventricular outflow tract. Atrophy of the myocardium results in **increased interstitial connective tissue**, often with concomitant extracellular amyloid deposition. Within the cardiomyocytes, there is a progressive accumulation of cytoplasmic granules containing brownish **lipofuscin pigment** (the result of indigestible byproducts of subcellular membrane lipid oxidation).

(Choice A) Dilated cardiomyopathy causes an increase in the left ventricular chamber size. There is an increased risk of mural thrombi with this condition due to contractile dysfunction.

(Choice B) Chronic hemolytic anemia may result in the formation of hemosiderin granules containing excess iron inside cardiac myocytes (hemosiderosis), which can be visualized with Prussian-blue stain. Excessive deposition of iron within the myocytes usually leads to dilated or restrictive cardiomyopathy.

(Choice C) Hypertrophic cardiomyopathy (idiopathic hypertrophic subaortic stenosis) causes asymmetric septal hypertrophy with disproportionate thickening of the ventricular septum compared to the left ventricular free wall. Microscopic examination shows extreme myocyte hypertrophy, disorganized myocyte bundles, and myofiber disarray.

(Choice D) Chronic ischemic heart disease tends to result in progressive congestive heart failure and ventricular dilation. Histologically, the major findings are diffuse subendocardial vacuolization and fibrosis.

(Choice E) Long-standing hypertension results in hypertensive heart disease. Although left ventricular chamber size may be reduced in this condition, the major morphologic change is concentric left ventricular hypertrophy.

Educational objective:

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Educational objective:
Normal morphological changes in the aging heart include a decrease in left ventricular chamber apex-to-base dimension, development of a sigmoid-shaped ventricular septum, myocardial atrophy with increased collagen deposition, and accumulation of cytoplasmic lipofuscin pigment within cardiomyocytes.

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A 5-week-old boy is being evaluated for a week-long history of rapid breathing and tiring with feeds. The infant was born at home after an uneventful pregnancy. The mother declined all prenatal testing and ultrasound evaluations. His temperature is 36.7 C (98 F), blood pressure is 76/38 mm Hg, pulse is 124/min, and respirations are 66/min. The patient's cardiovascular examination is notable for a hyperdynamic precordium, a mid-diastolic rumble at the left sternal border, and a 3/6 holosystolic murmur in the apex that radiates to the left axilla. An echocardiogram shows defects in the lower part of the interatrial septum and the interventricular septum. This patient's condition is most likely associated with which of the following genetic conditions?

- A. 22q11.2 deletion
- B. Autosomal trisomy
- C. Fibrillin mutation
- D. Frataxin mutation
- E. *Hamartin* gene mutation
- F. Sex chromosome monosomy

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- A. 22q11.2 deletion (23%)
- ✓ B. Autosomal trisomy (64%)
- C. Fibrillin mutation (5%)
- D. Frataxin mutation (3%)
- E. *Hamartin* gene mutation (1%)
- F. Sex chromosome monosomy (1%)

Correct

64%
Answered correctly

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Explanation

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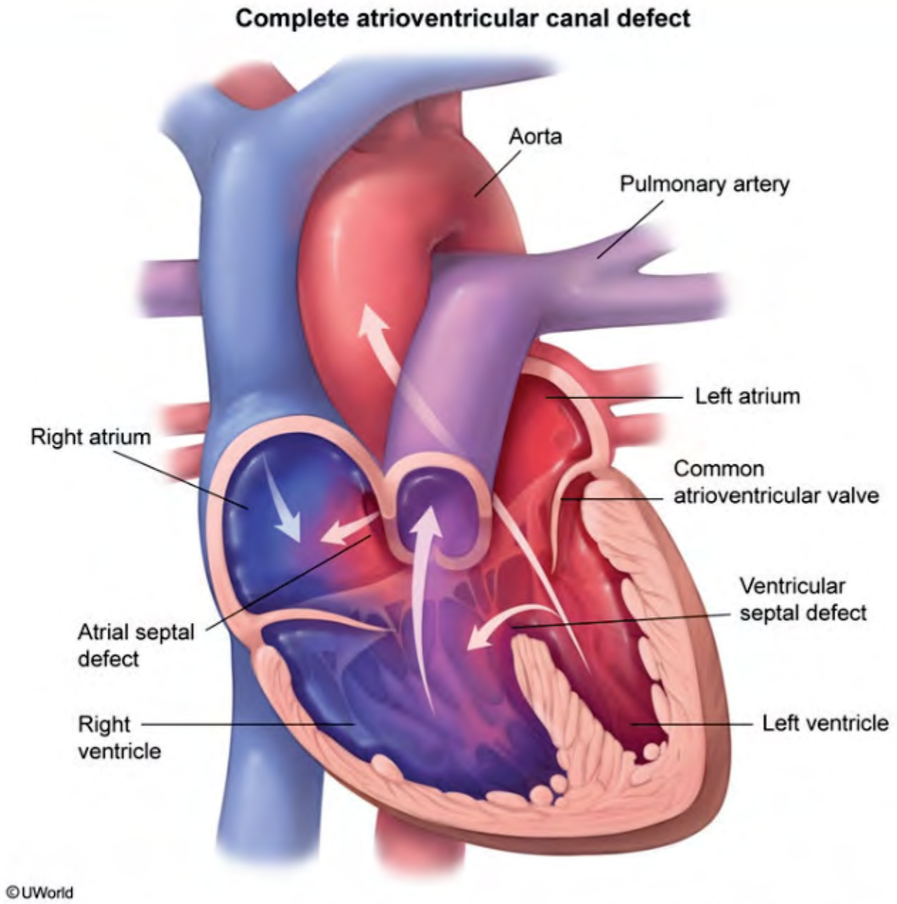

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This patient's echocardiogram suggests a **complete atrioventricular (AV)** canal defect, the most common type of cardiac defect in patients with **Down syndrome**, an autosomal trisomy. Failure of endocardial cushion fusion results in an ostium primum atrial septal defect; a ventricular septal defect; and a single AV valve. Significant left-to-right shunting and AV valve regurgitation lead to excessive pulmonary blood flow and symptoms of **heart failure** (eg, tachypnea, poor feeding). Auscultatory findings of AV valve regurgitation (holosystolic, best heard at apex) and increased pulmonary venous return (mid-diastolic rumble) are characteristic.

(Choice A) DiGeorge syndrome (22q11.2 deletion) is characterized by thymic aplasia (T cell deficiency) and hypoparathyroidism (hypocalcemia). It is associated with tetralogy of Fallot, truncus arteriosus, and transposition of the great arteries.

(Choice C) Marfan syndrome (fibrillin-1 mutation) is associated with cystic medial necrosis of the aorta, which may result in dissecting aortic aneurysms and aortic valve regurgitation. Mitral valve prolapse is also common, but septal defects are not.

(Choice D) Mutations in frataxin, a mitochondrial protein important in iron homeostasis and respiratory function, cause Friedreich ataxia. It is characterized by spinocerebellar degeneration and is associated with hypertrophic cardiomyopathy, but not septal defects.

(Choice E) Mutations in tuberin and harmartin are seen in tuberous sclerosis. These patients may develop cardiac rhabdomyomas in ventricular walls and AV valves, cutaneous angiofibromas (adenoma sebaceum), central nervous system hamartomas, and renal cysts. However, septal defects are not characteristic.

(Choice F) A missing sex chromosome (monosomy) is seen in Turner syndrome (45,X), which is associated with bicuspid aortic valve (most common cardiac lesion) and coarctation of the aorta.

Educational objective:
A complete atrioventricular (AV) canal defect is comprised of an atrial septal defect, a ventricular septal defect, and

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cardiac defect in patients with **Down syndrome**, an autosomal trisomy. Failure of endocardial cushion fusion results in an ostium primum atrial septal defect; a ventricular septal defect; and a single AV valve. Significant left-to-right shunting and AV valve regurgitation lead to excessive pulmonary blood flow and symptoms of **heart failure** (eg, tachypnea, poor feeding). Auscultatory findings of AV valve regurgitation (holosystolic, best heard at apex) and increased pulmonary venous return (mid-diastolic rumble) are characteristic.

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Educational objective:
A complete atrioventricular (AV) canal defect is comprised of an atrial septal defect, a ventricular septal defect, and a common AV valve. It is the most common congenital cardiac anomaly associated with Down syndrome.

References

- Genetic syndromes associated with congenital heart disease.

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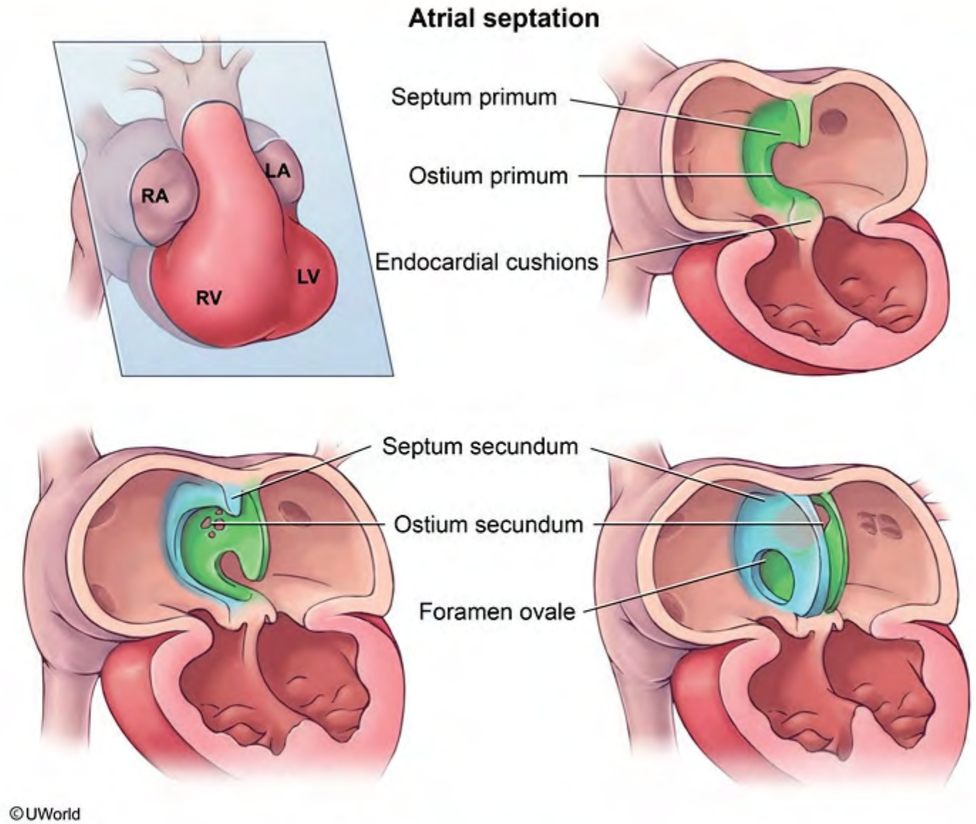

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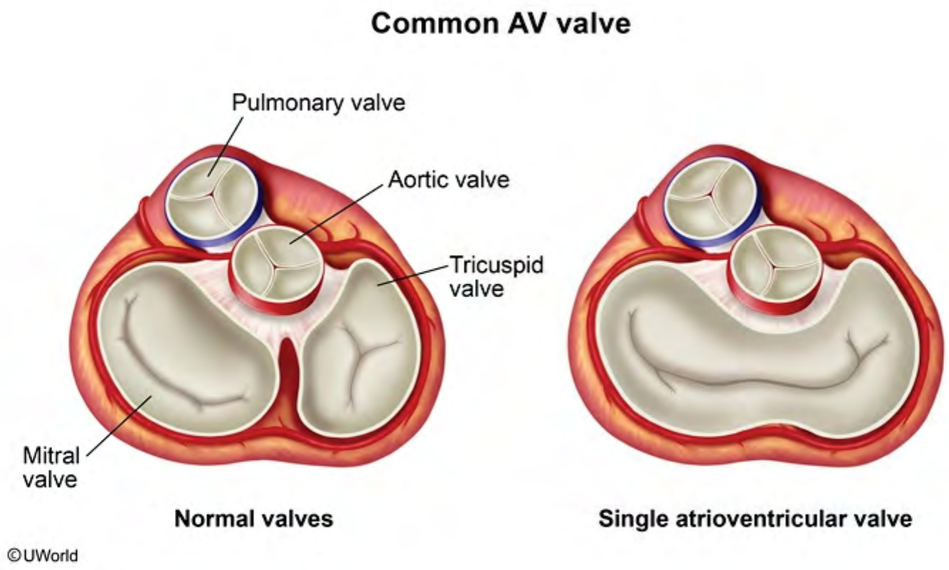

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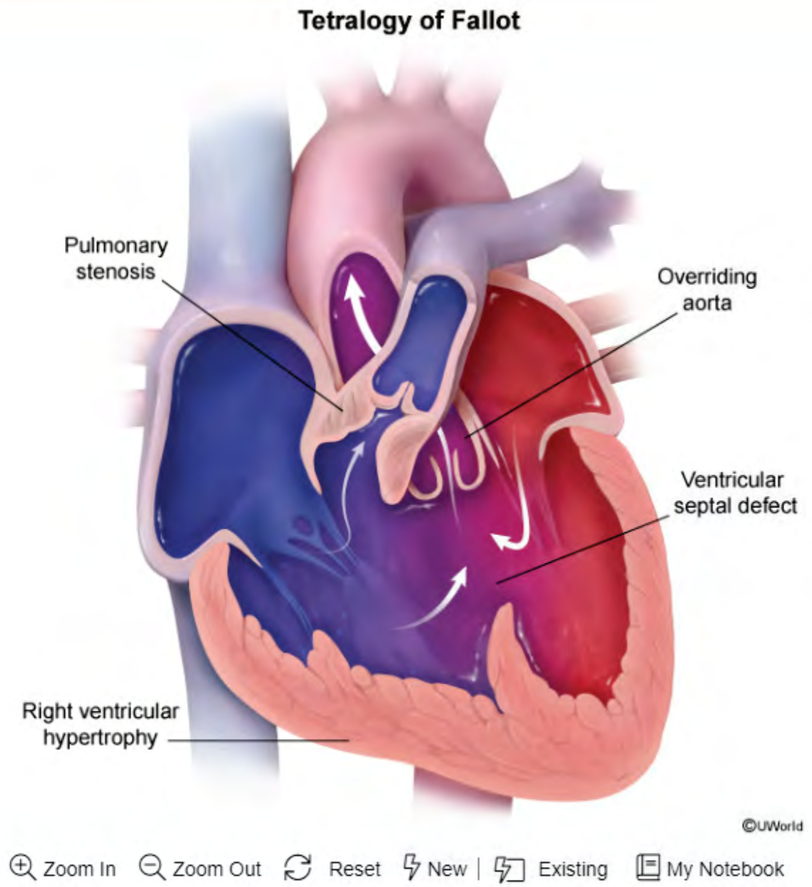

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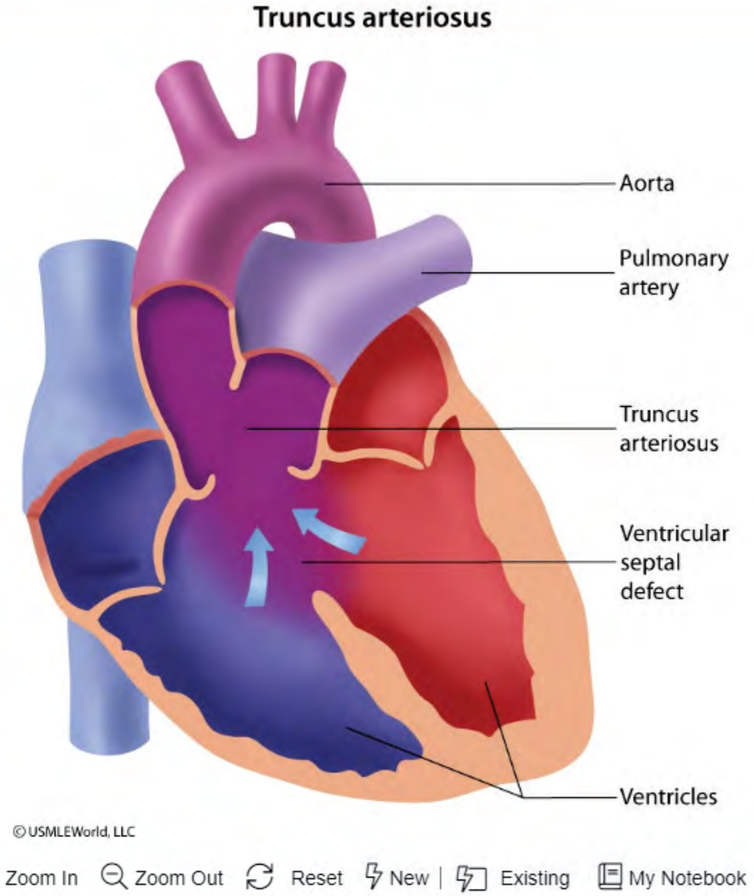

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Right — Left ventricle

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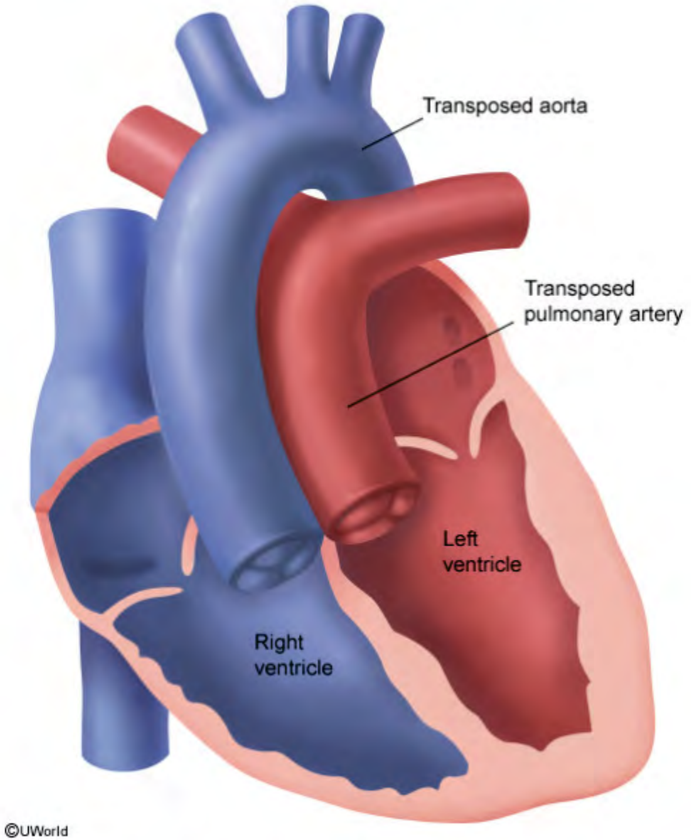
Transposition of the great vessels



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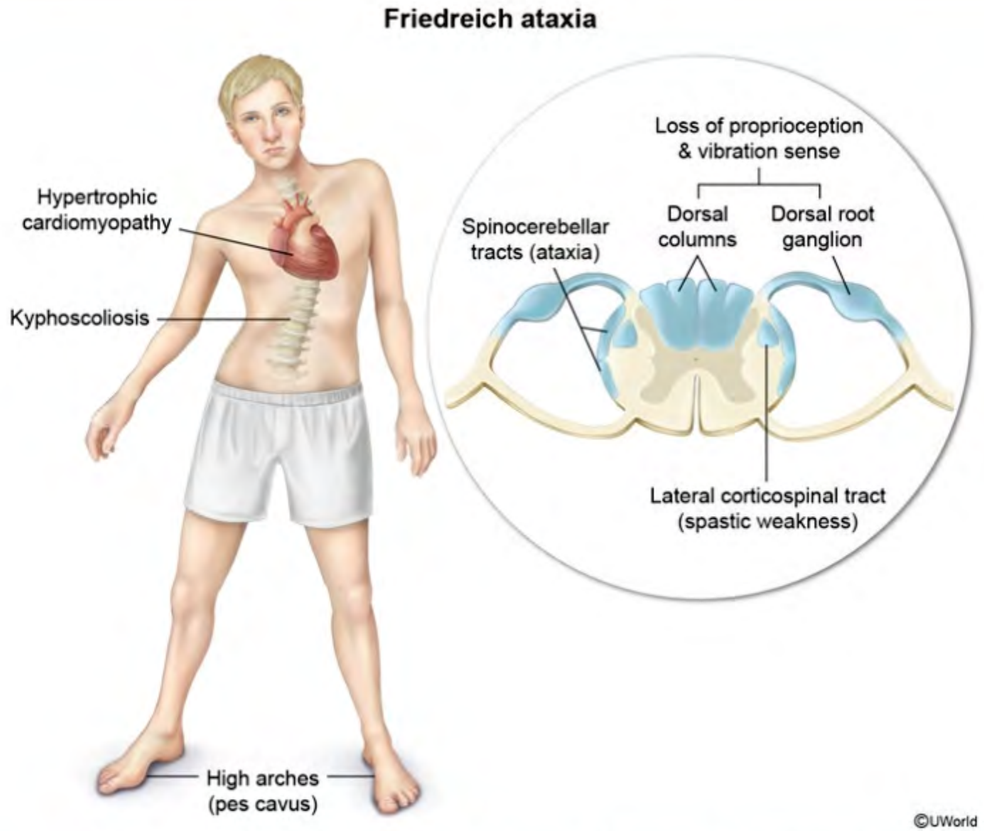

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A complete atrioventricular (AV) canal defect is comprised of an atrial septal defect, a ventricular septal defect, and

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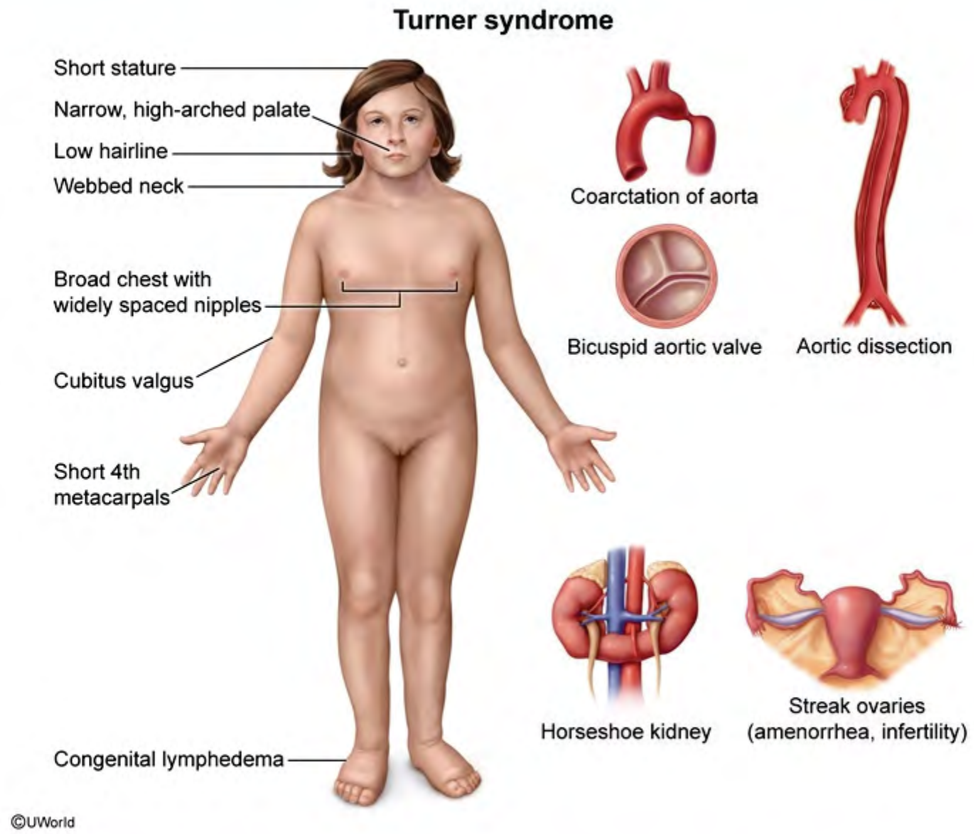

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Researchers are studying the coronary circulation in various healthy and diseased states. They measure blood flow and pressure (blue circle) in one of the epicardial coronary arteries of an experimental animal with cardiovascular physiology similar to that of humans. Then they clamp the artery to simulate 60% atherosclerotic stenosis and remeasure the flow and pressure distal to the lesion. Which of the following changes are most likely to be observed once steady state is achieved?

- A. A
- B. B
- C. C
- D. D

Submit

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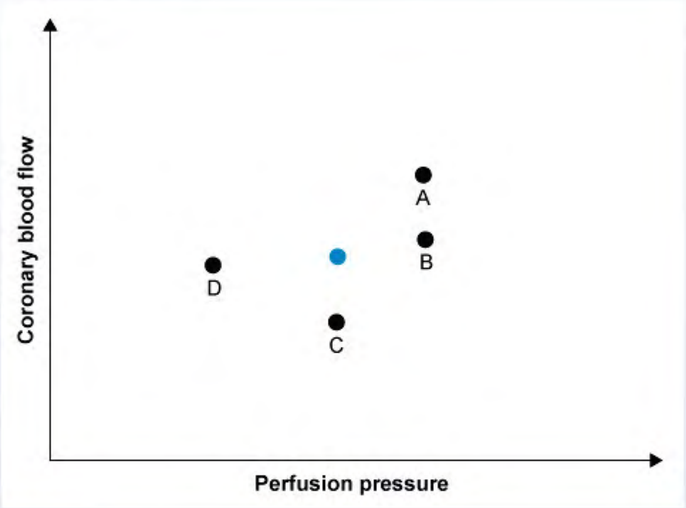

- A. A
- B. B
- C. C
- D. D

Submit

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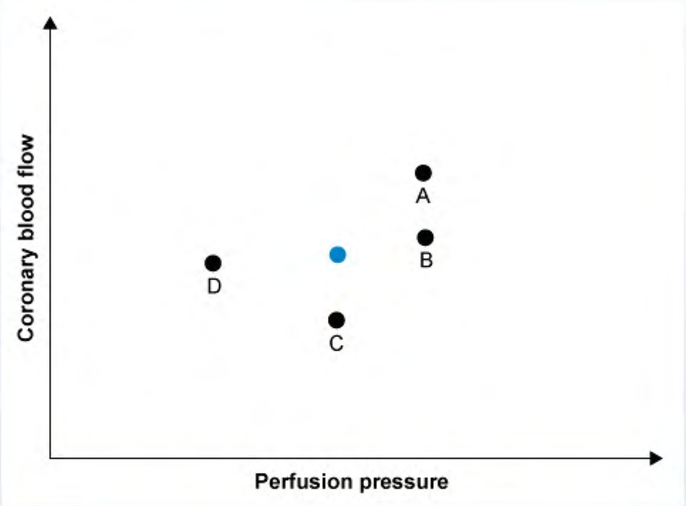

- A. A (5%)
- ✗ B. B (26%)
- C. C (35%)
- ✓ D. D (32%)

Incorrect
Correct answer
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32%
Answered correctly

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Time Spent

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Version

Explanation

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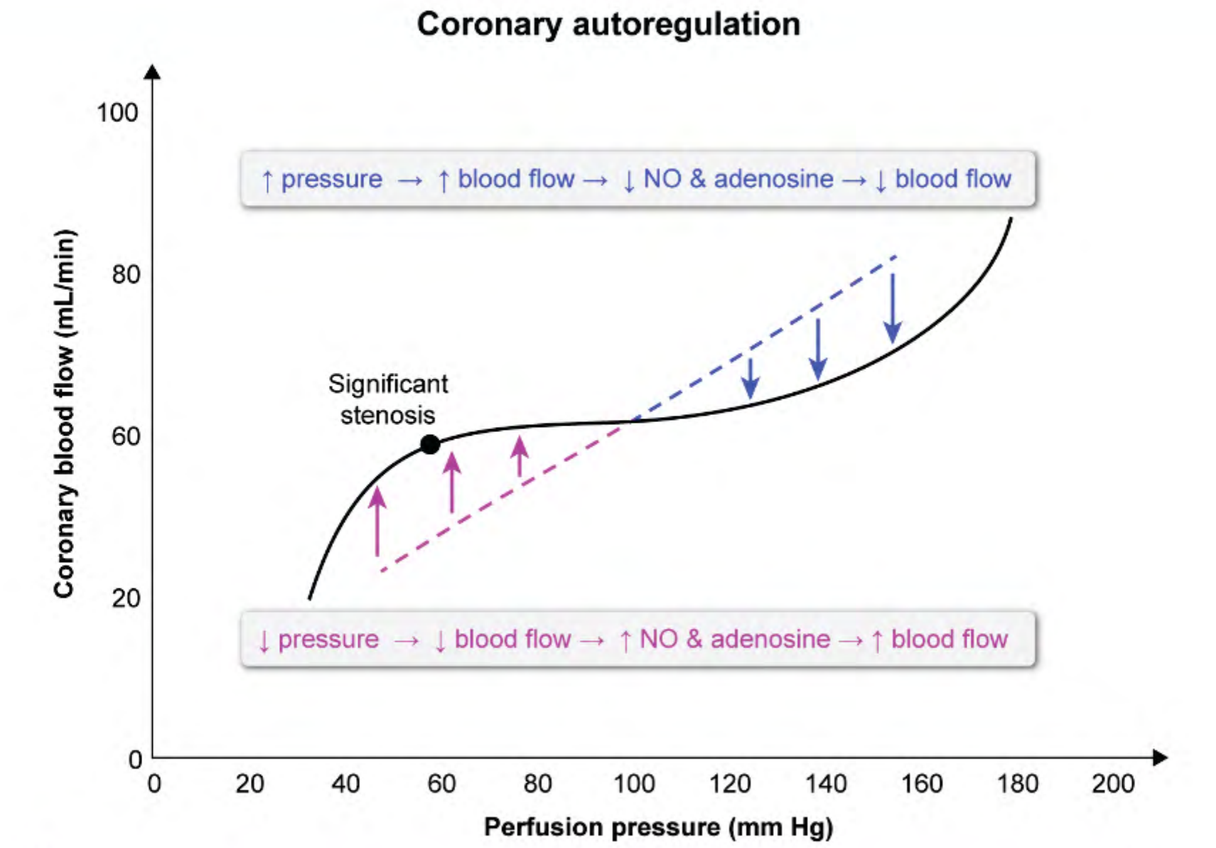

NO = nitric oxide.

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decrease in distal blood flow with corresponding myocardial ischemia. In response, the ischemic myocardium triggers the release of vasodilators (eg, **nitric oxide**, **adenosine**), to facilitate **arteriolar vasodilation** and reduce

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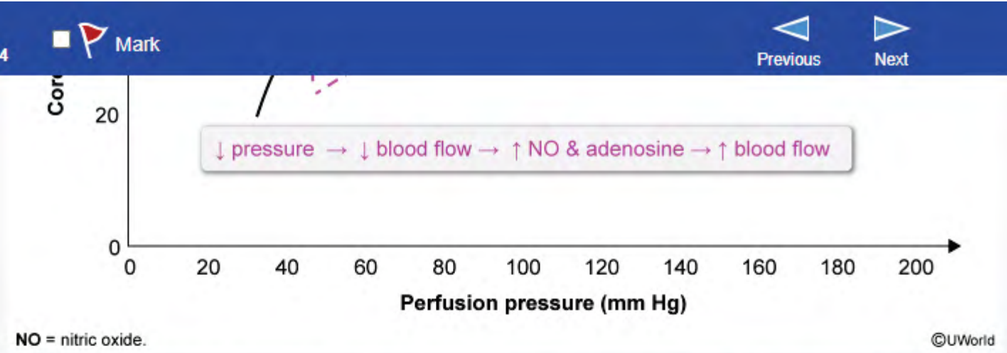

NO = nitric oxide.

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Coronary autoregulation helps maintain relatively constant coronary blood flow despite changes in perfusion pressure. A significant **stenosis** (eg, >50%) causes **reduced distal perfusion pressure**, resulting in an initial decrease in distal blood flow with corresponding myocardial ischemia. In response, the ischemic myocardium triggers the release of vasodilators (eg, **nitric oxide**, **adenosine**), to facilitate **arteriolar vasodilation** and reduce downstream vascular resistance. The reduced vascular resistance allows for a corrective increase in blood flow at the new, reduced perfusion pressure (blood flow = perfusion pressure / vascular resistance), keeping **blood flow nearly unchanged**.

The effects of coronary autoregulation are limited in the setting of extreme changes in perfusion pressure. Once arterioles reach the point of maximal vasodilation, vascular resistance cannot be further reduced, and a further decrease in perfusion pressure results in a precipitous drop in blood flow (as seen on the far-left side of the coronary autoregulation curve).

(Choices A, B, and C) Perfusion pressure decreases (rather than increases or remains the same) distal to a significant stenosis. Coronary autoregulation allows blood flow to remain nearly unchanged distal to the stenosis.

Educational objective:
Perfusion pressure decreases distal to a significant coronary artery stenosis, resulting in an initial decrease in blood flow. Coronary autoregulation stimulates arteriolar vasodilation, which reduces downstream vascular resistance and helps return blood flow to near-normal levels to prevent myocardial ischemia.

Physiology	Cardiovascular System	Coronary artery disease
Subject	System	Topic

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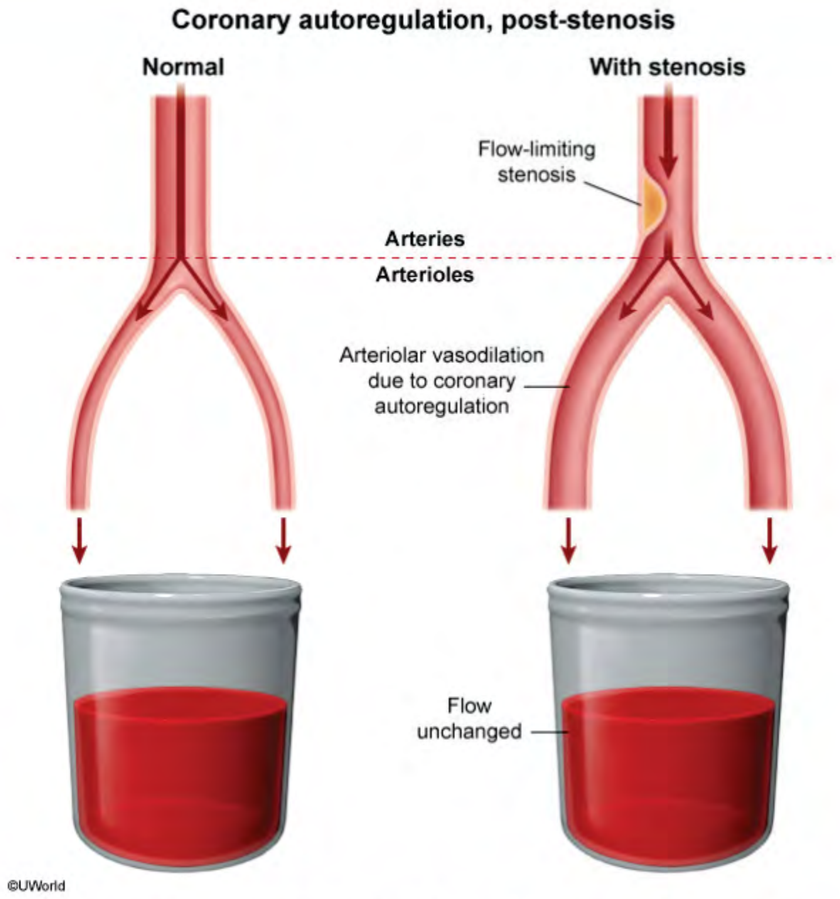

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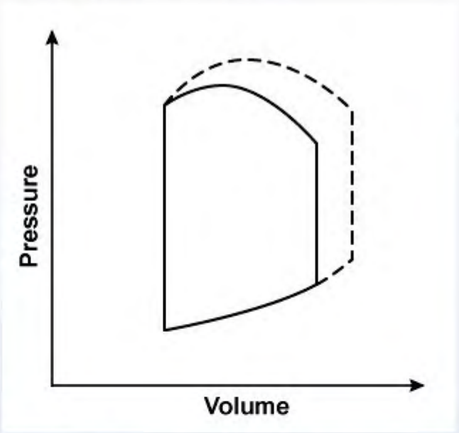
A 55-year-old man undergoes an elective surgery under general anesthesia. Two hours into the surgery, his left ventricular pressure-volume loop has changed from the solid line to the dashed line, as shown in the image below (the loop is unchanged in areas where there is no dashed line):



Which of the following is most likely responsible for this patient's hemodynamic change?

- A. Abdominal aorta clamping
- B. Blood volume loss
- C. Dobutamine administration
- D. Myocardial infarction
- E. Normal saline infusion

Submit

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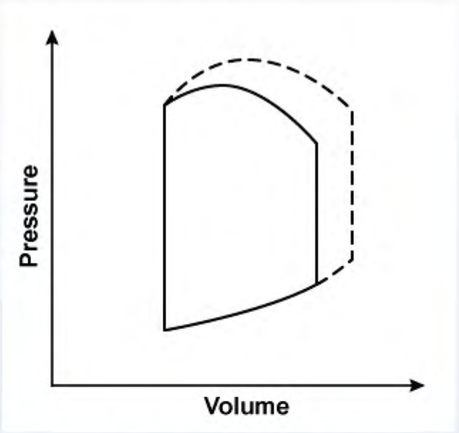

Which of the following is most likely responsible for this patient's hemodynamic change?

- ✗ A. Abdominal aorta clamping (5%)
- B. Blood volume loss (2%)
- C. Dobutamine administration (13%)
- D. Myocardial infarction (1%)
- ✓ E. Normal saline infusion (77%)

Incorrect
Correct answer
E

77%
Answered correctly

03 secs
Time Spent

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Explanation

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B. Blood volume loss (2%)

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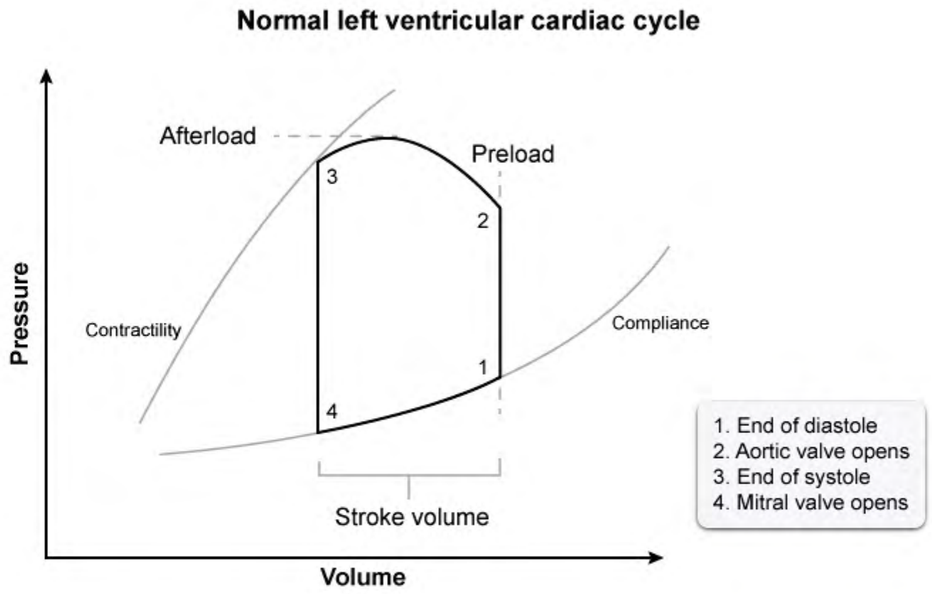

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The left ventricular (LV) pressure-volume loop in this patient shows a rightward shift in end-diastolic volume (point 1), indicating **increased preload**, which occurs with **infusion of normal saline** or other causes of increased blood volume (eg, blood transfusion, renal failure). Following the Frank-Starling law, the increased LV stretch from increased preload leads to **increased stroke volume** (the horizontal distance between points 1 and 3) and a **slight increase in afterload** (ie, the peak LV pressure reached during blood ejection). **Contractility is unchanged**.

(Choice A) Clamping of the abdominal aorta increases aortic pressure to increase afterload. Stroke volume decreases because the left ventricle is unable to eject as much blood against the increased aortic pressure. Preload is unchanged.

(Choice B) Blood loss decreases intravascular volume and leads to decreased preload. The decreased preload causes reduced stroke volume and a slight decrease in afterload (Frank-Starling).

(Choice C) Dobutamine primarily acts as a beta 1 receptor agonist and stimulates increased contractility. The increased contractility increases stroke volume and leads to a slight increase in afterload; preload is not increased.

(Choice D) Acute myocardial infarction disrupts myocardial function and often decreases contractility. The decreased contractility decreases stroke volume and causes a slight decrease in afterload. Preload is unchanged initially but is likely to increase if the decrease in contractility is sustained.

Educational objective:
A rightward shift of the bottom right corner (point 1) of the left ventricular (LV) pressure volume loop indicates increased end-diastolic volume or preload, which occurs with infusion of normal saline. The increased preload causes increased LV stretching and leads to increased stroke volume and a slight increase in afterload (Frank-Starling law).

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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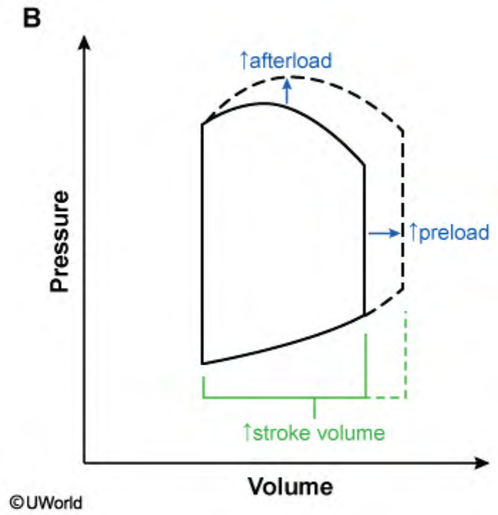

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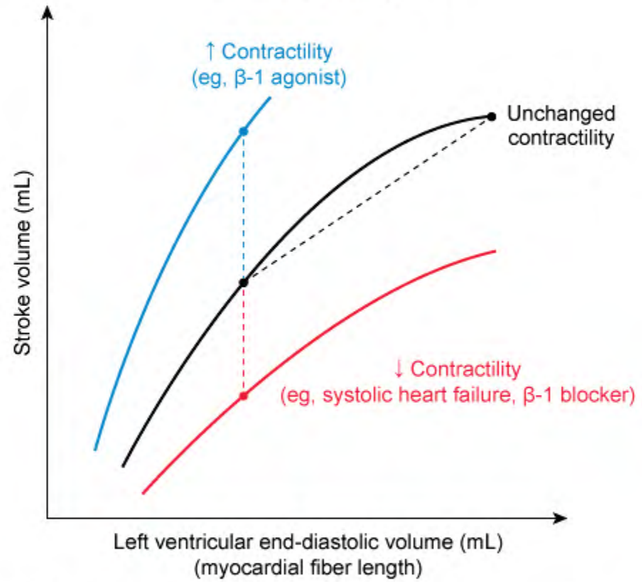
Frank-Starling law



*Contractility is intrinsic to the myocardium & does not change with changes in myocardial fiber length.

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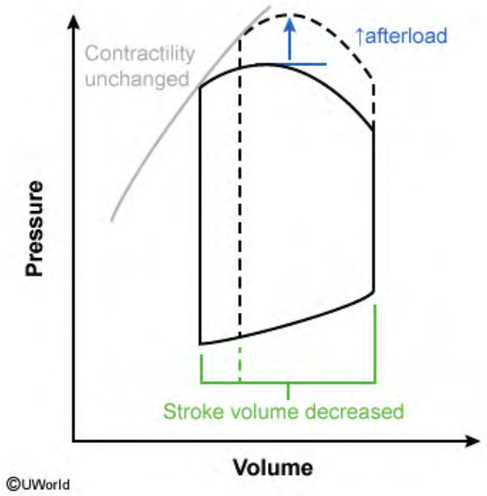

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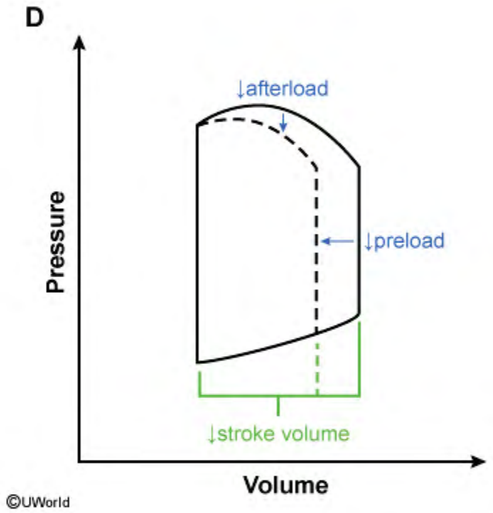

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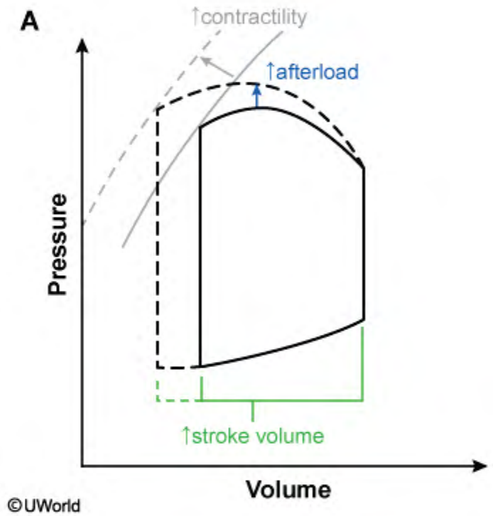

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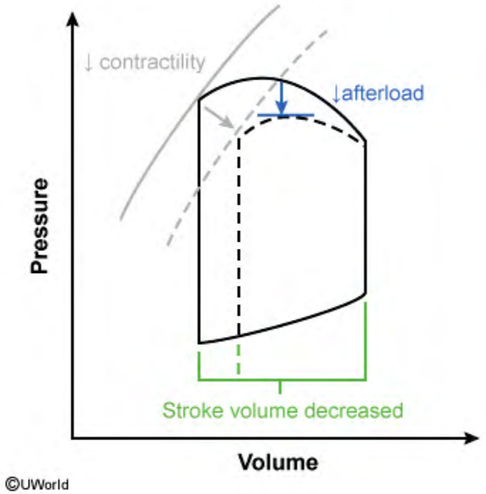

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A 63-year-old man comes to the emergency department due to pain and swelling of the right calf. He has a history of recurrent gastrointestinal hemorrhage and was discharged from the hospital 4 days ago following a prolonged admission for diverticular bleeding requiring multiple blood transfusions. Temperature is 36.7 C (98 F), blood pressure is 110/60 mm Hg, pulse is 92/min, and respirations are 18/min. Compression ultrasonography with Doppler reveals deep venous thrombosis in the right popliteal vein. A filter placement is planned to prevent embolization. A section of the patient's abdominal CT at the level of L2 is shown in the image below. The filter will most likely be placed in which of the following structures?

- A. A
- B. B
- C. C
- D. D
- E. E

Submit

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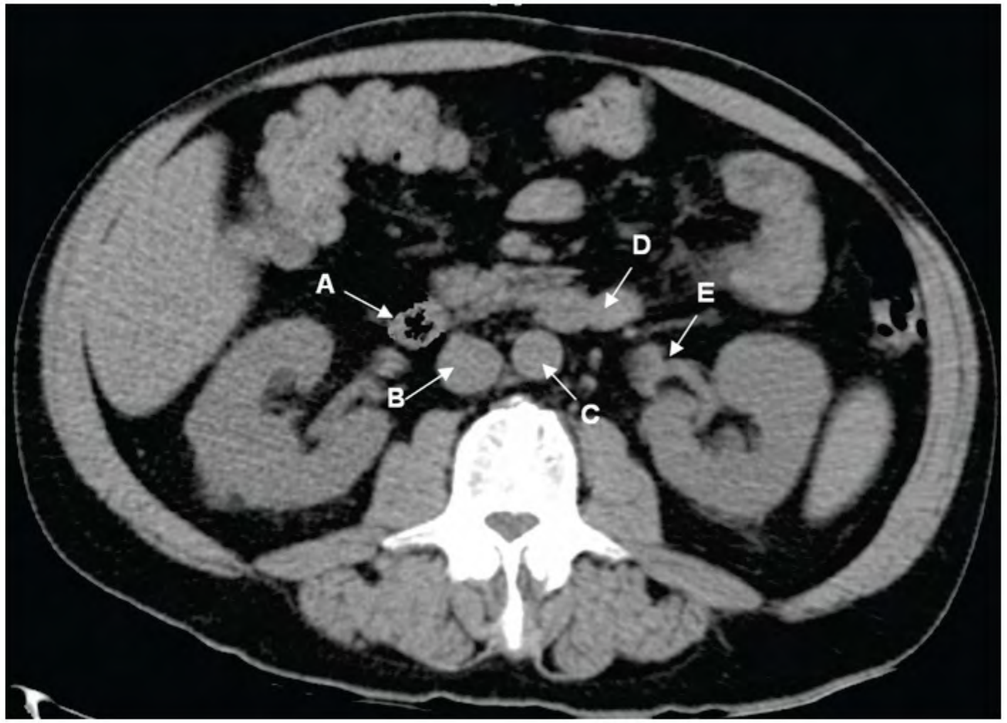

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B. B

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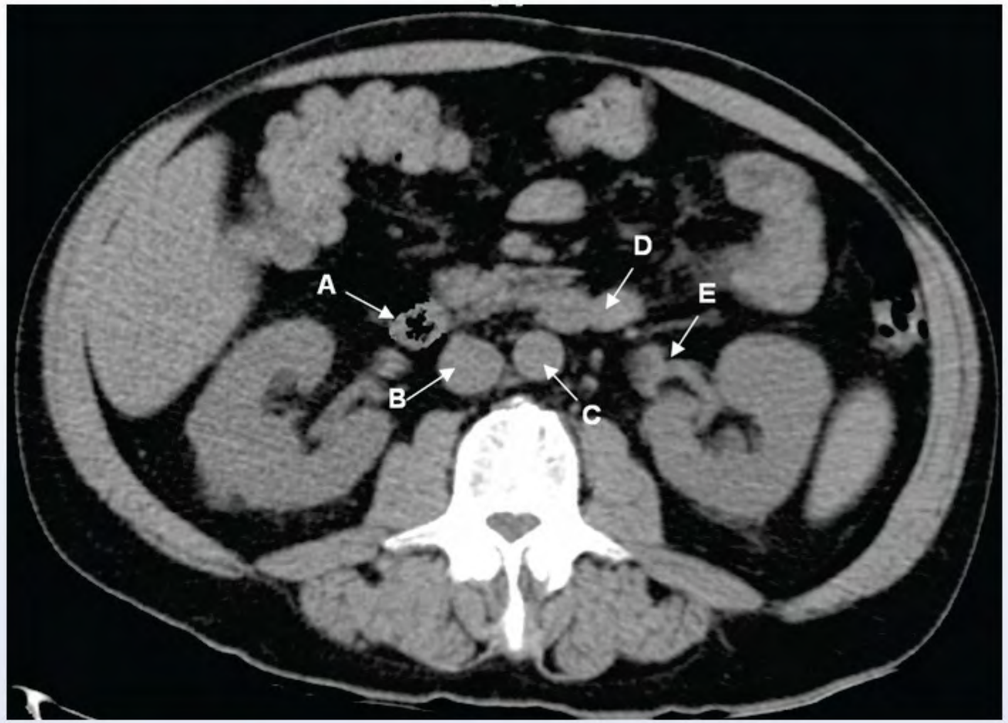
embolization. A section of the patient's abdominal CT at the level of L2 is shown in the image below. The filter will most likely be placed in which of the following structures?



- A. A
- B. B
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Submit

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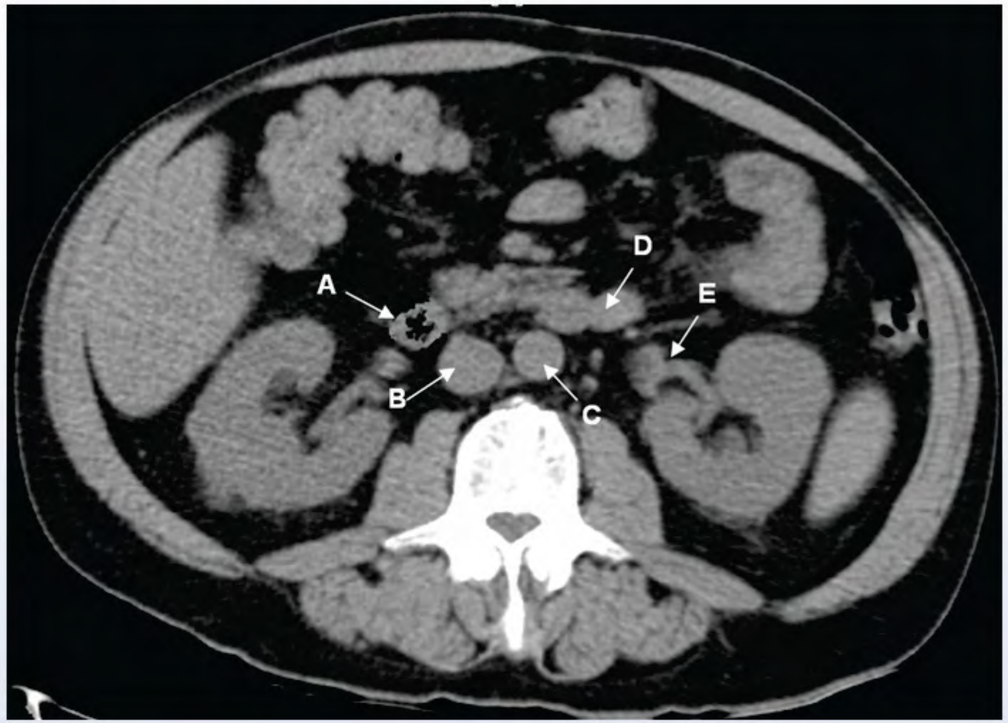

- A. A (11%)
- ✓ B. B (64%)
- C. C (16%)
- D. D (6%)
- E. E (1%)

Correct

64%
Answered correctly

12 secs
Time Spent

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Correct

64%
Answered correctly

12 secs
Time Spent

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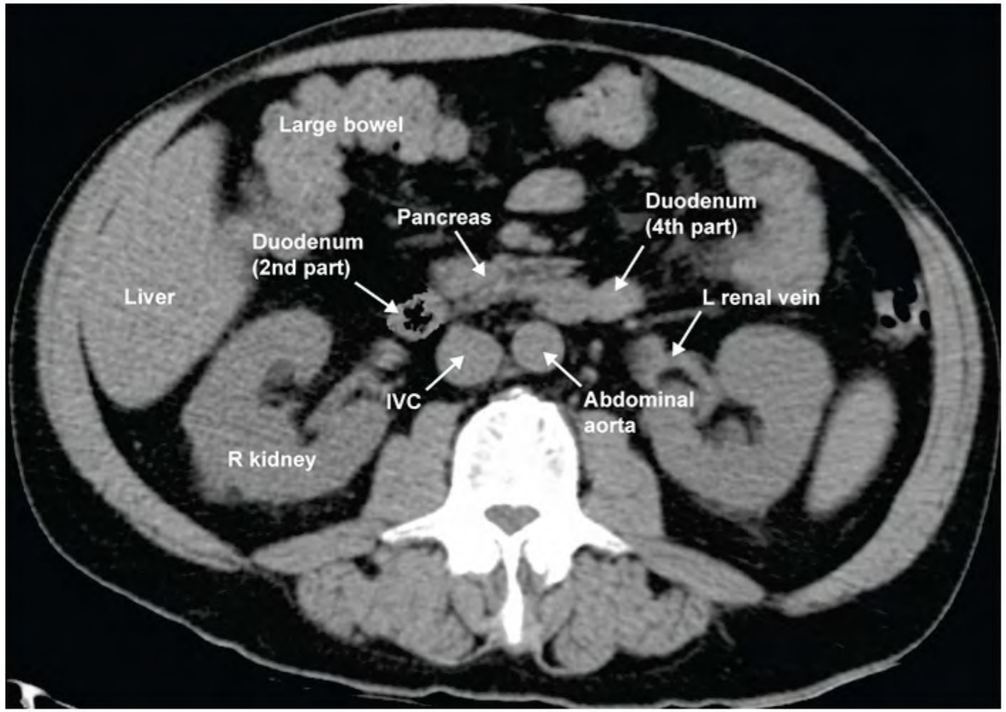

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CT images are always visualized as though standing at the patient's feet and looking through the patient's body

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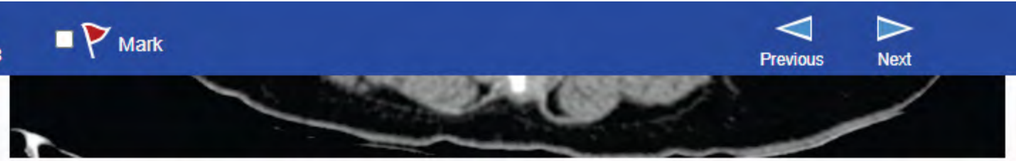

This patient with **deep venous thrombosis** (DVT) has had recurrent episodes of gastrointestinal hemorrhage, including a recent one requiring prolonged hospitalization. Given the risk of bleeding associated with anticoagulation therapy in this patient, **inferior vena cava (IVC) filter** placement is planned. IVC filters are designed to prevent the embolization of DVT from the legs to the lung vasculature (ie, pulmonary embolism), and are used in patients who have **contraindications to anticoagulation**.

CT images are always visualized as though standing at the patient's feet and looking through the patient's body toward the head; therefore, the left side of the image corresponds to the patient's right side. Both kidneys are viewed in this section, and the inferior pole of the right lobe of the liver is noted anterior to the right kidney and posterolateral to the large bowel. The filter will be placed in the IVC, which lies medial to the descending part of the duodenum and anterior to the right side of the vertebral body. The common iliac veins merge to become the IVC inferior to this plane of section at the L5 level.

(Choice A) With its dark center, representing an air-filled lumen, and mucosal folds, this structure is likely the second (descending) part of the duodenum, which typically lies at the level of L2.

(Choice C) This structure represents the abdominal aorta, which bifurcates at the level of L4. The structure is on the patient's left, so it is unlikely to be the IVC.

(Choice D) This structure likely represents the fourth (ascending) part of the duodenum. This CT section likely captures only the wall of the fourth part of the duodenum as it briefly courses transversely across the abdomen (before descending as the jejunum), so the lumen is not visible.

(Choice E) This structure is likely the left renal vein. The left renal vein receives drainage from the left gonadal vein and courses anterior to the aorta, but posterior to the superior mesenteric artery, before draining into the IVC.

Educational objective:
The inferior vena cava (IVC) courses through the abdomen and inferior thorax in a location anterior to the right half of the vertebral bodies. The renal veins join the IVC at the level of L1/L2, and the common iliac veins merge to become the IVC at the level of L5. IVC filters are placed in patients with deep venous thrombosis who have contraindications to anticoagulation therapy.

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Anatomy	Cardiovascular System	Pulmonary embolism
Subject	System	Topic

https://t.me/USMLEWorldStep1

A 60-year-old man comes to the office for a routine follow-up visit. He feels well overall except for an intermittent, mild, generalized headache. The patient has no known medical problems and takes no medications. He does not smoke, follows a generally healthy diet, and exercises daily. On examination, his blood pressure is 150/85 mm Hg, and he is started on lisinopril. At a follow-up visit, the patient's blood pressure is 128/78 mm Hg. He also has a dry cough that began a few weeks after starting lisinopril. This drug is stopped and losartan is now prescribed. The patient seems to be compliant with his medication; the cough resolves and he experiences no significant side effects. When compared with no treatment at all, this patient's current therapy is most likely to result in which of the following changes?

	Renin	Angiotensin I	Angiotensin II	Aldosterone	Bradykinin
○ A.	↓	↓	↓	↓	No change
○ B.	↑	↓	↓	↓	No change
○ C.	↑	↑	↓	↓	Increase
○ D.	↑	↑	↑	↓	No change
○ E.	↑	↑	↑	↑	No change

Submit

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	Renin	Angiotensin I	Angiotensin II	Aldosterone	Bradykinin	
✗ A.	↓	↓	↓	↓	No change	(8%)
B.	↑	↓	↓	↓	No change	(9%)
C.	↑	↑	↓	↓	Increase	(7%)
✓ D.	↑	↑	↑	↓	No change	(65%)
E.	↑	↑	↑	↑	No change	(8%)

Incorrect
Correct answer
D

65%
Answered correctly

03 secs
Time Spent

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Explanation

Renin-angiotensin-aldosterone system & antihypertensives

Angiotensinogen

https://t.me/USMLEWorldStep1

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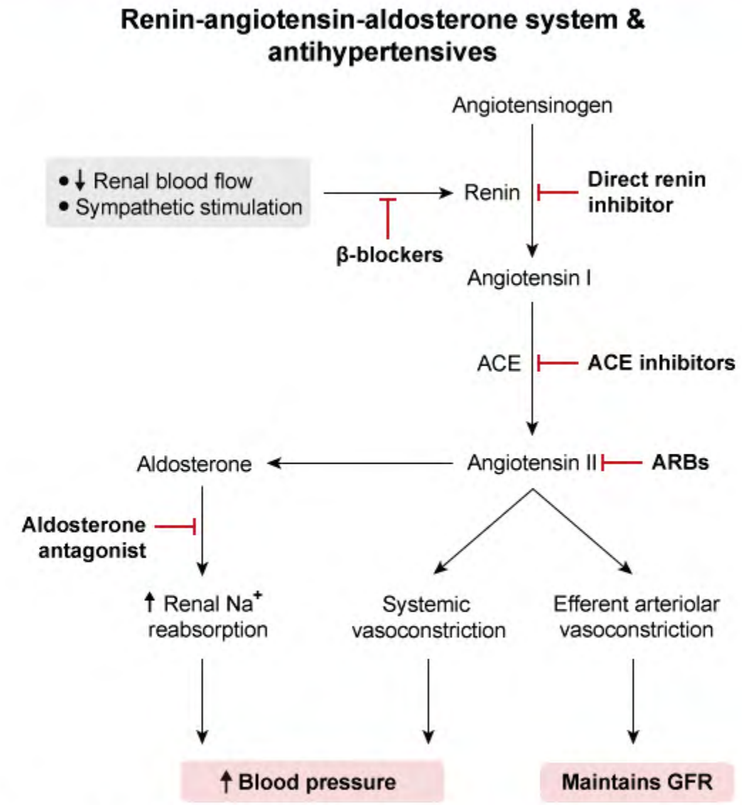

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Angiotensinogen

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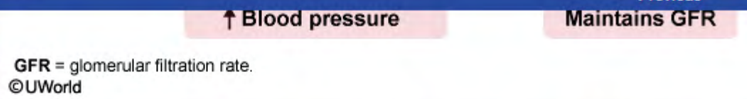

GFR = glomerular filtration rate.
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The renin-angiotensin-aldosterone system (RAAS) regulates arterial blood pressure and extracellular fluid balance. Renin release is stimulated by decreased sodium delivery to the distal tubule, low blood pressure (eg, low intravascular volume), and β1 sympathetic activity. Renin converts angiotensinogen to angiotensin I. Angiotensin I is then converted to angiotensin II by angiotensin-converting enzyme (ACE), which also degrades bradykinin. Angiotensin II stimulates the **type 1 angiotensin II receptors** found on vascular smooth muscle (causing vasoconstriction) and the adrenal cortex (stimulating aldosterone secretion). Systemic vasoconstriction and increased aldosterone-induced sodium reabsorption raise blood pressure, inhibiting further renin release from the kidneys (negative feedback).

Angiotensin II receptor blockers (ARBs) such as losartan competitively bind to angiotensin II receptors and block the effects of angiotensin II, resulting in **vascular smooth muscle relaxation** and **decreased aldosterone** secretion. This reduces blood pressure, stimulating renal renin production, which in turn increases conversion of angiotensin I to angiotensin II. Because ACE function remains intact with the use of ARBs, bradykinin levels are not significantly affected. This is in contrast to ACE inhibitors, which lower angiotensin II and aldosterone levels but increase bradykinin levels **(Choice C)**. Bradykinin is thought to increase prostaglandin production, which induces coughing due to bronchial irritation.

(Choice A) β1 adrenergic blockers would inhibit renin release from the juxtaglomerular apparatus, thereby suppressing the entire renin-angiotensin cascade (although lowering of the blood pressure would somewhat blunt this response). ACE activity is not regulated by renin, and so bradykinin levels are unchanged.

(Choice B) Direct renin inhibitors reduce plasma renin activity, which reduces levels of all downstream hormones. Suppression of the whole cascade lowers blood pressure, which would stimulate increased renin production. Plasma renin activity, however, is inhibited.

(Choice E) This combination of changes is seen with mineralocorticoid receptor antagonist (eg, spironolactone) or diuretic use. Mineralocorticoid receptor antagonists block aldosterone activity by binding to the mineralocorticoid receptor. Diuretics would cause a fall in blood pressure, stimulating the RAAS and increasing blood levels of all hormones.

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Educational objective:
Angiotensin II receptor blockers (ARBs) work by blocking angiotensin II type 1 receptors, inhibiting the effects of angiotensin II. This results in arterial vasodilation and decreased aldosterone secretion. The resulting fall in blood pressure increases renin, angiotensin I, and angiotensin II levels. ARBs do not affect the activity of angiotensin-converting enzyme, and therefore they do not affect bradykinin degradation and do not cause cough.

References

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A 55-year-old man is immediately brought to the hospital after experiencing a sudden collapse at home 20 minutes ago. The patient reported chest pain and then collapsed to the floor while trying to sit down. Medical history is significant for type 2 diabetes mellitus and hypertension. Temperature is 36.7 C (98.1 F), blood pressure is 80/50 mm Hg, pulse is 120/min, and respirations are 22/min. On examination, the patient is in marked respiratory distress. Bilateral crackles are present. An S3 is audible. ECG reveals ST-segment elevation in leads V2 through V6. Which of the following hemodynamic changes are most likely present in this patient?

	Pulmonary capillary wedge pressure	Central venous pressure	Coronary perfusion pressure
○ A.	Decreased	Decreased	Decreased
○ B.	Decreased	Increased	Decreased
○ C.	Increased	Decreased	Increased
○ D.	Increased	Increased	Decreased
○ E.	Increased	Increased	Increased

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A 55-year-old man is immediately brought to the hospital after experiencing a sudden collapse at home 20 minutes ago. The patient reported chest pain and then collapsed to the floor while trying to sit down. Medical history is significant for type 2 diabetes mellitus and hypertension. Temperature is 36.7 C (98.1 F), blood pressure is 80/50 mm Hg, pulse is 120/min, and respirations are 22/min. On examination, the patient is in marked respiratory distress. Bilateral crackles are present. An S3 is audible. ECG reveals ST-segment elevation in leads V2 through V6. Which of the following hemodynamic changes are most likely present in this patient?

		Pulmonary capillary wedge pressure	Central venous pressure	Coronary perfusion pressure	
	A.	Decreased	Decreased	Decreased	(6%)
✗	B.	Decreased	Increased	Decreased	(7%)
	C.	Increased	Decreased	Increased	(6%)
✓	D.	Increased	Increased	Decreased	(69%)
	E.	Increased	Increased	Increased	(9%)

Incorrect
Correct answer
D

69%
Answered correctly

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Time Spent

2023
Version

Explanation

Hemodynamic measurements in shock				
Parameter	Hypovolemic shock	Cardiogenic shock	Obstructive shock	Distributive shock

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Hemodynamic measurements in shock				
Parameter	**Hypovolemic shock**	**Cardiogenic shock**	**Obstructive shock**	**Distributive shock**
CVP (right-sided preload)	↓	↑	↑	↓
PCWP (left-sided preload)	↓	↑	↓*	↓
Cardiac index (LV output)	↓	↓	↓	↑**
SVR (afterload)	↑	↑	↑	↓
SvO_2	↓	↓	↓	↑**

*In tamponade, left-sided preload is decreased, but measured PCWP is paradoxically increased due to external compression by pericardial fluid.

**Cardiac index & SvO_2 are usually decreased in neurogenic shock due to impaired sympathetic reflexes.

CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **SvO_2** = mixed venous oxygen saturation; **SVR** = systemic vascular resistance.

This patient with sudden-onset chest pain and ST-segment elevation in contiguous ECG leads has an ST-elevation **myocardial infarction** (MI). Hypotension, pulmonary crackles, and an audible S3 suggest MI-induced acute left ventricular (LV) failure and **cardiogenic shock**.

In cardiogenic shock, LV systolic failure causes increased LV end-diastolic pressure, which is transmitted backward

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This patient with sudden-onset chest pain and ST-segment elevation in contiguous ECG leads has an ST-elevation **myocardial infarction** (MI). Hypotension, pulmonary crackles, and an audible S3 suggest MI-induced acute left ventricular (LV) failure and **cardiogenic shock**.

In cardiogenic shock, LV systolic failure causes increased LV end-diastolic pressure, which is transmitted backward to cause **increased pulmonary capillary wedge pressure** (PCWP) (an estimate of left atrial pressure) and pulmonary edema. The increased pressure also impairs right ventricular function, resulting in **increased central venous pressure** (CVP). Markedly decreased cardiac output leads to hypotension with **decreased coronary perfusion pressure** (as coronary perfusion pressure is driven by aortic diastolic pressure) **(Choices C and E)**. In response to the reduced cardiac output, compensatory peripheral vasoconstriction occurs to increase systemic vascular resistance in an effort to maintain organ and tissue perfusion.

(Choice A) Decreased CVP, PCWP, and coronary perfusion pressure are expected with hypovolemic and septic shock.

(Choice B) Increased CVP with decreased PCWP and coronary perfusion pressure are expected with obstructive shock due to pulmonary embolism (ie, blood flow blocked at the pulmonary arteries) or tension pneumothorax (ie, blood flow blocked at the right atrium due to thoracic pressure–induced vena cava collapse).

Educational objective:
Myocardial infarction may be complicated by acute left ventricular failure (LV) and cardiogenic shock. In cardiogenic shock, increased LV end-diastolic pressure is transmitted backward to cause increased pulmonary capillary wedge pressure (evidenced by pulmonary edema) and increased central venous pressure. There is markedly decreased cardiac output, which causes hypotension and decreased coronary perfusion pressure.

Physiology	Cardiovascular System	Cardiogenic shock
Subject	System	Topic

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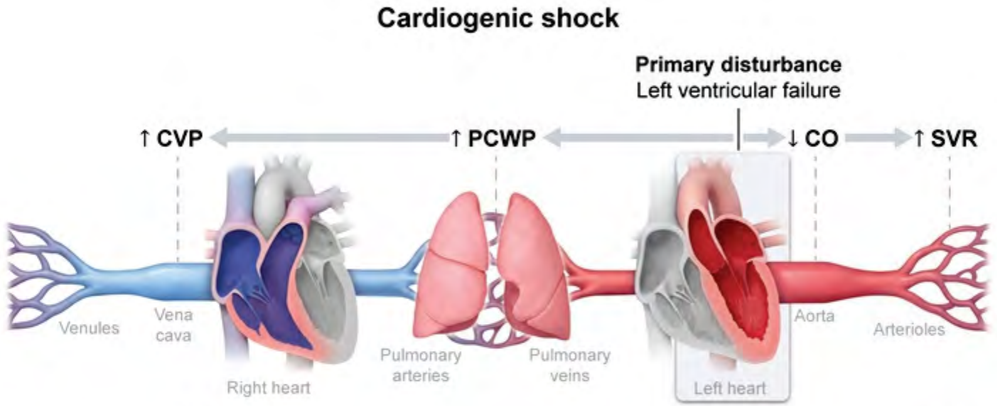

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Physiology
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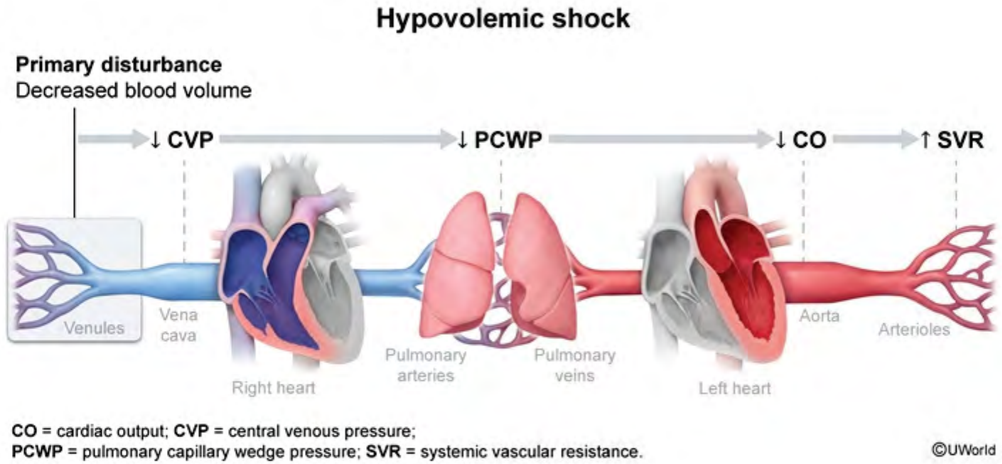

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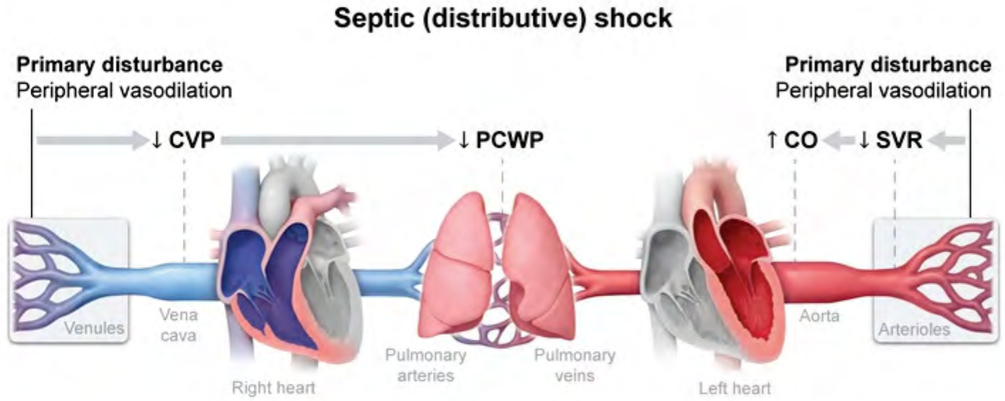

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Physiology
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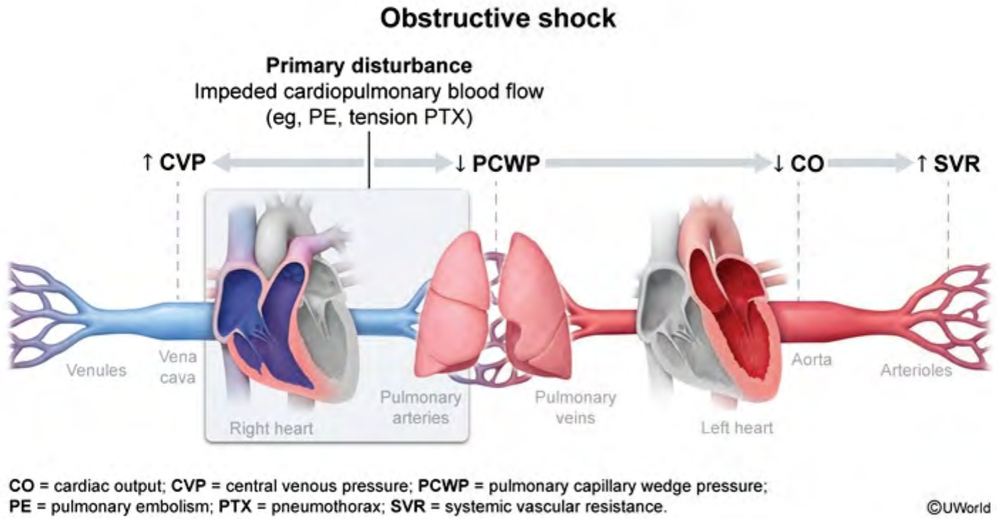

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Physiology
Subject

Cardiovascular System
System

Cardiogenic shock
Topic

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A 42-year-old man comes to the emergency department after a syncopal episode preceded by palpitations and lightheadedness. The patient has no significant medical history and takes no medications. He does not use tobacco, alcohol, or illicit drugs. The patient is a computer programmer and spends most of his time indoors. Vital signs are within normal limits with no orthostatic changes. Heart and lung sounds are normal. The liver span is increased with no tenderness. The skin appears darkly tanned. ECG demonstrates sinus node dysfunction, and echocardiography reveals abnormal diastolic relaxation of the left ventricle. Which of the following is the most likely cause of this patient's condition?

- A. Amyloid deposition
- B. Cortisol deficiency
- C. Granuloma formation
- D. Human leukocyte antigen gene mutation
- E. Iron accumulation
- F. Lysosomal glycogen accumulation

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- A. Amyloid deposition (6%)
- ✗ B. Cortisol deficiency (8%)
- C. Granuloma formation (0%)
- D. Human leukocyte antigen gene mutation (1%)
- ✓ E. Iron accumulation (80%)
- F. Lysosomal glycogen accumulation (2%)

Incorrect
Correct answer
E

80%
Answered correctly

03 mins, 12 secs
Time Spent

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Version

Explanation

Cardiac hemochromatosis	
Pathophysiology	• Excess intestinal iron absorption • Deposition of iron (ferritin, hemosiderin, free iron) in myocardium • Oxidative injury to myocytes
Cardiomyopathy	• Early: diastolic left ventricular dysfunction (restrictive pattern)

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Explanation

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Cardiomyopathy	• Early: diastolic left ventricular dysfunction (restrictive pattern) • Later: cardiac remodeling & dilated cardiomyopathy
Conduction system disease	• Sinus node dysfunction (sick sinus syndrome) • Atrial & ventricular arrhythmias • Sudden cardiac death

This patient has syncope associated with diastolic dysfunction, **hepatomegaly**, and skin **hyperpigmentation**. This presentation is suggestive of **hereditary hemochromatosis** (HH), an autosomal recessive disorder characterized by excessive intestinal iron absorption with **accumulation of the excess iron** in parenchymal tissues.

Cardiac manifestations of HH include heart failure and conduction system disease. In early stages, myocardial iron overload is expressed as **diastolic left ventricular (LV) dysfunction** with a restrictive filling pattern. As the disease advances, progressive ventricular remodeling leads to **dilated cardiomyopathy** and reduced LV ejection fraction. Iron deposition can also affect the cardiac conduction system. **Sinus node dysfunction** (ie, sick sinus syndrome) can lead to presyncope or syncope; malignant arrhythmias with sudden cardiac death occasionally occur.

On light microscopy, cardiac hemochromatosis is visible as brown, granular deposits (arrow) in myocytes that stain strongly with Prussian blue. Cardiomyopathy can be the first presenting manifestation of HH, although careful assessment can usually find evidence of iron deposition elsewhere (eg, elevated hepatic transaminases, hepatomegaly). Treatment includes therapeutic phlebotomy to remove excess iron stores, which can lead to

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(Choice A) Amyloidosis is caused by extracellular deposition of misfolded amyloid proteins. Multiorgan involvement (eg, cardiomyopathy, hepatomegaly) is common, but amyloidosis frequently involves the kidney (eg, nephrotic syndrome), nerves (eg, neuropathy), and muscles (eg, macroglossia). It is not associated with hyperpigmentation.

(Choice B) Primary adrenal insufficiency (ie, cortisol deficiency) can cause hyperpigmentation due to cosecretion of melanocyte-stimulating hormone with ACTH (both derived from proopiomelanocortin). However, hepatomegaly is not seen, and although hypotension is common, sinus node and diastolic dysfunction are not.

(Choice C) Sarcoidosis results from widespread formation of noncaseating granulomas. Cardiac infiltration can cause conduction blockade, and liver involvement may result in hepatomegaly. However, lung symptoms (eg, dyspnea, cough) are more common, and skin manifestations include papules and nodules rather than hyperpigmentation.

(Choice D) HH is associated with mutations in the *HFE* gene; the HFE protein binds to the transferrin receptor

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(Choice D) HH is associated with mutations in the *HFE* gene; the HFE protein binds to the transferrin receptor and regulates intestinal and hepatic iron uptake. Human leukocyte antigen (HLA) mutations are associated with autoimmune disorders (eg, celiac disease, type 1 diabetes).

(Choice F) Glycogen storage disease type II (ie, Pompe disease) is caused by a deficiency in alpha-glucosidase (acid maltase). It manifests shortly after birth with hypertrophic cardiomyopathy, hepatomegaly, muscle weakness, and failure to thrive. Skin hyperpigmentation is not typical.

Educational objective:
Hereditary hemochromatosis is characterized by excessive intestinal iron absorption with deposition of iron in parenchymal tissues. Cardiac manifestations include diastolic dysfunction (initial), dilated cardiomyopathy (later), and conduction system disease (eg, sinus node dysfunction).

Pathology	Cardiovascular System	Hemochromatosis
Subject	System	Topic

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This patient has syncope associated with diastolic dysfunction, **hepatomegaly**, and skin **hyperpigmentation**.

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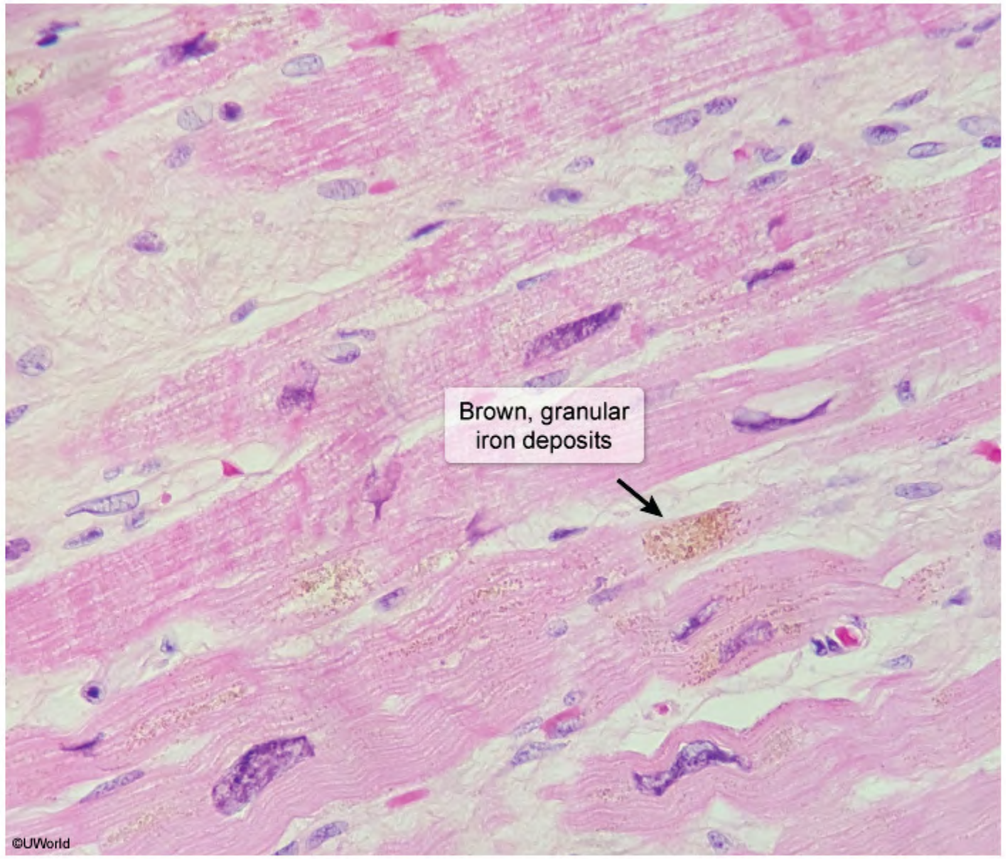

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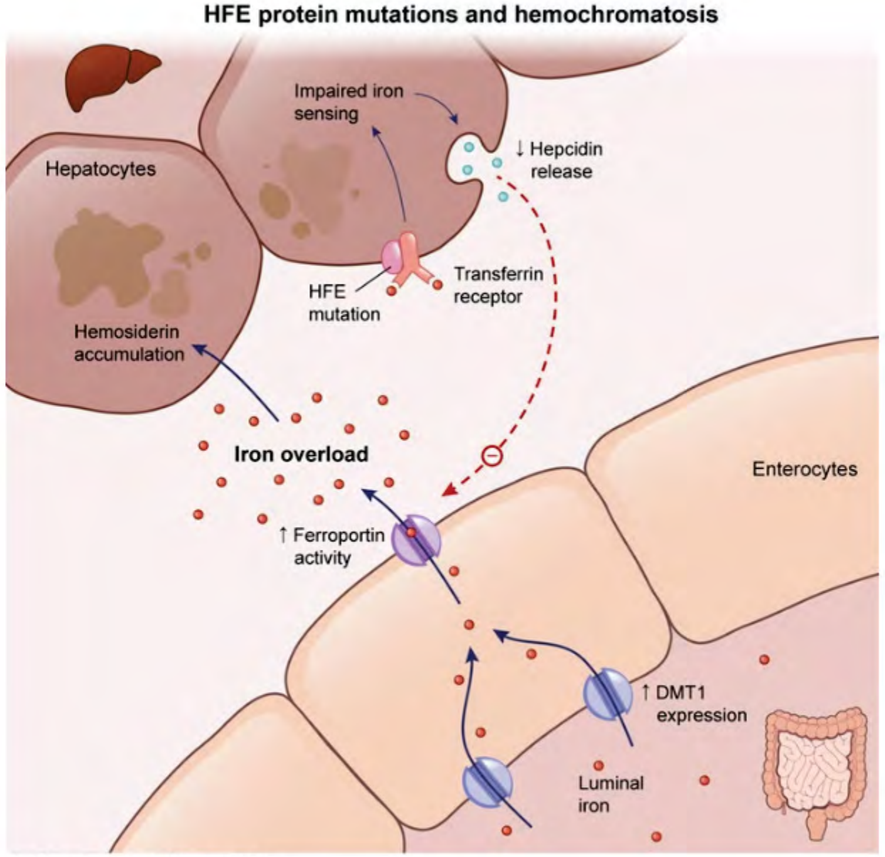

DMT1 = divalent metal transporter 1.
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An 82-year-old man comes to the office due to progressive dyspnea and fatigue over the last year, which now limit his daily activities. He has also noticed bilateral swelling of his feet. The patient has hypertension, which is controlled with amlodipine. His blood pressure is 122/72 mm Hg and pulse is 55/min. Physical examination reveals elevated jugular venous pressure with rapid 'y' descent and a prominent S4. Abdominal examination shows moderate ascites. The patient has 3+ bilateral lower extremity pitting edema. Echocardiogram reveals left atrial enlargement with marked left ventricular hypertrophy and normal left ventricular ejection fraction. Complete blood count and basic metabolic panel are within normal limits. Endomyocardial biopsy findings are shown below.

Which of the following is the most likely diagnosis?

- A. Cardiac myxoma
- B. Cardiac sarcoidosis
- C. Chagas disease
- D. Hemochromatosis
- E. Senile amyloidosis

Submit

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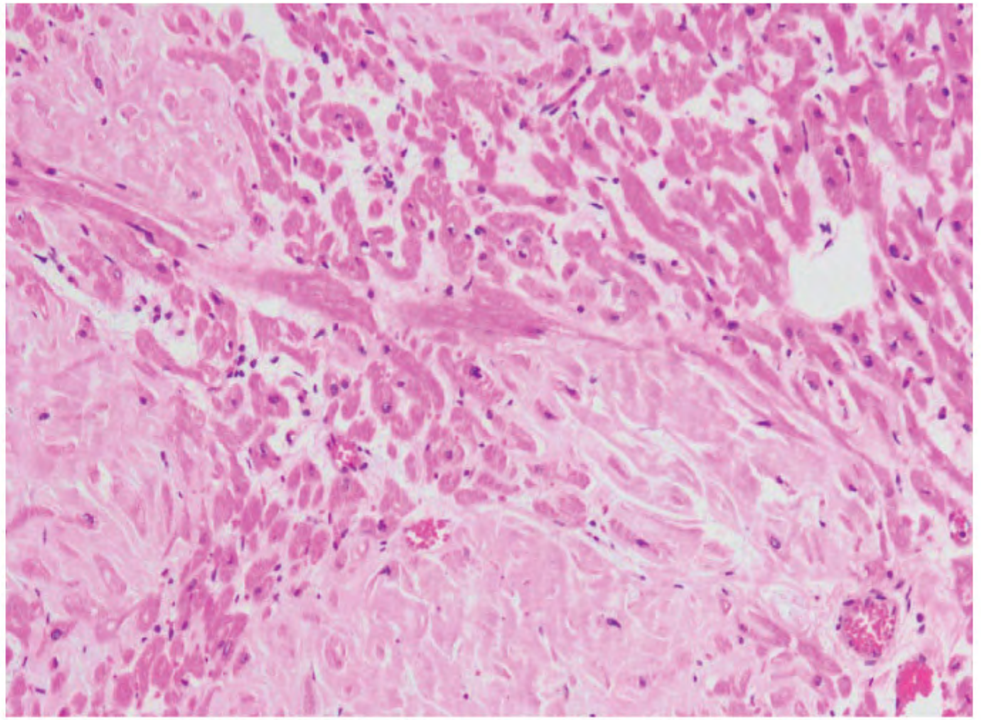

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Which

A. Cardiac myxoma

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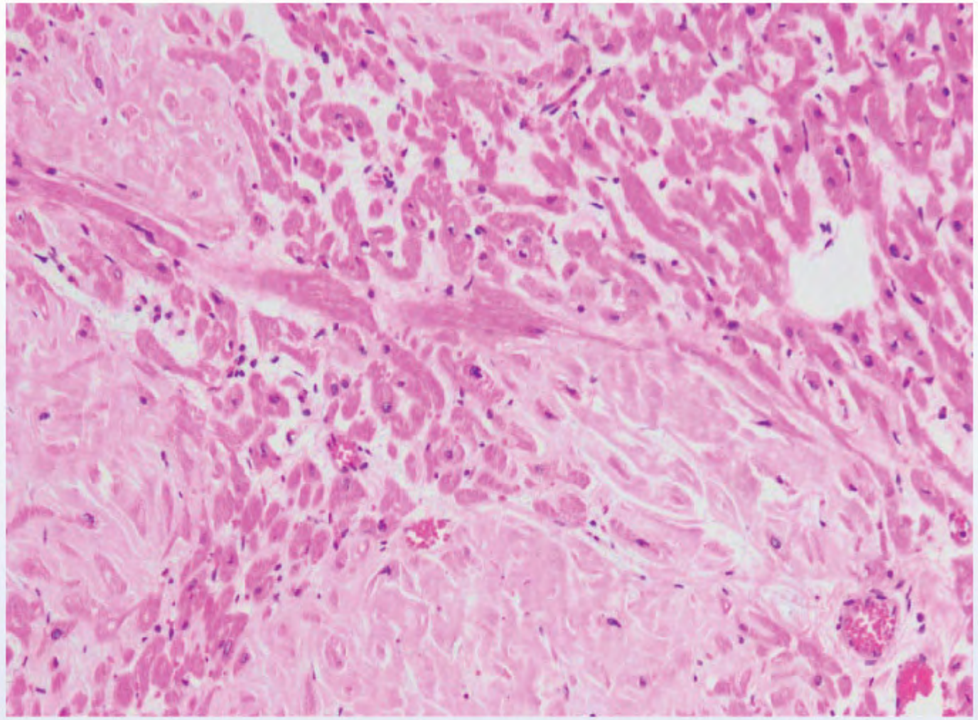

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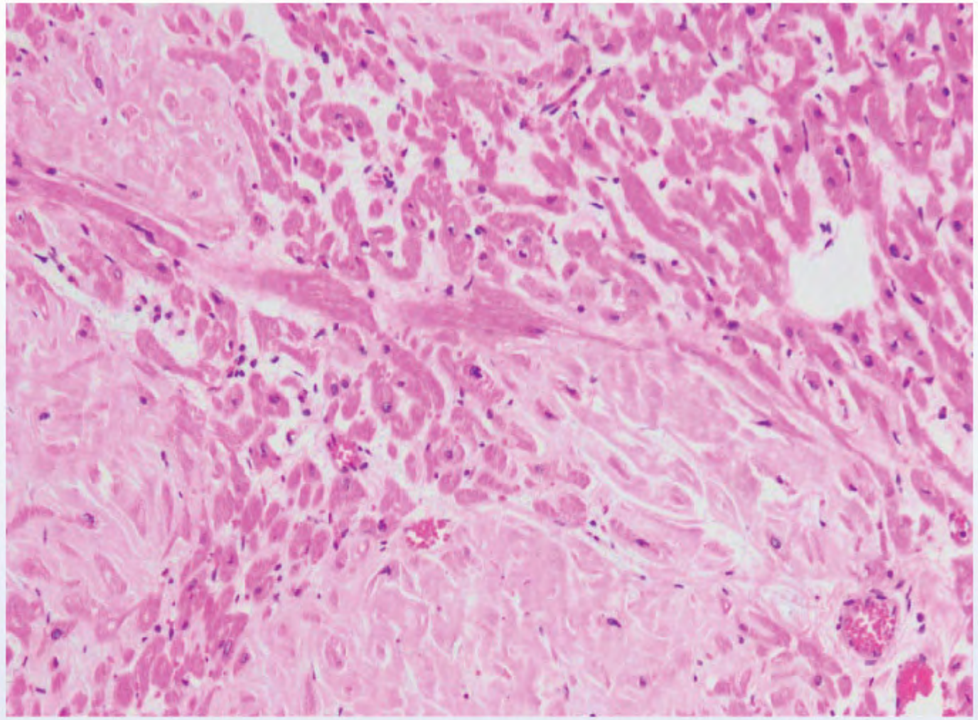

Which of the following is the most likely diagnosis?

- A. Cardiac myxoma (10%)
- ✗ B. Cardiac sarcoidosis (6%)
- C. Chagas disease (2%)
- D. Hemochromatosis (1%)
- ✓ E. Senile amyloidosis (78%)

Incorrect
Correct answer
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78%
Answered correctly

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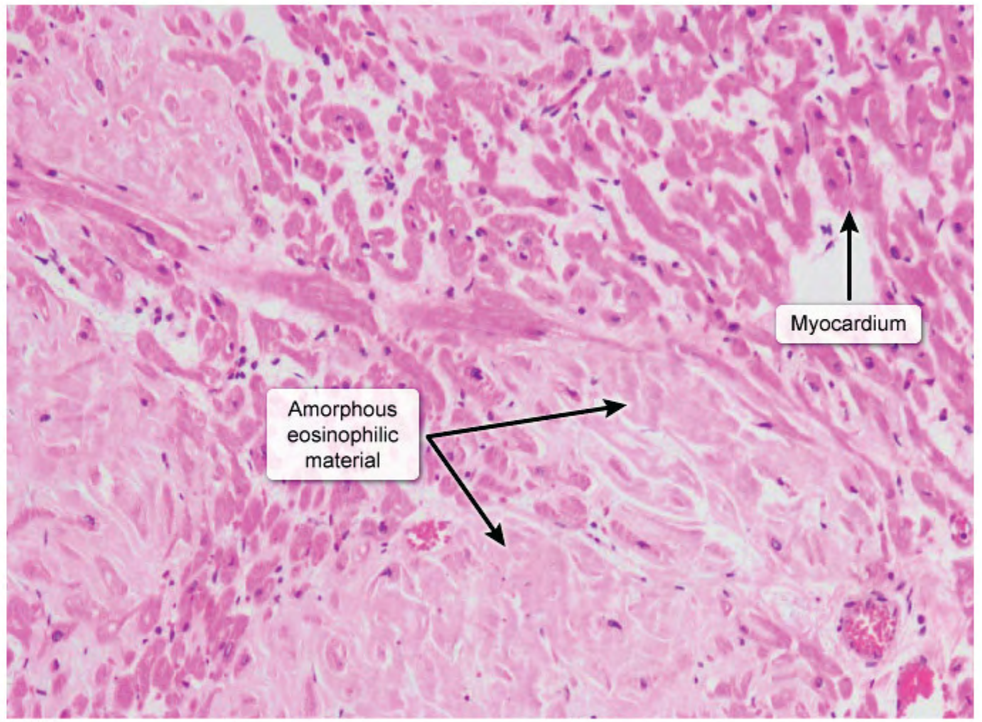
Cardiac amyloidosis



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ventricular hypertrophy, normal ejection fraction) are consistent with **diastolic heart failure** due to restrictive cardiomyopathy.

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This patient's clinical presentation (eg, progressive exertional dyspnea, edema, ascites, elevated jugular venous pressure with rapid 'y' descent, prominent S4) and echocardiogram findings (eg, left atrial enlargement, left ventricular hypertrophy, normal ejection fraction) are consistent with **diastolic heart failure** due to restrictive cardiomyopathy.

Restrictive cardiomyopathy can be idiopathic or caused by infiltrative diseases (eg, amyloidosis, sarcoidosis, hemochromatosis), radiation fibrosis, or endomyocardial fibrosis. Although most cases are associated with normal ventricular wall thickness, infiltrative conditions such as cardiac amyloidosis may lead to significant **ventricular hypertrophy**. Infiltrative diseases can also cause conduction system abnormalities (eg, bradycardia) that may ultimately require pacemaker implantation.

Cardiac amyloidosis results from abnormal extracellular deposition of insoluble proteins such as monoclonal light chains (AL amyloidosis), mutated transthyretin (familial ATTR amyloidosis), or wild-type transthyretin (senile systemic amyloidosis) in myocardial tissue. Endomyocardial biopsy typically reveals cross-sections of normal myocardial cells with other areas of myocardium infiltrated by an **amorphous and acellular pink material**. Congo red stain classically shows apple-green birefringence under polarized light microscopy.

(Choice A) Cardiac myxomas are the most common primary cardiac neoplasms, with approximately 80% originating in the left atrium. Left atrial myxomas can obstruct blood flow across the mitral valve, resulting in syncope and symptoms of heart failure. Endomyocardial biopsy typically reveals numerous bland stellate cells in a background of myxoid ground substance.

(Choice B) Cardiac sarcoidosis can present with restrictive or dilated cardiomyopathy along with conduction defects and atrial or ventricular arrhythmias; sudden death can also occur. Microscopic examination of the myocardium reveals the presence of noncaseating granulomas containing giant cells.

(Choice C) Chagas disease occurs secondary to *Trypanosoma cruzi* infection and is a common cause of myopericarditis and dilated cardiomyopathy in Latin America. Microscopic examination typically shows multiple parasitic protozoa within the myocardial fibers with a dense cellular infiltrate and myofiber destruction.

(Choice D) Hemochromatosis leads to excessive iron deposition in the myocardium and can cause conduction

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ultimately require pacemaker implantation.

Cardiac amyloidosis results from abnormal extracellular deposition of insoluble proteins such as monoclonal light chains (AL amyloidosis), mutated transthyretin (familial ATTR amyloidosis), or wild-type transthyretin (senile systemic amyloidosis) in myocardial tissue. Endomyocardial biopsy typically reveals cross-sections of normal myocardial cells with other areas of myocardium infiltrated by an **amorphous and acellular pink material**. Congo red stain classically shows apple-green birefringence under polarized light microscopy.

(Choice A) Cardiac myxomas are the most common primary cardiac neoplasms, with approximately 80% originating in the left atrium. Left atrial myxomas can obstruct blood flow across the mitral valve, resulting in syncope and symptoms of heart failure. Endomyocardial biopsy typically reveals numerous bland stellate cells in a background of myxoid ground substance.

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(Choice D) Hemochromatosis leads to excessive iron deposition in the myocardium and can cause conduction abnormalities and restrictive or dilated cardiomyopathy. Endomyocardial biopsy typically reveals excessive iron deposition in the myocardium, which can be confirmed with Prussian blue staining.

Educational objective:
Restrictive cardiomyopathy can be caused by infiltrative diseases (eg, amyloidosis, sarcoidosis, hemochromatosis) and often results in diastolic heart failure due to ventricular hypertrophy with impaired ventricular filling. Cardiac amyloidosis is characterized histologically by areas of myocardium infiltrated by an amorphous and acellular pink material (amyloid).

Pathology	Cardiovascular System	Amyloidosis
Subject	System	Topic

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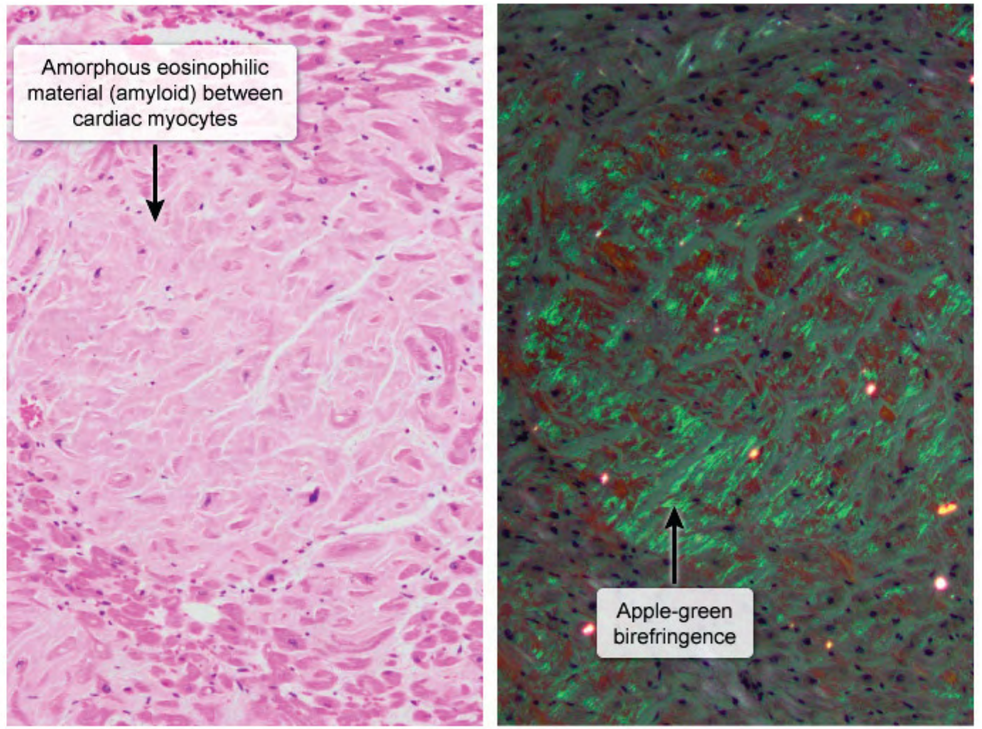
Amyloid



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Hematoxylin & eosin stain

Congo red stain under polarized light

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(Choice D) Hemochromatosis leads to excessive iron deposition in the myocardium and can cause conduction

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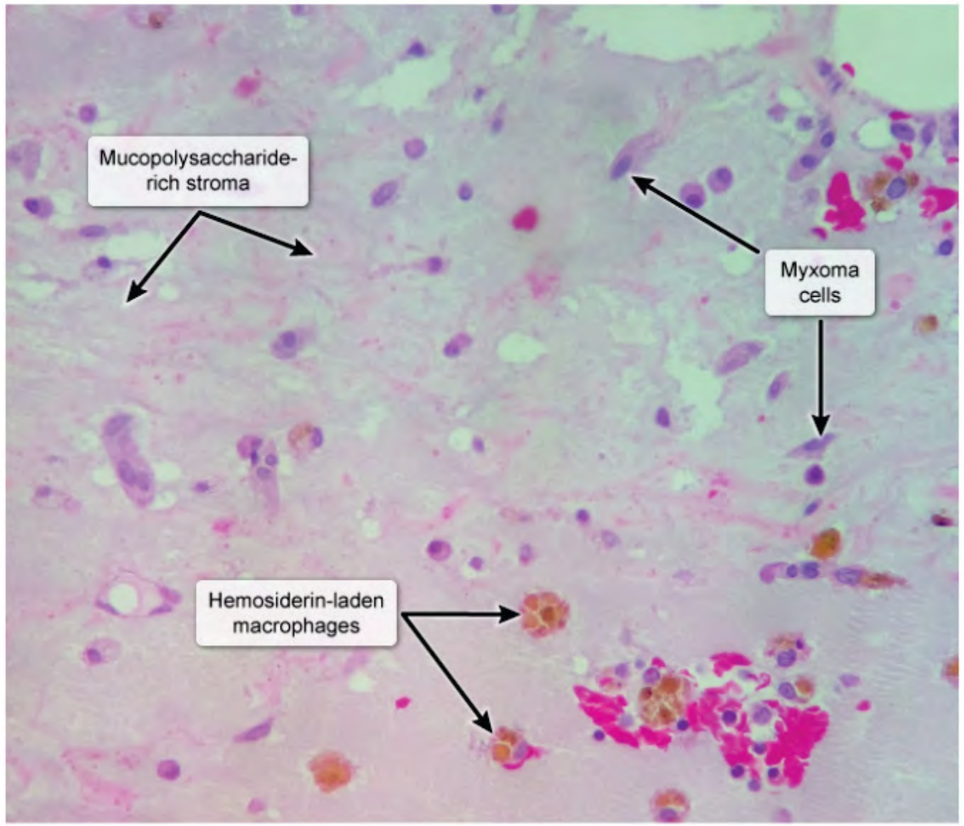
Cardiac myxoma



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(Choice D) Hemochromatosis leads to excessive iron deposition in the myocardium and can cause conduction

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ventricular hypertrophy, normal ejection fraction) are consistent with **diastolic heart failure** due to restrictive cardio

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Educational objective:

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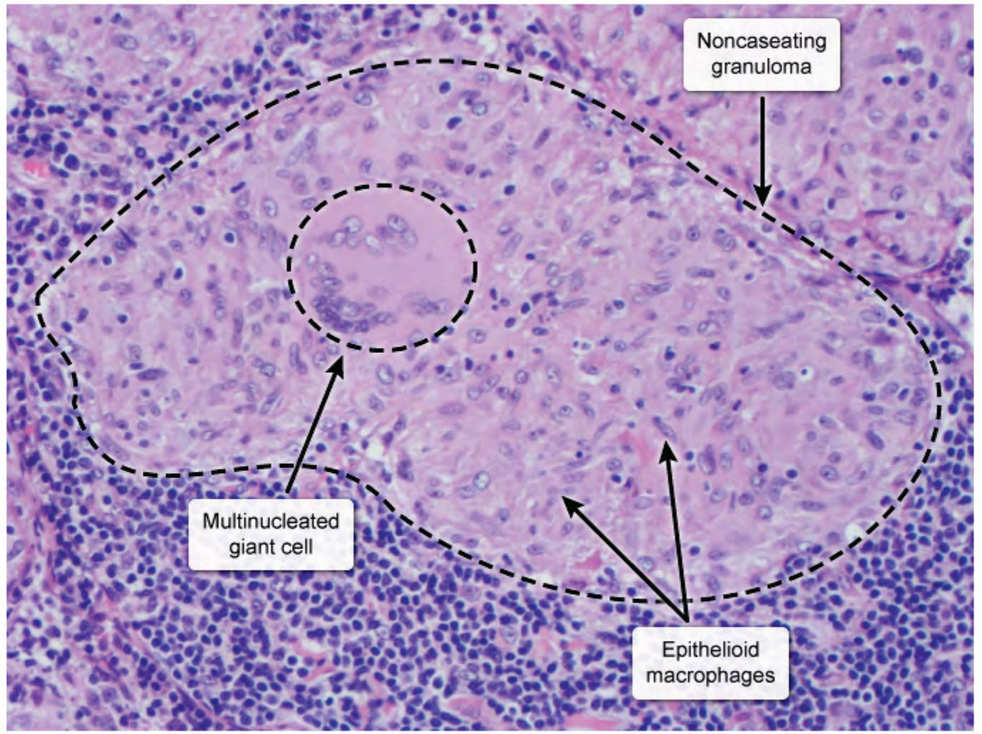
Sarcoidosis



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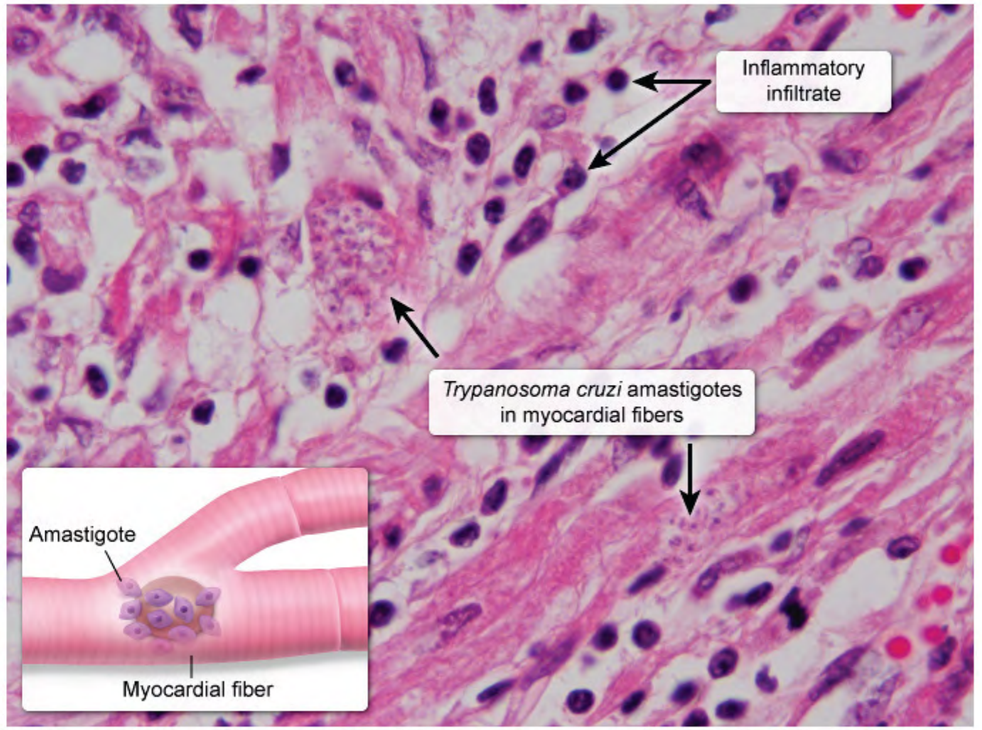
Chagas disease



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hemochromatosis), radiation fibrosis, or endomyocardial fibrosis. Although most cases are associated with normal

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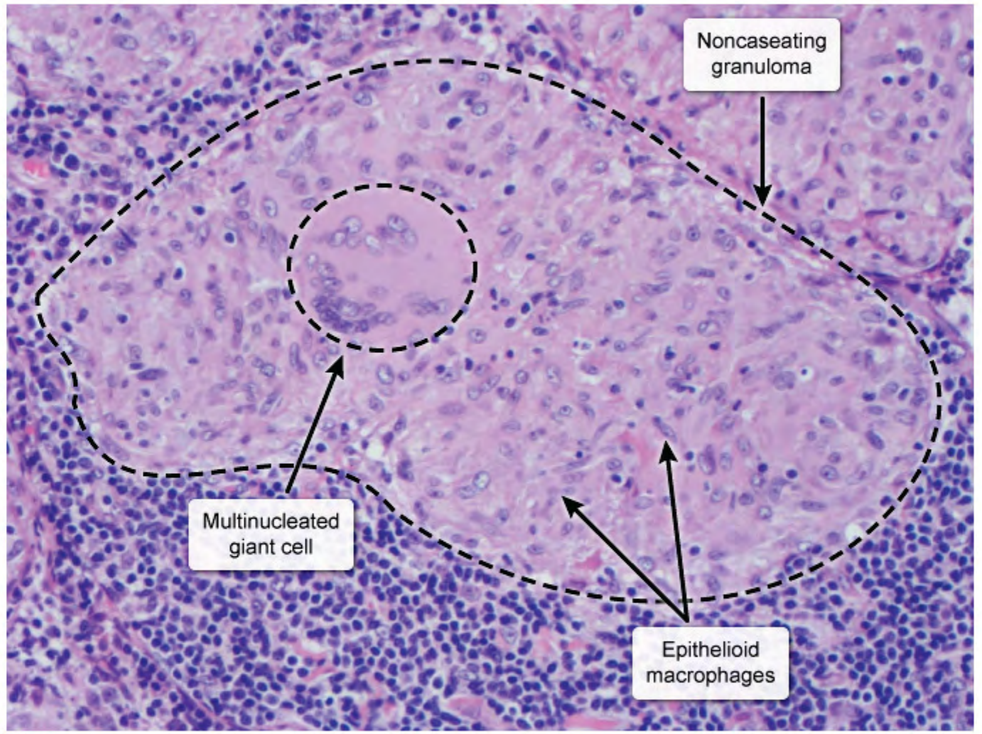

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ultimately require pacemaker implantation.

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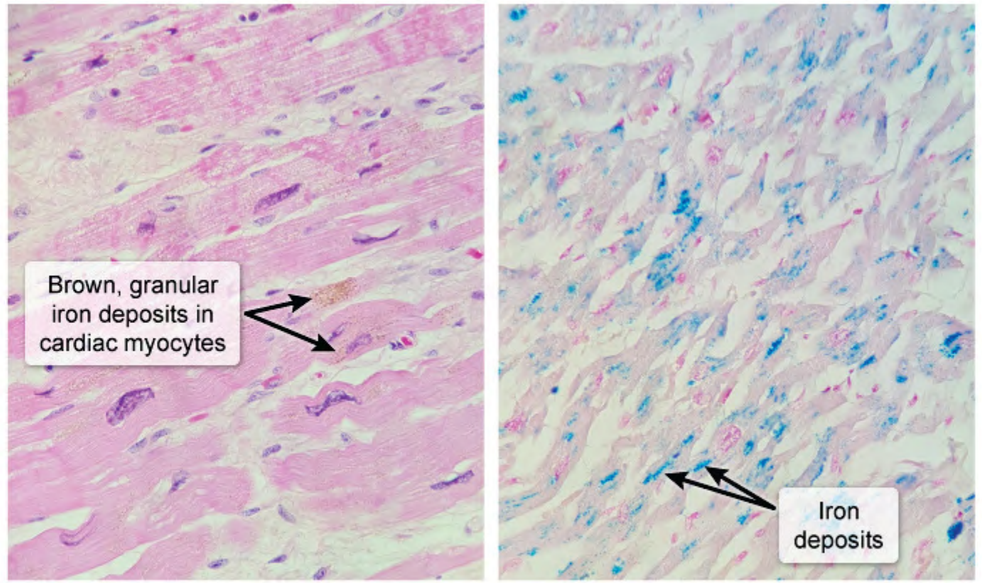
Hemochromatosis



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H&E stain

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Pathology
Subject

Cardiovascular System
System

Amyloidosis
Topic

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A 60-year-old man who was recently diagnosed with hypertension comes to the office for follow-up. He was treated with lisinopril but stopped a week ago due to a dry, nagging cough. Past medical history is notable for type 2 diabetes mellitus with moderately increased albuminuria but normal creatinine clearance. His other medications include metformin and rosuvastatin. The patient does not smoke or drink alcohol. His blood pressure is 150/92 mm Hg. BMI is 31 kg/m^2. Physical examination, including the heart and lungs, is unremarkable. Which of the following is the best treatment for this patient's hypertension?

- A. Diltiazem
- B. Hydralazine
- C. Metoprolol
- D. Ramipril
- E. Valsartan

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A 60-year-old man who was recently diagnosed with hypertension comes to the office for follow-up. He was treated with lisinopril but stopped a week ago due to a dry, nagging cough. Past medical history is notable for type 2 diabetes mellitus with moderately increased albuminuria but normal creatinine clearance. His other medications include metformin and rosuvastatin. The patient does not smoke or drink alcohol. His blood pressure is 150/92 mm Hg. BMI is 31 kg/m^2. Physical examination, including the heart and lungs, is unremarkable. Which of the following is the best treatment for this patient's hypertension?

- A. Diltiazem (2%)
- ✗ B. Hydralazine (3%)
- C. Metoprolol (5%)
- D. Ramipril (3%)
- ✓ E. Valsartan (85%)

Incorrect
Correct answer
E

85%
Answered correctly

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Time Spent

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Explanation

Diabetes causes impaired autoregulation of glomerular blood flow, leading to significant elevations of intraglomerular pressures and chronic glomerular injury. Patients with diabetes and hypertension are at especially high long-term risk for chronic kidney disease. Angiotensin-converting enzyme (ACE) inhibitors (eg, lisinopril) cause preferential dilation of the glomerular efferent arteriole, lowering intraglomerular pressures and reducing the risk of chronic glomerular injury.

In addition to converting angiotensin I to angiotensin II, ACE normally degrades **bradykinin** and substance P. Inhibition of ACE leads to elevated bradykinin and substance P levels and a nonproductive cough. **Angiotensin receptor blockers** (ARBs) have hemodynamic effects similar to those of ACE inhibitors but do not affect

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Diabetes causes impaired autoregulation of glomerular blood flow, leading to significant elevations of intraglomerular pressures and chronic glomerular injury. Patients with diabetes and hypertension are at especially high long-term risk for chronic kidney disease. Angiotensin-converting enzyme (ACE) inhibitors (eg, lisinopril) cause preferential dilation of the glomerular efferent arteriole, lowering intraglomerular pressures and reducing the risk of chronic glomerular injury.

In addition to converting angiotensin I to angiotensin II, ACE normally degrades **bradykinin** and substance P. Inhibition of ACE leads to elevated bradykinin and substance P levels and a nonproductive cough. **Angiotensin receptor blockers** (ARBs) have hemodynamic effects similar to those of ACE inhibitors but do not affect bradykinin levels and do not cause a cough. Replacing this patient's ACE inhibitor with an ARB (eg, losartan, valsartan) will eliminate the cough while still providing the same long-term renovascular benefits.

(Choices A, B, and C) Careful blood pressure control can help reduce the long-term risk of kidney injury, regardless of the agent used. However, none of these drugs are as effective as ACE inhibitors or ARBs for preventing the progression of chronic kidney disease.

(Choice D) Replacement of one ACE inhibitor with another carries a high likelihood of inducing the same nonproductive cough.

Educational objective:
Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) reduce the risk of chronic kidney disease in patients with hypertension and diabetes. ACE inhibitors raise levels of bradykinin and can cause a nonproductive cough, an effect not seen with ARBs.

References

- The RAAS in the pathogenesis and treatment of diabetic nephropathy.
- Angiotensin-converting enzyme inhibitor-induced cough: ACCP evidence-based clinical practice guidelines.

Pharmacology	Cardiovascular System	ACE inhibitors
Subject	System	Topic

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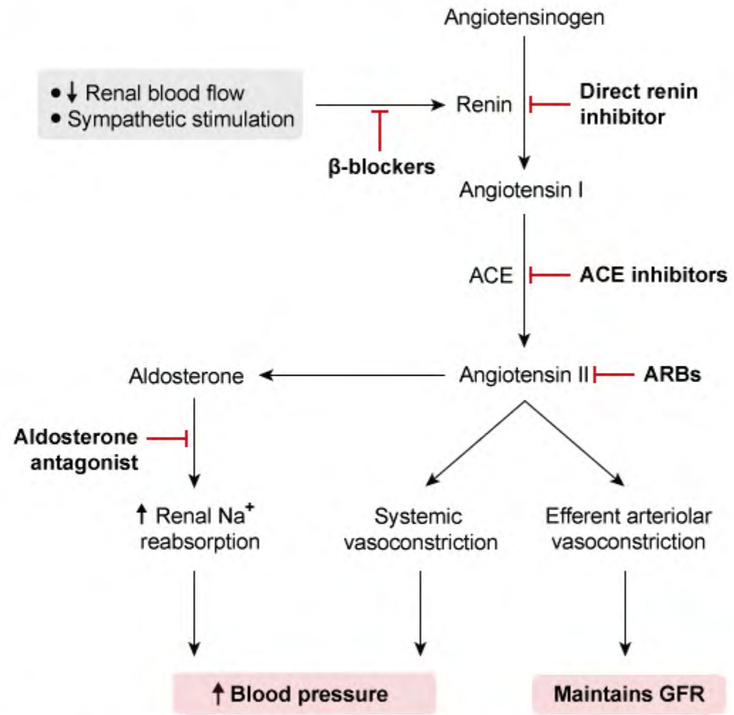
Exhibit Display

Renin-angiotensin-aldosterone system & antihypertensives



GFR = glomerular filtration rate.

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A 72-year-old man comes to the office due to palpitations and mild fatigue for the last 3 weeks. He has otherwise been feeling well. The patient walks 2 miles around his neighborhood twice per week with no chest pain or shortness of breath. Medical history is remarkable for benign prostatic hyperplasia, for which he takes terazosin. He drinks 1-2 cups of coffee every day and a glass of wine once or twice per month. Temperature is 37 C (98.6 F), blood pressure is 128/68 mm Hg, and pulse is 78/min and irregular. Physical examination reveals a 2/6 early-peaking systolic murmur at the right upper sternal border. Carotid pulses are prompt and full bilaterally. The lungs are clear to auscultation. There is no peripheral edema. ECG is shown in the exhibit. Which of the following is most likely the strongest risk factor for this patient's presentation?

- A. Advanced age
- B. Alcohol use
- C. Aortic stenosis
- D. Coffee consumption
- E. Terazosin use

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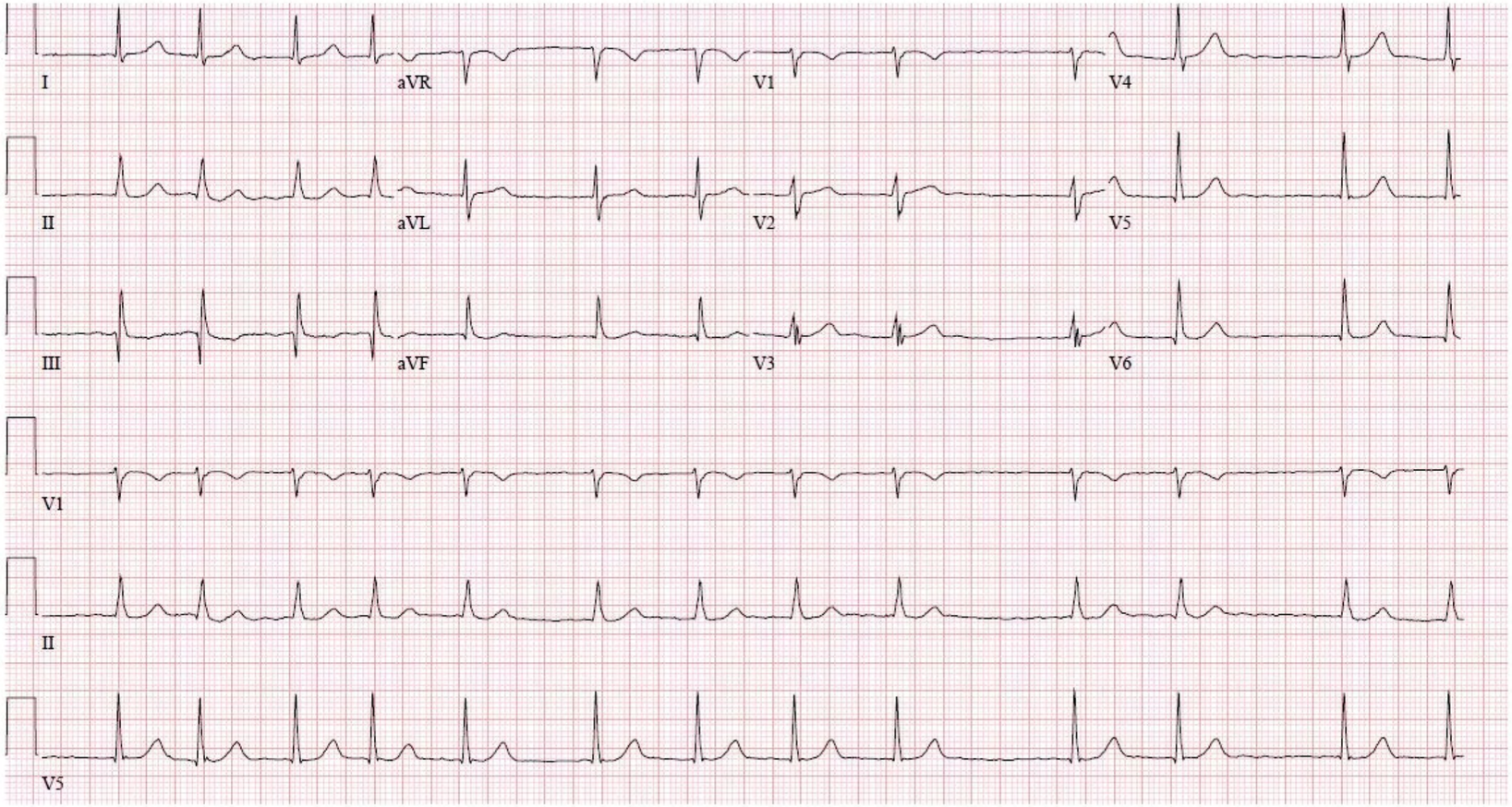

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- ✓ A. Advanced age (45%)
- ✗ B. Alcohol use (3%)
- C. Aortic stenosis (29%)
- D. Coffee consumption (12%)
- E. Terazosin use (8%)

Incorrect
Correct answer
A

45%
Answered correctly

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Time Spent

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Explanation

Atrial fibrillation	
Pathogenesis	• Atrial remodeling due to: ◦ Age-related myocardial changes ◦ Atrial enlargement from heart disease (eg, HTN, MS) • Foci of rapid electrical activity: ◦ Commonly originate in pulmonary veins

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Explanation

Atrial fibrillation	

Pathogenesis	• Atrial remodeling due to:
	  ◦ Age-related myocardial changes
	  ◦ Atrial enlargement from heart disease (eg, HTN, MS)
	• Foci of rapid electrical activity:
	  ◦ Commonly originate in pulmonary veins
	  ◦ Alcohol intake & ↑ sympathetic drive may contribute

Clinical presentation	• Asymptomatic or palpitations ± dyspnea, fatigue • Irregularly irregular pulse • May present with thromboembolic event (eg, stroke)
Diagnosis	• ECG shows variable R-R intervals & no P waves • Continuous ECG often needed for paroxysmal disease
HTN = hypertension; **MS** = mitral stenosis.	

This patient with several weeks of palpitations and fatigue has **atrial fibrillation**, which is recognized on ECG by an irregularly irregular rhythm (irregular R-R intervals) and an absence of organized P waves. Atrial fibrillation usually occurs in the setting of **atrial remodeling**, which describes changes to the atrial myocardium that result from both normal **aging** and underlying comorbidities that cause **atrial dilation** (eg, hypertension, heart failure, mitral valve disease). Coronary artery disease and its associated risk factors (eg, diabetes mellitus, smoking) are also associated with atrial fibrillation, although their contribution may be primarily driven by ischemia that leads to left ventricular dysfunction and consequent left atrial dilation.

Age, independent of comorbidities, is one of the **strongest risk factors** for atrial fibrillation. Atrial fibrillation is relatively rare in those age <40, but the prevalence is estimated at approximately 10% in those age >75 and approaches 20% in those age >85.

(Choices B and D) After remodeling has created a susceptible substrate, certain triggers (eg, increased

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(Choices B and D) After remodeling has created a susceptible substrate, certain triggers (eg, increased sympathetic drive) can initiate atrial fibrillation. Heavy alcohol use (eg, large binges or chronic ingestion of >1-2 drinks per day) is one such trigger, but occasional alcohol intake is unlikely to significantly contribute. High caffeine intake (eg, coffee) may somewhat encourage the triggering of atrial fibrillation (via increased sympathetic stimulation), but there is little evidence to support this association.

(Choice C) This patient's early peaking systolic murmur at the right upper sternal border with prompt and full carotid pulses is consistent with mild aortic stenosis. Both mitral regurgitation and especially mitral stenosis are strongly associated with atrial fibrillation as these valvular defects can cause left atrial dilation even in the absence of heart failure. However, aortic stenosis typically must be severe to cause left atrial dilation; mild aortic stenosis is not associated with atrial fibrillation.

(Choice E) Beta adrenergic agonists (eg, albuterol) may sometimes trigger atrial fibrillation by stimulating the generation of ectopic conduction foci. However, terazosin is an alpha blocker and does not significantly affect cardiac conduction.

Educational objective:
Age, independent of comorbidities, is one of the strongest risk factors for atrial fibrillation as age-related change to the atrial myocardium is one of the primary contributors to atrial remodeling. Left atrial dilation is the other primary

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usually occurs in the setting of **atrial remodeling**, which describes changes to the atrial myocardium that result from both normal **aging** and underlying comorbidities that cause **atrial dilation** (eg, hypertension, heart failure, mitral valve disease). Coronary artery disease and its associated risk factors (eg, diabetes mellitus, smoking) are also associated with atrial fibrillation, although their contribution may be primarily driven by ischemia that leads to left ventricular dysfunction and consequent left atrial dilation.

Age, independent of comorbidities, is one of the **strongest risk factors** for atrial fibrillation. Atrial fibrillation is relatively rare in those age <40, but the prevalence is estimated at approximately 10% in those age >75 and approaches 20% in those age >85.

(Choices B and D) After remodeling has created a susceptible substrate, certain triggers (eg, increased sympathetic drive) can initiate atrial fibrillation. Heavy alcohol use (eg, large binges or chronic ingestion of >1-2 drinks per day) is one such trigger, but occasional alcohol intake is unlikely to significantly contribute. High caffeine intake (eg, coffee) may somewhat encourage the triggering of atrial fibrillation (via increased sympathetic stimulation), but there is little evidence to support this association.

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Educational objective:
Age, independent of comorbidities, is one of the strongest risk factors for atrial fibrillation as age-related change to the atrial myocardium is one of the primary contributors to atrial remodeling. Left atrial dilation is the other primary contributor to atrial remodeling; therefore, comorbidities that cause left atrial dilation (eg, hypertension, heart failure, mitral valve disease) are also strongly associated with atrial fibrillation.

References

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Diagnosis	• ECG shows variable R-R intervals & no P waves

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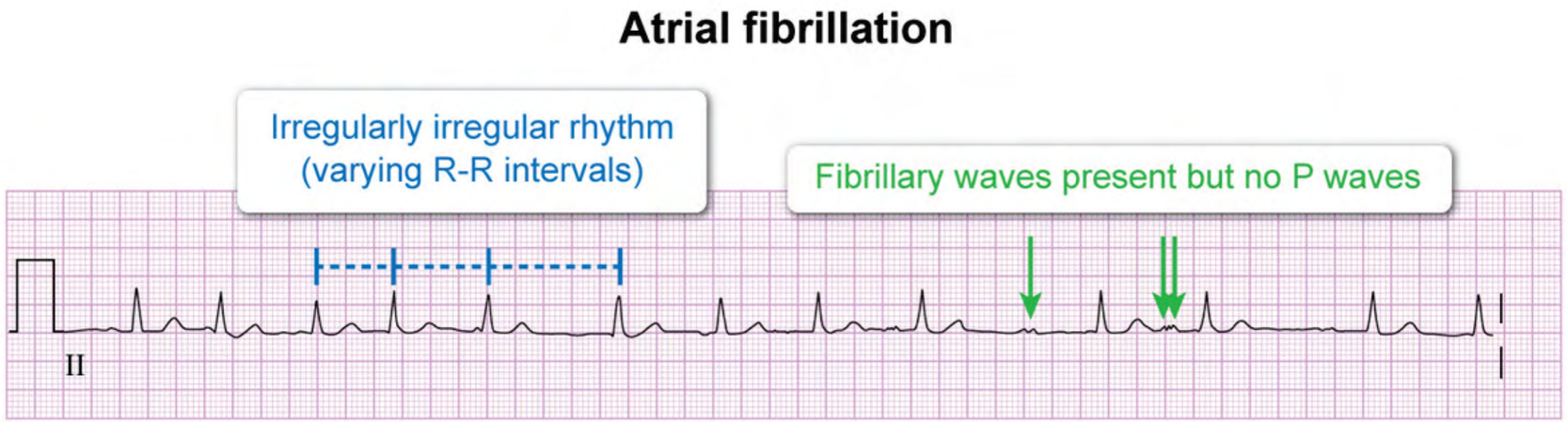

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A 72-year-old man comes to the office due to progressive shortness of breath. Over the past year, the patient has been unable to perform many of his usual outdoor activities, including afternoon walks and working in the yard. During the last several weeks, he has frequently become easily fatigued and short of breath, and at night he needs several pillows to sleep comfortably. He has also had ankle swelling. The patient has had no chest pain, syncope, abdominal pain, or cough. He is a lifetime nonsmoker. Bibasilar crackles are heard on physical examination. Echocardiography reveals no valvular disease and a nondilated left ventricle with an ejection fraction of 55%. Which of the following is most strongly associated with this patient's current condition?

- A. Glomerular hyperfiltration
- B. Increased left ventricular afterload
- C. Increased left ventricular compliance
- D. Left ventricular thrombus formation
- E. Right-to-left intracardiac shunting

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A 72-year-old man comes to the office due to progressive shortness of breath. Over the past year, the patient has been unable to perform many of his usual outdoor activities, including afternoon walks and working in the yard. During the last several weeks, he has frequently become easily fatigued and short of breath, and at night he needs several pillows to sleep comfortably. He has also had ankle swelling. The patient has had no chest pain, syncope, abdominal pain, or cough. He is a lifetime nonsmoker. Bibasilar crackles are heard on physical examination. Echocardiography reveals no valvular disease and a nondilated left ventricle with an ejection fraction of 55%. Which of the following is most strongly associated with this patient's current condition?

- ✗ A. Glomerular hyperfiltration (17%)
- ✓ B. Increased left ventricular afterload (60%)
- C. Increased left ventricular compliance (12%)
- D. Left ventricular thrombus formation (3%)
- E. Right-to-left intracardiac shunting (5%)

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Correct answer
B

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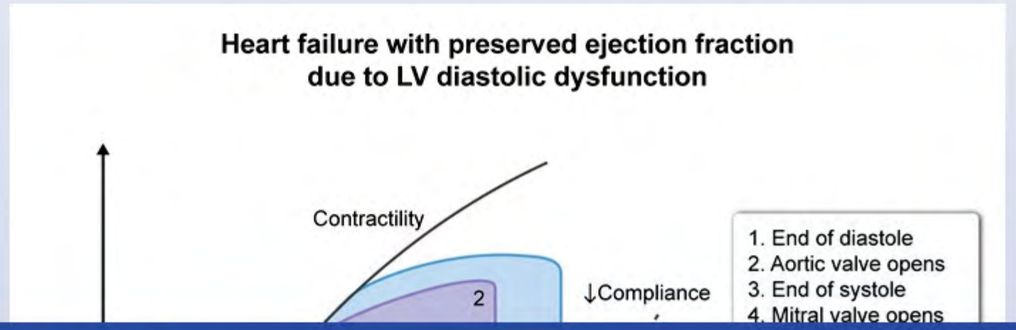
Explanation



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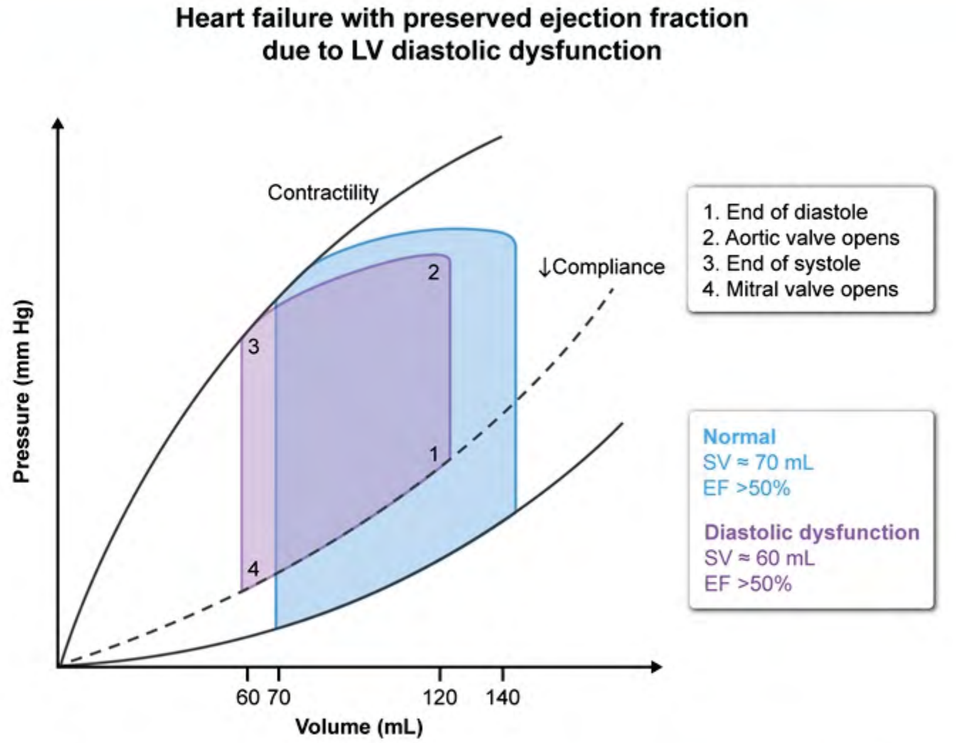

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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This patient has clinical signs and symptoms of decompensated heart failure (eg, dyspnea, fatigue, orthopnea, lower extremity edema, bibasilar crackles); however, echocardiography shows a preserved left ventricular (LV) ejection fraction (>50%) without chamber dilation. **Heart failure with preserved ejection fraction** (HFpEF) develops due to **diastolic dysfunction**, which frequently occurs in the setting of prolonged **systemic hypertension** (ie, increased afterload).

Over time, increased pressure load causes the left ventricle to undergo concentric hypertrophy with the addition of myocardial contractile fibers in parallel to increase contractile strength. The thickened LV walls become stiff and less compliant, leading to impaired diastolic relaxation and **increased LV end-diastolic pressure** (point 1). Gradually, the elevated pressure transmits back to the pulmonary veins and capillaries, resulting in pulmonary edema. There is also an eventual decline in LV end-diastolic volume (also indicated at point 1) leading to **reduced cardiac output** and further decompensation (stroke volume decreases despite preserved ejection fraction).

(Choice A) The decreased cardiac output in heart failure leads to reduced renal perfusion pressure and glomerular hypofiltration. To compensate, the renin-angiotensin-aldosterone system is activated, and angiotensin II constricts glomerular efferent arterioles in an effort to increase glomerular filtration rate back to normal.

(Choice C) Diastolic dysfunction involves a decrease in LV compliance. LV compliance may increase with eccentric hypertrophy, as occurs in response to LV volume overload (eg, dilated cardiomyopathy).

(Choice D) LV thrombus typically results from localized stagnation of blood as can occur with an LV aneurysm or in the setting of a severely dilated LV cavity with systolic dysfunction. It is not typically associated with diastolic dysfunction and HFpEF.

(Choice E) Atrial and ventricular septal defects are often asymptomatic but can eventually lead to right-to-left intracardiac shunting (Eisenmenger syndrome), typically characterized by cyanosis. This patient's orthopnea, lower extremity edema, and bibasilar crackles suggesting pulmonary edema are more consistent with heart failure.

Educational objective:
Diastolic dysfunction is a common cause of heart failure with preserved ejection fraction. It typically results from concentric left ventricular hypertrophy in response to prolonged systemic hypertension (ie, increased afterload).

References

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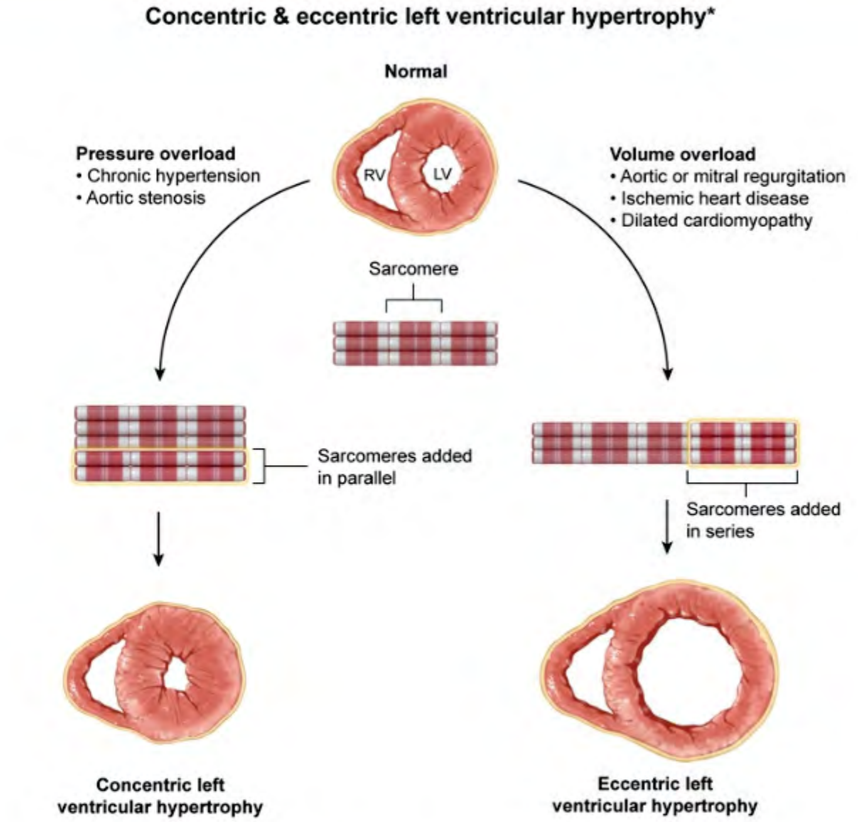

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This patient has clinical signs and symptoms of decompensated heart failure (eg, dyspnea, fatigue, orthopnea, lower
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Diastolic dysfunction is a common cause of heart failure with preserved ejection fraction. It typically results from concentric left ventricular hypertrophy in response to prolonged systemic hypertension (ie, increased afterload).

References

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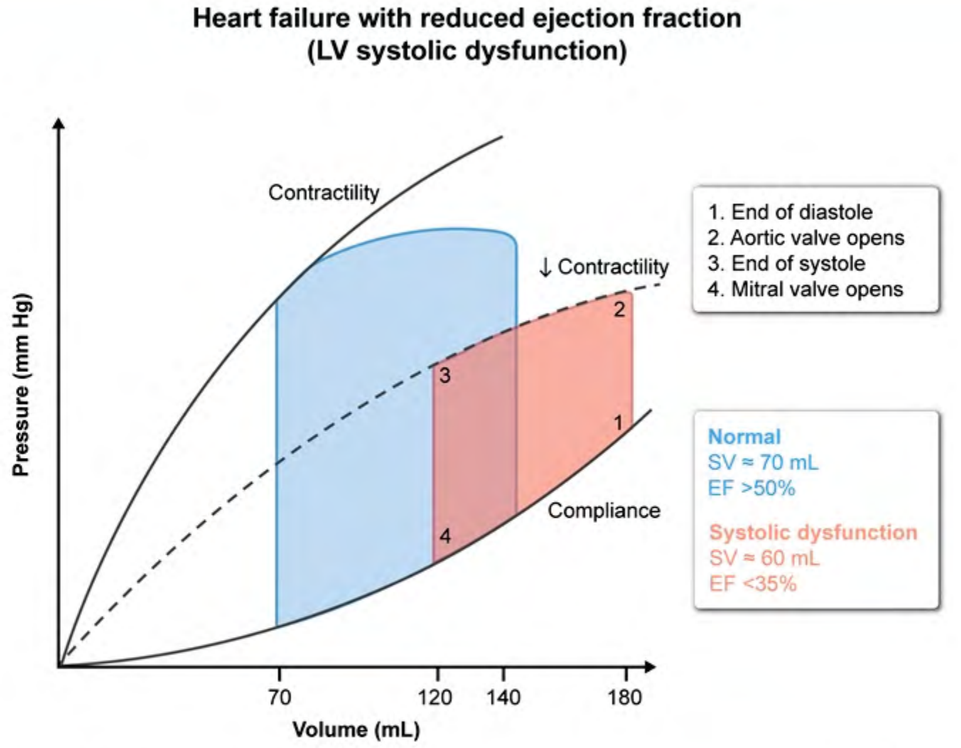

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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A 17-year-old girl is brought to the emergency department after an episode of syncope. The patient was sitting on the couch watching television when she suddenly passed out. She had no symptoms prior to the event and regained consciousness spontaneously after approximately 2 minutes. Her family noticed a brief twitching while the patient was unconscious, but she had no tongue biting or bowel or bladder incontinence. The patient is taking an antimicrobial agent for a recent infection but has no other chronic medical conditions. Her uncle died suddenly 2 years ago. A detailed evaluation reveals a decrease-in-function mutation in the *KCNH2* gene, which codes for a voltage-gated potassium channel that is active during the repolarization phase of cardiomyocyte action potential. The patient's medication was causing further impairment of the ion channel function, leading to the incident of syncope. This patient was most likely taking which of the following medications?

- A. Acyclovir
- B. Amoxicillin
- C. Cephalexin
- D. Doxycycline
- E. Erythromycin
- F. Terbinafine

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- ✗ A. Acyclovir (4%)
- B. Amoxicillin (3%)
- C. Cephalexin (3%)
- D. Doxycycline (15%)
- ✓ E. Erythromycin (63%)
- F. Terbinafine (9%)

Incorrect
Correct answer
E

63%
Answered correctly

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Explanation

Causes of QT-interval prolongation	
	• Medications ◦ Macrolides & fluoroquinolones

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Causes of QT-interval prolongation	
Acquired	• Medications ◦ Macrolides & fluoroquinolones ◦ Antiemetics (eg, ondansetron) ◦ Azoles (eg, fluconazole) ◦ Antipsychotics, TCAs & SSRIs ◦ Some opioids (eg, methadone, oxycodone) ◦ Class Ia antiarrhythmics (eg, quinidine) ◦ Class III antiarrhythmics (eg, dofetilide, sotalol) • Electrolyte abnormalities ◦ ↓ Magnesium, ↓ potassium, ↓ calcium
Congenital	• Romano-Ward syndrome (autosomal dominant) • Jervell & Lange-Nielsen syndrome (autosomal recessive)*
*Associated with sensorineural hearing loss. **SSRI** = selective serotonin reuptake inhibitor; **TCA** = tricyclic antidepressant.	

A decrease-in-function mutation affecting the *KCNH2* gene is one of the most common causes of **congenital long QT syndrome** (LQTS); it can be inherited in autosomal dominant (Romano-Ward syndrome) or autosomal recessive (Jervell and Lange-Nielsen syndrome) fashion. The mutation **impairs the function** of the **voltage-gated potassium channels** controlling the delayed rectifier potassium current that allows **repolarization** of the cardiomyocyte action potential. Delayed repolarization manifests as a prolonged QT interval on ECG and predisposes to the development of **torsades de pointes** (TdP) (a form of polymorphic ventricular tachycardia), which can cause syncope or sudden cardiac death.

Most patients with underlying congenital LQTS do not develop TdP unless exposed to an external influence. **Medications** are the most common cause of **acquired LQTS** and the most important exacerbating factor in patients with congenital LQTS. Most medications that prolong the QT interval do so by delaying the repolarizing

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SSRI = selective serotonin reuptake inhibitor; **TCA** = tricyclic antidepressant.

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Other medications that prolong the QT interval include fluoroquinolone antibiotics (eg, ciprofloxacin, moxifloxacin) and many antipsychotics (eg, haloperidol, risperidone) and antiemetics (eg, ondansetron, metoclopramide). The coadministration of multiple QT-prolonging medications may precipitate TdP in patients without congenital LQTS.

(Choice A) Some antiviral medications may prolong the QT interval—including protease inhibitors used in the treatment of HIV (eg, saquinavir, atazanavir)—however, acyclovir does not.

(Choices B, C, and D) Penicillins (eg, amoxicillin), cephalosporins (eg, cephalexin), and tetracyclines (eg, doxycycline) have not been shown to prolong the QT interval.

(Choice F) Azoles (eg, fluconazole, voriconazole) prolong the QT interval; however, most other antifungal medications, including terbinafine (a squalene epoxidase inhibitor), do not.

Educational objective:
Congenital long QT syndrome is commonly caused by a mutation that slows the delayed rectifier potassium current that repolarizes the cardiomyocyte action potential. Certain medications (eg, macrolide antibiotics, antipsychotics,

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recessive (Jervell and Lange-Nielsen syndrome) fashion. The mutation **impairs the function** of the **voltage-gated potassium channels** controlling the delayed rectifier potassium current that allows **repolarization** of the cardiomyocyte action potential. Delayed repolarization manifests as a prolonged QT interval on ECG and predisposes to the development of **torsades de pointes** (TdP) (a form of polymorphic ventricular tachycardia), which can cause syncope or sudden cardiac death.

Most patients with underlying congenital LQTS do not develop TdP unless exposed to an external influence. **Medications** are the most common cause of **acquired LQTS** and the most important exacerbating factor in patients with congenital LQTS. Most medications that prolong the QT interval do so by delaying the repolarizing potassium current via blockade of the same voltage-gated potassium channels affected by the *KCNH2* gene mutation. **Macrolide antibiotics** (eg, erythromycin, azithromycin) prolong the QT interval and increase the risk of TdP, especially in patients with underlying congenital LQTS.

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Educational objective:
Congenital long QT syndrome is commonly caused by a mutation that slows the delayed rectifier potassium current that repolarizes the cardiomyocyte action potential. Certain medications (eg, macrolide antibiotics, antipsychotics, antiemetics) also slow the potassium repolarization current and prolong the QT interval. Excessive QT prolongation can trigger serious cardiac arrhythmia (ie, torsades de pointes), resulting in syncope or sudden cardiac death.

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- Class III antiarrhythmics (eg, dofetilide, sotalol)

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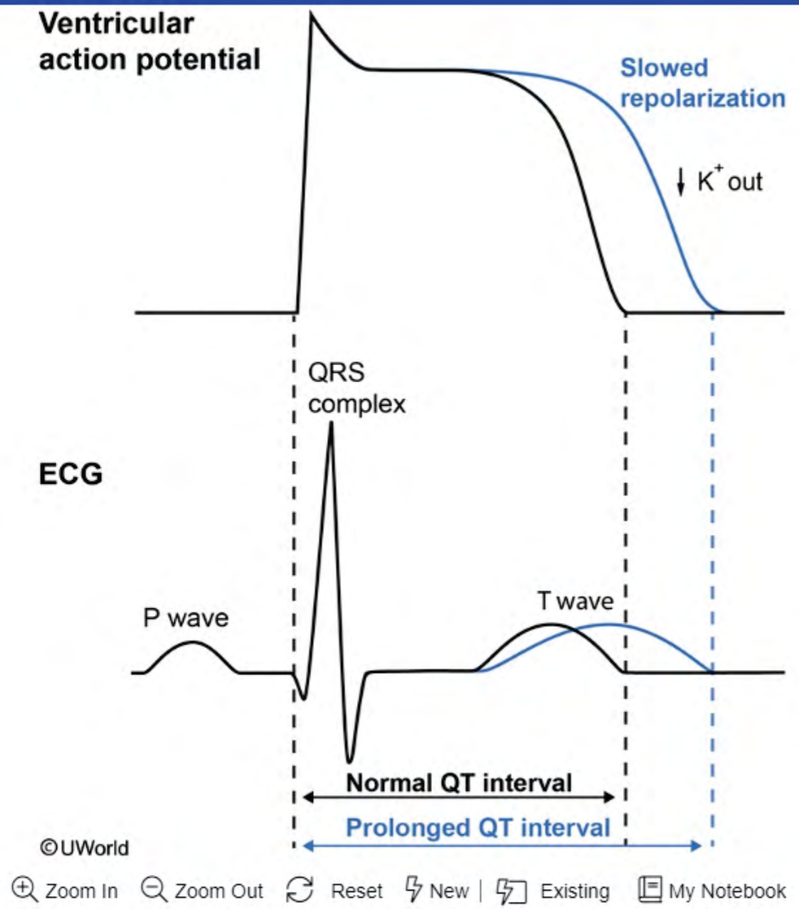

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- Class III antiarrhythmics (eg, dofetilide, sotalol)

Electrolyte abnormalities

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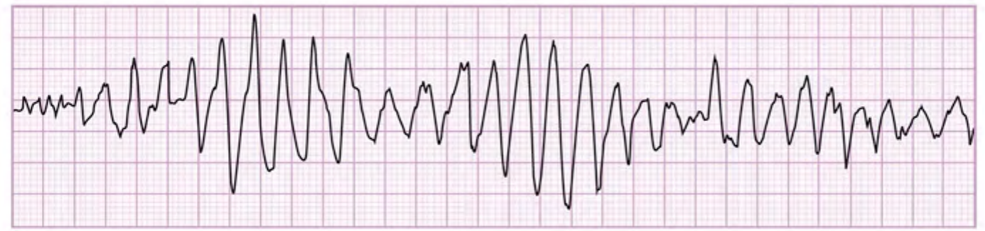

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A 35-year-old primigravid woman comes to the clinic during the second trimester. The patient reports that she has had some tiredness, which she attributes to pregnancy. She takes 1 nap during the day. She has no chest pain, cough, or lower extremity swelling. As a child, the patient was told that she has a murmur but does not know any details and says that she has felt well until recently. On physical examination, a 3/6 midsystolic murmur is heard at the left upper sternal border and S2 is widely split. Which of the following is the most likely diagnosis?

- A. Benign flow murmur
- B. Bicuspid aortic valve
- C. Hypertrophic cardiomyopathy
- D. Pulmonic stenosis
- E. Ventricular septal defect

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- A. Benign flow murmur (20%)
- ✕ B. Bicuspid aortic valve (7%)
- C. Hypertrophic cardiomyopathy (1%)
- ✓ D. Pulmonic stenosis (59%)
- E. Ventricular septal defect (10%)

Incorrect
Correct answer
D

59%
Answered correctly

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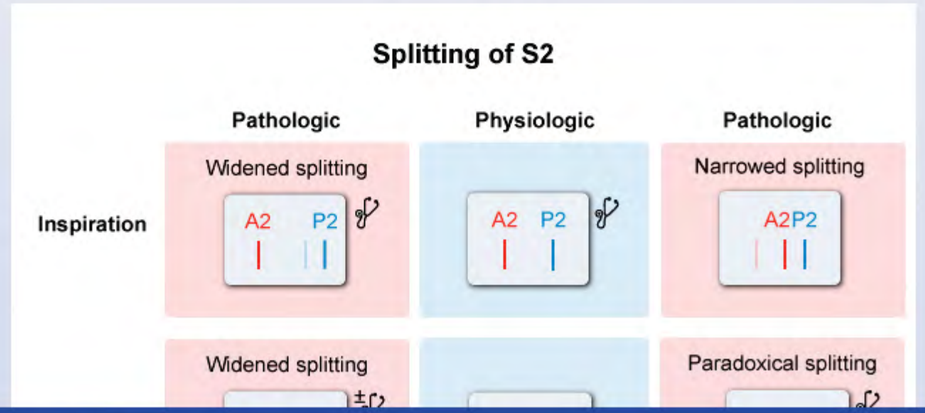
Explanation



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A 35-year-old primigravid woman comes to the clinic during the second trimester. The patient reports that she has had sc
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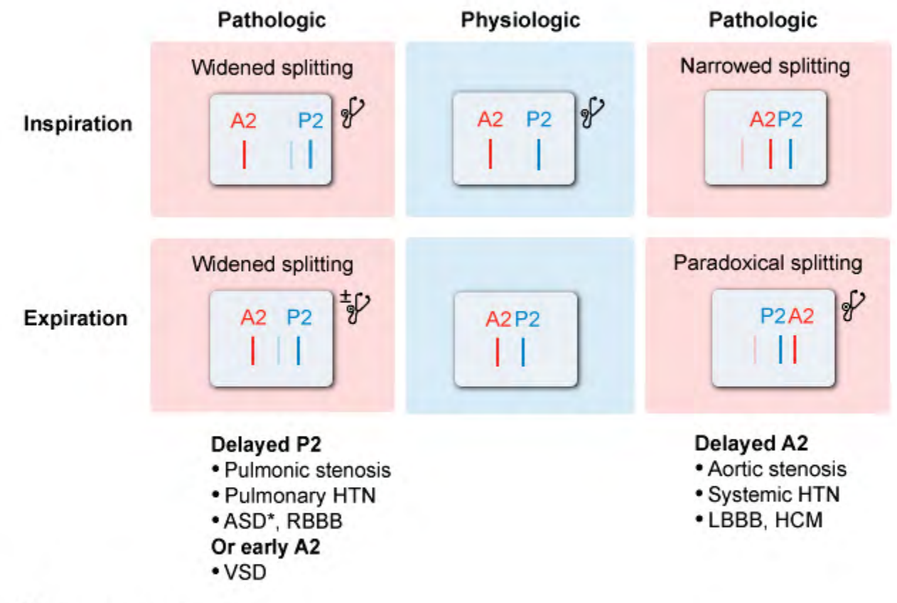
Splitting of S2



Delayed P2
- Pulmonic stenosis
- Pulmonary HTN
- ASD*, RBBB

Or early A2
- VSD

Delayed A2
- Aortic stenosis
- Systemic HTN
- LBBB, HCM

= Split is audibly discernible.

*Widened splitting is fixed throughout respiratory cycle.

ASD = atrial septal defect; **HCM** = hypertrophic cardiomyopathy; **HTN** = hypertension; **LBBB** = left bundle-branch block; **RBBB** = right bundle-branch block; **VSD** = ventricular septal defect.

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Widened splitting
Paradoxical splitting

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Widened splitting is fixed throughout respiratory cycle.
ASD = atrial septal defect; **HCM** = hypertrophic cardiomyopathy; **HTN** = hypertension;
LBBB = left bundle-branch block; **RBBB** = right bundle-branch block; **VSD** = ventricular septal defect.
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This patient likely has **pulmonic valve stenosis** (PS), which most commonly occurs as an isolated **congenital defect** and rarely occurs as an acquired defect (eg, rheumatic fever, carcinoid syndrome). Severe PS is typically diagnosed early in life due to development of right-sided heart failure, but patients with relatively mild PS often do not present with symptoms (eg, dyspnea, fatigue) until adulthood. The physiologic changes of pregnancy (eg, increased plasma volume) can unmask underlying PS.

Cardiac auscultation in PS reveals a pulmonic **ejection click** followed by a harsh **crescendo-decrescendo systolic murmur** best heard at the left upper sternal border. The stenosis causes the pulmonic valve to close later than usual as it takes longer for the right ventricle to push blood through the narrowed valve. This results in **widened splitting** of the aortic and pulmonic components of S2. Inspiration draws additional blood into the right side of heart, which increases the intensity of the murmur and further widens the splitting of S2.

Pulmonary hypertension is another cause of widened splitting of S2 because more time is required for the right ventricle to overcome pulmonary arterial pressure (ie, the pulmonic valve opens later and closes later).

(Choice A) A benign flow murmur can occur in patients with high cardiac output states (eg, pregnancy) due to increased flow across the aortic and pulmonic valves; however, the murmur is typically low grade (2/6 or less) and abnormal splitting of S2 is not present.

(Choice B) A bicuspid aortic valve is the most common congenital heart defect seen in adults and can lead to aortic stenosis in relatively young patients (eg, those in their 50s). However, aortic stenosis causes delayed closure of the aortic valve, leading to narrowed or paradoxical splitting of S2.

(Choice C) Hypertrophic cardiomyopathy can cause a systolic murmur that mimics that of aortic stenosis and is best heard at the mid-left sternal border. Aortic valve closure may be delayed due to prolonged ejection of blood through the left ventricular outflow tract.

(Choice E) A small- or moderate-sized ventricular septal defect may present in adulthood with symptoms of heart failure. There is typically widened splitting of S2 due to early closure of the aortic valve (less blood is ejected into the aorta because some blood is ejected into the right ventricle). However, a holosystolic murmur best heard at the left lower sternal border is expected.

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Educational objective:
Pulmonic valve stenosis causes a crescendo-decrescendo systolic murmur (best heard at the left upper sternal border) and delays closure of the pulmonic valve, resulting in widened splitting of S2. Inspiration increases blood flow to the right side of the heart, causing increased intensity of the murmur and even later closure of the pulmonic valve.

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Widened splitting is fixed throughout respiratory cycle.
ASD = atrial septal defect; **HCM** = hypertrophic cardiomyopathy; **HTN** = hypertension; **LBBB** = left bundle-branch block; **RBBB** = right bundle-branch block; **VSD** = ventricular septal defect.

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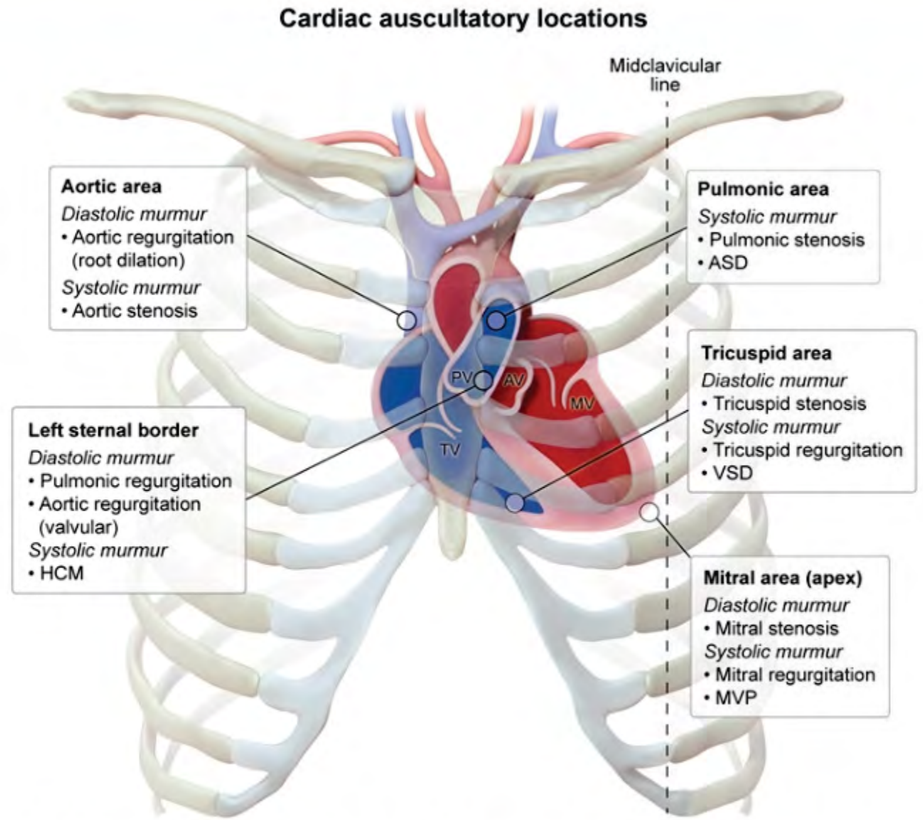

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Cardiac murmurs

Systolic	Aortic stenosis*	S1 A2 P2 S1	Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*	S1 S2 S1	Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy	S1 S2 S1	Crescendo-decrescendo at mid–left sternal border
	Mitral valve prolapse	S1 Click S2 S1	Midsystolic click followed by late systolic murmur
	Atrial septal defect	S1 A2 P2 S1	Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect	S1 S2 S1	Holosystolic at left sternal border
Diastolic	Aortic regurgitation*	S1 S2 S1	Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*	S1 S2 OS S1	Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus	S1 S2 S1	Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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In an experiment, 3 L of isotonic saline are infused intravenously into a healthy volunteer after multiple baseline physiologic parameters are recorded. Serial blood pressure measurements show an increase in the systolic and diastolic blood pressure. Ultrasonography reveals an increase in the left ventricular volume and engorgement of the inferior vena cava. Blood levels of a peptide hormone have also increased compared to baseline. This hormone is most likely to increase which of the following in this test subject?

- A. Acid content of the urine
- B. Glomerular filtration rate
- C. Renal sodium reabsorption
- D. Renal water reabsorption
- E. Vascular resistance

Submit

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- A. Acid content of the urine (3%)
- ✓ B. Glomerular filtration rate (73%)
- C. Renal sodium reabsorption (9%)
- ✗ D. Renal water reabsorption (5%)
- E. Vascular resistance (7%)

Incorrect
Correct answer
B

73%
Answered correctly

03 secs
Time Spent

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Explanation

Renal effects of natriuretic peptides

↑ Intracardiac filling pressures

Release of ANP (atria) & BNP (ventricles)

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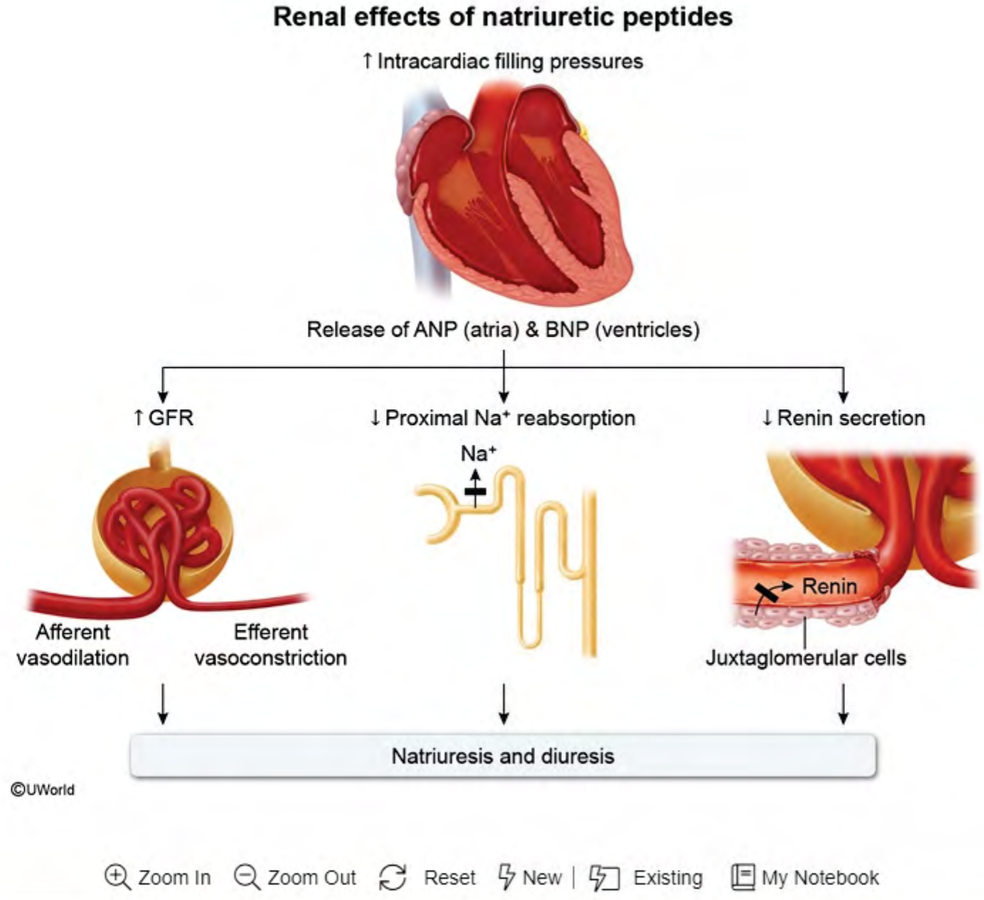

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Release of ANP (atria) & BNP (ventricles)

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Natriuresis and diuresis

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Intravenous infusion of isotonic saline leads to **intravascular volume expansion** with an increase in intracardiac volume and filling pressures. The resultant increase in myocardial wall stretch leads to the release of endogenous natriuretic peptide hormones, including **atrial natriuretic peptide** (ANP) and **brain natriuretic peptide** (BNP) from the atria and ventricles, respectively.

Both ANP and BNP activate guanylate cyclase, which increases intracellular cyclic GMP, and leads to downstream physiologic effects in various tissues. In the kidney, natriuretic peptides promote afferent glomerular arteriolar vasodilation and efferent arteriolar constriction, causing **increased glomerular filtration rate**; this leads to increased **natriuresis** (sodium excretion) and **diuresis** (fluid excretion). Natriuretic peptides also directly inhibit proximal tubular sodium reabsorption and renin secretion. Decreased renin secretion results in reduced angiotensin II and aldosterone levels, further promoting natriuresis and diuresis.

(Choice A) An increase in aldosterone levels causes sodium and water retention, hypokalemia, and metabolic alkalosis due to increased urinary hydrogen excretion. Infusion of isotonic saline inhibits aldosterone release and causes a decrease in urinary hydrogen excretion and acid content of the urine.

(Choice C) Activation of the renin-angiotensin-aldosterone system (eg, hypotension, hypovolemia, increased sympathetic activity) leads to an increase in renin secretion, which in turn leads to increased production of angiotensin II and aldosterone. Angiotensin II increases sodium reabsorption in the proximal tubules and stimulates aldosterone secretion from the adrenal cortex, which in turn promotes sodium and water reabsorption in the distal tubule. An increase in intravascular volume by isotonic saline inhibits renin release and causes a decrease in sodium and water reabsorption.

(Choice D) Infusion of isotonic saline inhibits the release of antidiuretic hormone (vasopressin), reducing renal water reabsorption in the collecting duct.

(Choice E) Angiotensin II causes arteriolar vasoconstriction via direct actions on AT1 receptors on the cell membrane. Infusion of isotonic saline decreases renin release with a reduction in angiotensin II production, resulting in decreased systemic vascular resistance.

Educational objective:

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Both ANP and BNP activate guanylate cyclase, which increases intracellular cyclic GMP, and leads to downstream physiologic effects in various tissues. In the kidney, natriuretic peptides promote afferent glomerular arteriolar vasodilation and efferent arteriolar constriction, causing **increased glomerular filtration rate**; this leads to increased **natriuresis** (sodium excretion) and **diuresis** (fluid excretion). Natriuretic peptides also directly inhibit proximal tubular sodium reabsorption and renin secretion. Decreased renin secretion results in reduced angiotensin II and aldosterone levels, further promoting natriuresis and diuresis.

(Choice A) An increase in aldosterone levels causes sodium and water retention, hypokalemia, and metabolic alkalosis due to increased urinary hydrogen excretion. Infusion of isotonic saline inhibits aldosterone release and causes a decrease in urinary hydrogen excretion and acid content of the urine.

(Choice C) Activation of the renin-angiotensin-aldosterone system (eg, hypotension, hypovolemia, increased sympathetic activity) leads to an increase in renin secretion, which in turn leads to increased production of angiotensin II and aldosterone. Angiotensin II increases sodium reabsorption in the proximal tubules and stimulates aldosterone secretion from the adrenal cortex, which in turn promotes sodium and water reabsorption in the distal tubule. An increase in intravascular volume by isotonic saline inhibits renin release and causes a decrease in sodium and water reabsorption.

(Choice D) Infusion of isotonic saline inhibits the release of antidiuretic hormone (vasopressin), reducing renal water reabsorption in the collecting duct.

(Choice E) Angiotensin II causes arteriolar vasoconstriction via direct actions on AT1 receptors on the cell membrane. Infusion of isotonic saline decreases renin release with a reduction in angiotensin II production, resulting in decreased systemic vascular resistance.

Educational objective:
Atrial natriuretic peptide and brain natriuretic peptide are released from the atria and ventricles, respectively, in response to myocardial wall stretch due to intravascular volume expansion. These endogenous hormones promote increased glomerular filtration rate, natriuresis, and diuresis.

References

- Natriuretic peptides: their structures, receptors, physiologic functions and therapeutic applications.

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volume and filling pressures. The resultant increase in myocardial wall stretch leads to the release of endogenous natriuretic peptide hormones, including **atrial natriuretic peptide** (ANP) and **brain natriuretic peptide** (BNP) from the atr

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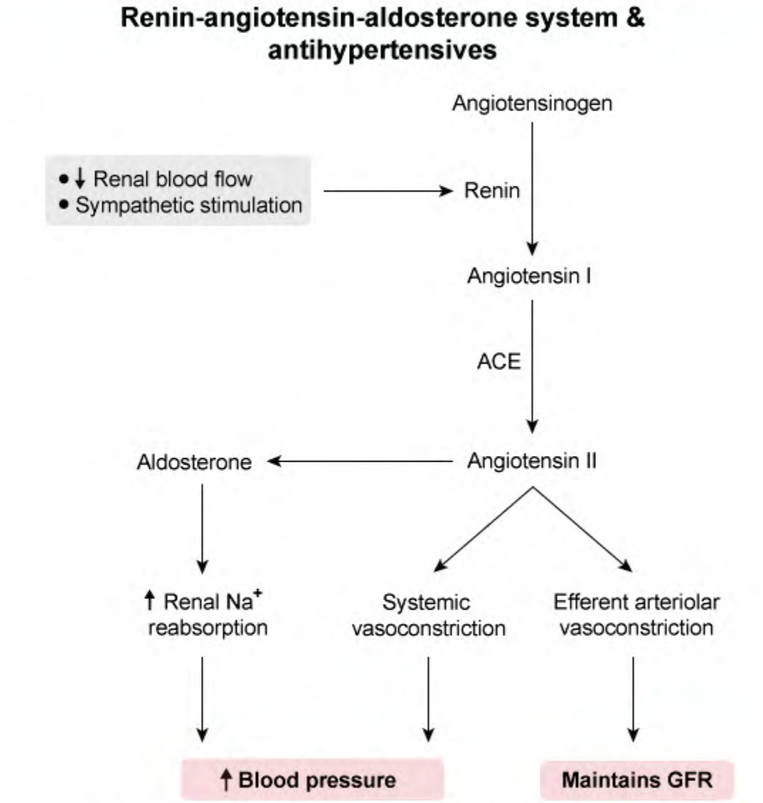
Exhibit Display



GFR = glomerular filtration rate.
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A 35-year-old previously healthy woman comes to the hospital due to increasing shortness of breath and cough. Over the past few months, she has had arthralgias, fatigue, low-grade fever, and weight loss. Laboratory results are as follows:

Complete blood count		
	Hemoglobin	10.4 g/dL
	Leukocytes	8,000/mm^3
Serum chemistry		
	Blood urea nitrogen	28 mg/dL
	Creatinine	2.7 mg/dL
Urinalysis		
	Nitrites	negative
	Bacteria	none
	Red blood cells	20-30/hpf

CT scan of chest is shown in the exhibit. Histologic examination of the lung would most likely reveal which of the following findings?

- A. Fibrinoid degeneration of the vessel wall with luminal narrowing
- B. Necrotizing arteritis and focal collections of epithelioid histiocytes
- C. Necrotizing granuloma with a predominance of eosinophils
- D. Proliferation of irregularly shaped glands and pleomorphic cells

Submit

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Exhibit Display

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	Creatinine	2.7 mg/dL
Urinalysis		
	Nitrites	negative
	Bacteria	none
	Red blood cells	20-30/hpf

CT scan of chest is shown in the exhibit. Histologic examination of the lung would most likely reveal which of the following findings?

- A. Fibrinoid degeneration of the vessel wall with luminal narrowing (21%)
- ✓ B. Necrotizing arteritis and focal collections of epithelioid histiocytes (39%)
- ✗ C. Necrotizing granuloma with a predominance of eosinophils (31%)
- D. Proliferation of irregularly shaped glands and pleomorphic cells (6%)

Incorrect
Correct answer

39%
Answered correctly

21 secs
Time Spent

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Explanation

Granulomatosis with polyangiitis	
Manifestations	• Upper respiratory: sinusitis/otitis, saddle-nose deformity • Lower respiratory: lung nodules/cavitation • Renal: rapidly progressive glomerulonephritis • Skin: livedo reticularis, nonhealing ulcers
Diagnosis	• ANCA positive, particularly c-ANCA (PR3-ANCA) • Biopsy: necrotizing vasculitis with granulomas and little/no complement & immunoglobulin
ANCA = antineutrophil cytoplasmic antibody; **PR3** = proteinase-3.	

This patient has chronic constitutional and pulmonary symptoms associated with patchy lung infiltrates/cavitation and laboratory findings of glomerulonephritis (hematuria, increased creatinine). These findings are suggestive of **granulomatosis with polyangiitis** (GPA), a **small vessel vasculitis** associated with antineutrophilic cytoplasmic antibodies **(ANCA)**. Patients with GPA generally present with weeks or months of **constitutional symptoms** (eg, arthralgias, fatigue, low-grade fever, weight loss) and manifestations in the following areas:

- Upper airway: otitis media, nasal crusting/ulcers, rhinorrhea
- Lower airway: hoarseness, cough, stridor, hemoptysis, pulmonary infiltrates
- Kidney: rapidly progressive glomerulonephritis

Laboratory evaluation frequently shows **anemia** of chronic disease due to elevated levels of inflammatory cytokines and **renal insufficiency** due to **glomerulonephritis** (eg, microscopic **hematuria**, red cell casts, negative nitrate/bacteriuria). Chest imaging often reveals patchy lung infiltrates, nodules, and/or cavitations.

Although ANCA is positive in >85% of patients with GPA (c-ANCA in particular), it is a non-specific marker. Therefore, tissue biopsy is generally required for diagnosis; histopathology will show a **necrotizing arteritis** with **granulomatous inflammation** (eg, **epithelioid histiocytes**, multinucleated giant cells) and a mixture of

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- Lower airway: hoarseness, cough, stridor, hemoptysis, pulmonary infiltrates
- Kidney: rapidly progressive glomerulonephritis

Laboratory evaluation frequently shows **anemia** of chronic disease due to elevated levels of inflammatory cytokines and **renal insufficiency** due to **glomerulonephritis** (eg, microscopic **hematuria**, red cell casts, negative nitrate/bacteriuria). Chest imaging often reveals patchy lung infiltrates, nodules, and/or cavitations.

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(Choice A) Polyarteritis nodosa (PAN) is marked by transmural segmental inflammation of medium-sized muscular arteries with fibrinoid necrosis, which causes lumen narrowing, thrombosis, and organ ischemia. Although PAN often affects the kidney, the lung is generally spared. In contrast to GPA, PAN is *not* associated with ANCA.

(Choice C) Eosinophilic granulomatosis with polyangiitis (Churg-Strauss) is marked by the formation of necrotizing granulomas with a predominance of eosinophils. Although renal insufficiency and glomerulonephritis occur in a minority of patients, almost all patients have years of preceding asthma. In addition, cavitary lesions are uncommon.

(Choice D) Adenocarcinoma of the lung is generally marked by the proliferation of irregular glands and pleiomorphic cells. Although this condition can cause normocytic anemia, cough, shortness of breath, and weight loss, it is not strongly associated with renal insufficiency or cavitary lung lesions (cavitary lesions are more common with squamous cell carcinoma). In addition, most cases arise in older patients.

Educational objective:
Granulomatosis with polyangiitis is an ANCA-associated systemic vasculitis that typically presents with upper (eg, purulent nasal discharge) and lower (eg, cavitary lung lesion) respiratory manifestations and renal insufficiency due to glomerulonephritis. Biopsy typically reveals a necrotizing arteritis with granulomatous inflammation (eg, epithelioid histiocytes, multinucleated giant cells) and lack of immunoglobulin/complement deposition.

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This patient has chronic constitutional and pulmonary symptoms associated with patchy lung infiltrates/cavitation and la

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Exhibit Display

Granulomatosis with polyangiitis

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A 65-year-old man with type 2 diabetes mellitus comes to the office due to decreased sexual performance over the last 6 months. The patient says his libido, muscle strength, and energy level are normal, but his erections are inconsistent, weak, and of inadequate duration for sexual activity. Past medical history is positive for hypertension, but he has no history of coronary artery disease and no suspicious chest pain. Bilateral ankle brachial index testing is within normal limits. The patient is treated with sildenafil, which gives significant improvement in his sexual performance. The intracellular signaling of this drug is most similar to that of which of the following substances?

- A. 1,25-dihydroxycholecalciferol
- B. Brain natriuretic peptide
- C. Gamma-aminobutyric acid
- D. Insulin
- E. Interleukin-2
- F. Platelet-derived growth factor

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- A. 1,25-dihydroxycholecalciferol (3%)
- ✓ B. Brain natriuretic peptide (70%)
- C. Gamma-aminobutyric acid (7%)
- D. Insulin (6%)
- E. Interleukin-2 (3%)
- F. Platelet-derived growth factor (9%)

Correct | 70% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Atrial and **brain natriuretic peptides** (ANP and BNP) are polypeptide hormones that exert their effect by binding ANP receptors. These transmembrane receptors are linked to a **guanylyl cyclase** that converts guanosine 5′-triphosphate to cyclic guanosine 3′,5′-monophosphate (cGMP). cGMP then activates a downstream cGMP-dependent protein kinase, leading to relaxation of vascular smooth muscle and subsequent **vasodilation**. Nitric oxide activates the same cGMP second messenger system by binding a cytosolic guanylyl cyclase.

Penile erection is initiated by release of acetylcholine and nitric oxide (NO) from parasympathetic fibers, leading to

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Penile erection is initiated by release of acetylcholine and nitric oxide (NO) from parasympathetic fibers, leading to a rise in intracellular cGMP and vasodilation of the arteries supplying the corpus cavernosum. Sildenafil is a **phosphodiesterase-5 inhibitor** that decreases degradation of cGMP, enhancing the effect of parasympathetic activation in erectile tissues. Because vasodilatory nitrates (eg, nitroglycerin, isosorbide mononitrate) work by releasing NO, coadministration of sildenafil with nitrates can lead to excessive cGMP-induced vasodilation and severe hypotension.

(Choice A) 1,25-dihydroxycholecalciferol is the active form of vitamin D; it is a sterol vitamin that freely crosses cell membranes and interacts with a receptor that primarily resides in the nucleus.

(Choice C) Gamma-aminobutyric acid is an inhibitory neurotransmitter that acts to hyperpolarize nerve cells by binding ligand-gated ion channels or metabotropic receptors.

(Choice D) The insulin receptor consists of 2 alpha and 2 beta subunits. The alpha subunits are extracellular and provide the binding site for insulin. The beta subunits are intracellular and contain tyrosine kinase domains.

(Choice E) Interleukin-2 (IL-2) receptor is a transmembrane protein complex that acts via a multipathway system, including a JAK-STAT signaling component, a MAP kinase pathway, and phosphoinositide 3-kinase.

(Choice F) Platelet-derived growth factor stimulates cell growth and division, including angiogenesis. It acts via a tyrosine kinase receptor.

Educational objective:
Atrial natriuretic peptide, brain natriuretic peptide, and nitric oxide activate guanylyl cyclase and increase conversion of guanosine 5′-triphosphate to cyclic guanosine 3′,5′-monophosphate (cGMP). Phosphodiesterase

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Educational objective:
Atrial natriuretic peptide, brain natriuretic peptide, and nitric oxide activate guanylyl cyclase and increase conversion of guanosine 5′-triphosphate to cyclic guanosine 3′,5′-monophosphate (cGMP). Phosphodiesterase inhibitors (eg, sildenafil) decrease the degradation of cGMP. Elevated intracellular cGMP levels lead to relaxation of vascular smooth muscle and vasodilation.

References

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A 54-year-old man is transferred to the hospital from his cardiologist's office due to atrial fibrillation with rapid ventricular response. His symptoms began 24 hours earlier with palpitations, lightheadedness, and chest tightness. The patient has had similar episodes several times during the last year. Past history is notable for mitral valve repair 3 years ago for severe symptomatic mitral regurgitation. The patient undergoes direct current cardioversion due to worsening hemodynamic instability, with resolution of the arrhythmia and all associated symptoms. He is considered for long-term amiodarone therapy to prevent future arrhythmic episodes. Which of the following should be tested before initiating this medication?

- A. 24-hour urinary cortisol
- B. 24-hour urinary metanephrines
- C. Oral glucose tolerance test
- D. Serum prolactin
- E. Serum testosterone
- F. Serum TSH

Submit

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- ✗ A. 24-hour urinary cortisol (4%)
- B. 24-hour urinary metanephrines (9%)
- C. Oral glucose tolerance test (7%)
- D. Serum prolactin (1%)
- E. Serum testosterone (1%)
- ✓ F. Serum TSH (77%)

Incorrect
Correct answer
F

77%
Answered correctly

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Explanation

Major adverse effects of amiodarone	
Cardiac	• Sinus bradycardia, heart block • Proarrhythmias: QT prolongation
Pulmonary	• Chronic interstitial pneumonitis
	• Hypothyroidism

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Explanation

Major adverse effects of amiodarone	
Cardiac	• Sinus bradycardia, heart block • Proarrhythmias: QT prolongation
Pulmonary	• Chronic interstitial pneumonitis
Endocrine	• Hypothyroidism • Hyperthyroidism
Gastrointestinal/ hepatic	• Elevated transaminases, hepatitis
Ocular	• Corneal microdeposits • Optic neuropathy
Dermatologic	• Blue-gray skin discoloration
Neurologic	• Peripheral neuropathy

Amiodarone is a class III anti-arrhythmic agent used to suppress life-threatening rhythm disturbances. Because it is 40% iodine by weight, am**IOD**arone can cause a number of alterations in thyroid function. It can cause **hypothyroidism** due to decreased production of thyroid hormone. Individuals with pre-existing autoimmune thyroid disease are at greatest risk and so should be screened for subclinical hypothyroidism with a **serum TSH** assay prior to initiating amiodarone therapy. Amiodarone can also cause **hyperthyroidism** due to increased **thyroid hormone synthesis** or **destructive thyroiditis** with release of preformed thyroid hormone.

(Choices A and B) Amiodarone is not associated with altered function of the adrenal cortex or medulla.

(Choice C) The oral glucose tolerance test is used to screen for diabetes mellitus, primarily in pregnant women. Amiodarone is not associated with significant alterations in insulin sensitivity or blood glucose levels.

(Choice D) Release of prolactin is primarily regulated by inhibitory effects of dopamine from the hypothalamus

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Dermatologic	• Blue-gray skin discoloration
Neurologic	• Peripheral neuropathy

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(Choice D) Release of prolactin is primarily regulated by inhibitory effects of dopamine from the hypothalamus. Release is stimulated to a small degree by thyrotropin-releasing hormone and therefore may be mildly affected by amiodarone. However, these effects would not be clinically significant.

(Choice E) Amiodarone can cause erectile dysfunction, which may be related to its alpha and beta adrenoreceptor blocking effect. Amiodarone does not significantly alter serum testosterone levels.

Educational objective:
Amiodarone is 40% iodine by weight. It can cause hypothyroidism due to decreased production of thyroid hormone. Amiodarone can also cause hyperthyroidism due to increased thyroid hormone synthesis or destructive thyroiditis with release of preformed thyroid hormone.

References

- Effects of amiodarone therapy on thyroid function.

Pharmacology (Subject) | Cardiovascular System (System) | Amiodarone (Topic)

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A 34-year-old man comes to the office due to heart palpitations that are particularly prominent at night. With moderate exertion he also experiences head-pounding accompanied by involuntary head-bobbing. The patient recently emigrated from Southeast Asia and remembers being diagnosed with a heart murmur years before, but he cannot recall the type of murmur and has never received any treatment. He has no other medical conditions and takes no medications. He does not use tobacco, alcohol, or illicit drugs. The patient's father has coronary artery disease and his mother has type 2 diabetes mellitus. Based on this patient's history, which of the following findings is most likely to be present on further evaluation?

- A. Impaired left ventricular contractility
- B. Left ventricular outflow obstruction
- C. Restricted left ventricular filling
- D. Systolic and diastolic hypertension
- E. Widening of the pulse pressure

Submit

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- A. Impaired left ventricular contractility (2%)
- ✗ B. Left ventricular outflow obstruction (9%)
- C. Restricted left ventricular filling (5%)
- D. Systolic and diastolic hypertension (2%)
- ✓ E. Widening of the pulse pressure (79%)

Incorrect
Correct answer
E

79%
Answered correctly

02 secs
Time Spent

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Explanation

Chronic aortic regurgitation	
Common etiologies	• Congenital bicuspid aortic valve • Postinflammatory (eg, rheumatic heart disease, endocarditis) • Aortic root dilation (eg, Marfan syndrome, syphilis)
Pathophysiology	• Backflow from aorta into LV → ↑ LV end-diastolic volume • LV initially compensates with eccentric hypertrophy → ↑ SV & CO

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Chronic aortic regurgitation	
Common etiologies	• Congenital bicuspid aortic valve • Postinflammatory (eg, rheumatic heart disease, endocarditis) • Aortic root dilation (eg, Marfan syndrome, syphilis)
Pathophysiology	• Backflow from aorta into LV → ↑ LV end-diastolic volume • LV initially compensates with eccentric hypertrophy → ↑ SV & CO • Eventual LV dysfunction → ↓ SV & CO → heart failure
Clinical findings	• Decrescendo diastolic murmur • Widened pulse pressure (↑SBP & ↓DBP) • Rapid rise-rapid fall ("water-hammer") pulsation • Abrupt carotid distension & collapse, "pistol-shot" femoral pulses
CO = cardiac output; **DBP** = diastolic blood pressure; **LV** = left ventricle; **SBP** = systolic blood pressure; **SV** = stroke volume.	

This patient has a number of clinical findings that are characteristic of **chronic aortic regurgitation** (AR). Failure of the aortic valve to tightly seal off the aorta from the left ventricle at the end of systole leads to rapid loss of aortic pressure and an overall **decrease in diastolic blood pressure**. In addition, the left ventricle undergoes eccentric hypertrophy to initially compensate for an increase in left ventricular end-diastolic volume, which leads to increased stroke volume and cardiac output. Because pulse pressure (the difference between diastolic and systolic blood pressure) is proportional to stroke volume, the compensatory **increased stroke volume** on top of reduced diastolic pressure causes the characteristic **widened pulse pressure** seen with AR.

This high-amplitude pulsation with rapid diastolic collapse is responsible for the unusual pulsation findings in AR. Forceful pulsations in the intracranial arteries can cause **head bobbing** with each heart beat (de Musset sign), and patients may experience **palpitations** due to forceful left ventricular contraction. In addition, physical examination can show abrupt distension and collapse of the carotid arteries (Corrigan sign) and peripheral arteries ("water-hammer" pulse), and reveal "pistol-shot" femoral pulses (Traube sign) on auscultation.

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hypertrophy to initially compensate for an increase in left ventricular end-diastolic volume, which leads to increased stroke volume and cardiac output. Because pulse pressure (the difference between diastolic and systolic blood pressure) is proportional to stroke volume, the compensatory **increased stroke volume** on top of reduced diastolic pressure causes the characteristic **widened pulse pressure** seen with AR.

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(Choice A) Impaired left ventricular contractility occurs later in the course of AR and eventually leads to heart failure. Because head-bobbing and other characteristic findings in AR are due to increased stroke volume and high-amplitude, rapid rise-rapid fall pulsation, these findings are less prominent once impaired left ventricular contractility (and decreased stroke volume) sets in.

(Choices B and C) Left ventricular outflow tract obstruction, such as occurs with aortic stenosis, leads to reduced stroke volume and characteristic weak and delayed pulses (pulsus parvus et tardus). Restriction of left ventricular filling, as occurs in mitral stenosis, also reduces stroke volume due to reduced left ventricular end-diastolic volume (reduced preload). Clinical findings associated with high-amplitude pulsation will not be present with reduced stroke volume.

(Choice D) Combined systolic and diastolic hypertension is not necessarily accompanied by widened pulse pressure; the signs of high amplitude, rapid rise-rapid fall pulsation are more characteristic of chronic AR.

Educational objective:
Chronic aortic regurgitation (AR) causes a reduction in diastolic blood pressure and a compensatory increase in left ventricular stroke volume. These changes create a high-amplitude, rapid rise-rapid fall pulsation (ie, widened pulse pressure) and the other characteristic findings of AR (eg, head bobbing, "pistol-shot" femoral pulses).

Pathophysiology	Cardiovascular System	Aortic regurgitation
Subject	System	Topic

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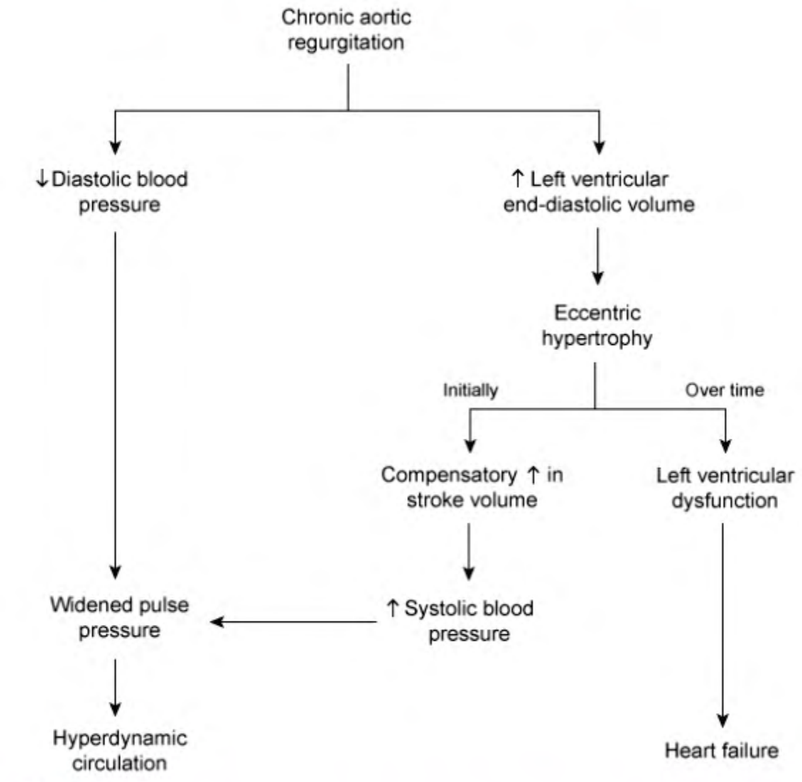
Pathophysiology	• Backflow from aorta into LV → ↑ LV end-diastolic volume • LV initially compensates with eccentric hypertrophy → ↑ SV & CO

Exhibit Display

Pathophysiology of chronic aortic regurgitation



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(Choices B and C) Left ventricular outflow tract obstruction, such as occurs with aortic stenosis, leads to reduced stroke volume and characteristic weak and delayed pulses (pulsus parvus et tardus). Restriction of left ventricular

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 63-year-old man is brought to the emergency department due to an episode of syncope. The patient was recently discharged back to a nursing home after being hospitalized for a urinary tract infection. He has a history of coronary artery disease and takes aspirin, clopidogrel, and atorvastatin. His other medical conditions include type 2 diabetes mellitus and hypertension. The patient does not use tobacco, alcohol, or illicit drugs. Blood pressure is 68/32 mm Hg and pulse is 118/min. Invasive hemodynamic monitoring reveals an elevated right atrial pressure of 17 mm Hg and a pulmonary capillary wedge pressure of 7 mm Hg (normal: 8-10 mm Hg).

Item 1 of 2

Which of the following is the most likely cause of this patient's current condition?

- A. Anaphylactic shock
- B. Gastrointestinal bleeding
- C. Right ventricular infarction
- D. Septic shock
- E. Severe aortic stenosis

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Item 2 of 2

Intravenous fluids and vasopressors are administered, but the patient remains hypotensive and dies 3 hours later. Autopsy examination is performed and reveals bilateral hippocampal necrosis. Which of the following areas of the colon would be most likely to also demonstrate necrosis?

- A. Ascending colon
- B. Cecum
- C. Hepatic flexure
- D. Ileocolic junction
- E. Splenic flexure

Submit

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 63-year-old man is brought to the emergency department due to an episode of syncope. The patient was recently discharged back to a nursing home after being hospitalized for a urinary tract infection. He has a history of coronary artery disease and takes aspirin, clopidogrel, and atorvastatin. His other medical conditions include type 2 diabetes mellitus and hypertension. The patient does not use tobacco, alcohol, or illicit drugs. Blood pressure is 68/32 mm Hg and pulse is 118/min. Invasive hemodynamic monitoring reveals an elevated right atrial pressure of 17 mm Hg and a pulmonary capillary wedge pressure of 7 mm Hg (normal: 8-10 mm Hg).

Item 1 of 2

Which of the following is the most likely cause of this patient's current condition?

- A. Anaphylactic shock (1%)
- ✗ B. Gastrointestinal bleeding (4%)
- ✓ C. Right ventricular infarction (62%)
- D. Septic shock (28%)
- E. Severe aortic stenosis (3%)

Incorrect
Correct answer
C

62%
Answered correctly

06 secs
Time Spent

2023
Version

Explanation

Right-sided heart failure

Primary disturbance
Right ventricular dysfunction

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Item 1 of 2

Which of the following is the most likely cause of this patient's current condition?

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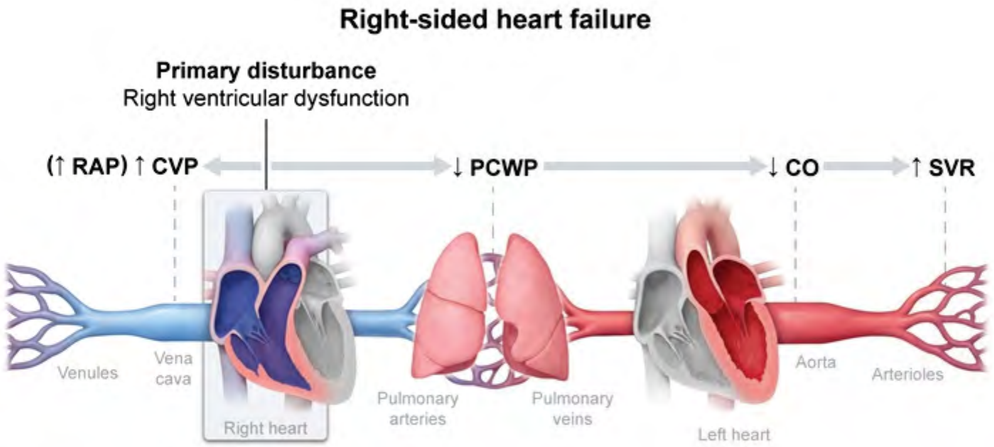

CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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PCWP = pulmonary capillary wedge pressure; **RAP** = right atrial pressure; **SVR** = systemic vascular resistance.

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This patient is in shock (eg, severe hypotension, end organ dysfunction [syncope]) due to an unknown cause. Hemodynamic monitoring (eg, pulmonary artery catheterization) shows **decreased pulmonary capillary wedge pressure (PCWP)** and **increased right atrial pressure (RAP)**, which indicate impaired blood flow from the right ventricle to the left side of the heart **(right-sided heart failure)**. Common causes of right-sided heart failure include obstruction of pulmonary circulation (eg, pulmonary arterial hypertension, pulmonary embolism) and **right ventricular infarction** (likely in this patient due to multiple cardiovascular risk factors).

In right ventricular infarction, the right ventricle is unable to effectively pump blood forward into the pulmonary arteries, causing increased pressure in the right atrium (ie, elevated central venous pressure). The decreased right ventricular outflow also lowers PCWP (an indicator of left-sided preload), which subsequently reduces cardiac output and causes hypotension. A reflexive increase in heart rate is mostly intact in this patient, though it is often impaired in right ventricular infarction due to frequent ischemic involvement of the conduction system.

(Choices A and D) Anaphylactic shock and septic shock are associated with peripheral vasodilation and massive fluid shift to the extravascular space. The depleted intravascular volume lowers the mean circulatory filling pressure, reducing both RAP and PCWP, while hypotension caused by widespread peripheral vasodilation leads to a compensatory increase in cardiac output.

(Choice B) Gastrointestinal bleeding can cause hypovolemic shock with decreased RAP, PCWP, and cardiac output. Decreased cardiac output triggers an increase in sympathetic tone with peripheral vasoconstriction (increased systemic vascular resistance) to help maintain perfusion pressure.

(Choice E) Aortic stenosis causes left ventricular outflow obstruction and would increase pressure in the left atrium (increased PCWP) and pulmonary circulation. In severe cases, right-sided heart failure can also develop, leading to increased RAP. Systemic vascular resistance is increased with aortic stenosis in response to a reduction in cardiac output.

Educational objective:
Right ventricular infarction (right-sided heart failure) can lead to shock via impaired forward blood flow to the left heart, which lowers left-sided preload (decreased pulmonary capillary wedge pressure) and decreases cardiac

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Hemodynamic monitoring (eg, pulmonary artery catheterization) shows **decreased pulmonary capillary wedge pressure (PCWP)** and **increased right atrial pressure (RAP)**, which indicate impaired blood flow from the right ventricle to the left side of the heart **(right-sided heart failure)**. Common causes of right-sided heart failure include obstruction of pulmonary circulation (eg, pulmonary arterial hypertension, pulmonary embolism) and **right ventricular infarction** (likely in this patient due to multiple cardiovascular risk factors).

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(Choices A and D) Anaphylactic shock and septic shock are associated with peripheral vasodilation and massive fluid shift to the extravascular space. The depleted intravascular volume lowers the mean circulatory filling pressure, reducing both RAP and PCWP, while hypotension caused by widespread peripheral vasodilation leads to a compensatory increase in cardiac output.

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Educational objective:
Right ventricular infarction (right-sided heart failure) can lead to shock via impaired forward blood flow to the left heart, which lowers left-sided preload (decreased pulmonary capillary wedge pressure) and decreases cardiac output. The reduced right ventricular output also raises right atrial and central venous pressure.

Pathology Cardiovascular System Myocardial infarction

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Hemodynamic monitoring (eg, pulmonary artery catheterization) shows **decreased pulmonary capillary wedge**

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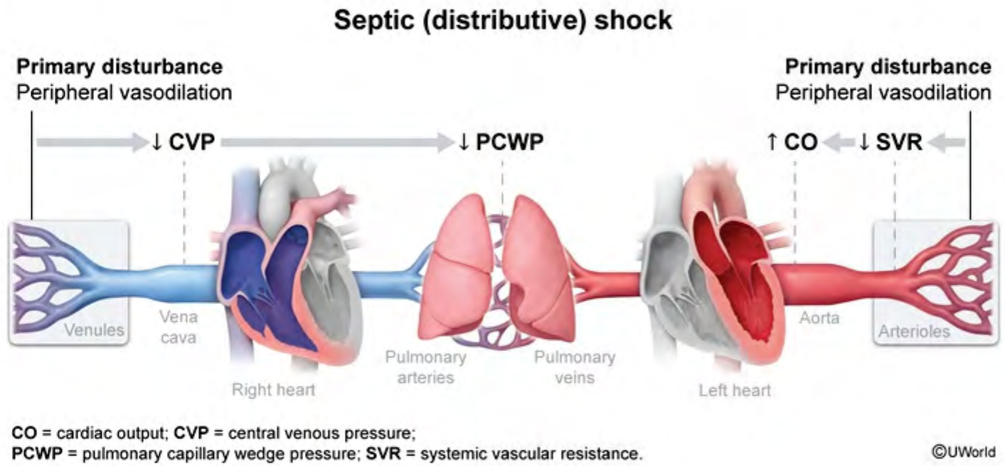

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Pathology | Cardiovascular System | Myocardial infarction

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Hemodynamic monitoring (eg, pulmonary artery catheterization) shows **decreased pulmonary capillary wedge**

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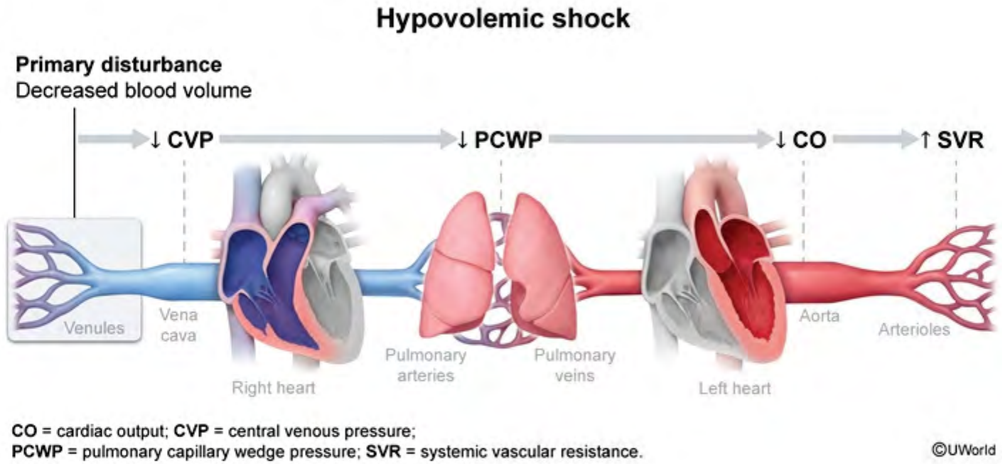

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Pathology | Cardiovascular System | Myocardial infarction

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Item 2 of 2

Intravenous fluids and vasopressors are administered, but the patient remains hypotensive and dies 3 hours later. Autopsy examination is performed and reveals bilateral hippocampal necrosis. Which of the following areas of the colon would be most likely to also demonstrate necrosis?

- A. Ascending colon (2%)
- ✗ B. Cecum (1%)
- C. Hepatic flexure (3%)
- D. Ileocolic junction (5%)
- ✓ E. Splenic flexure (86%)

Incorrect
Correct answer
E

86%
Answered correctly

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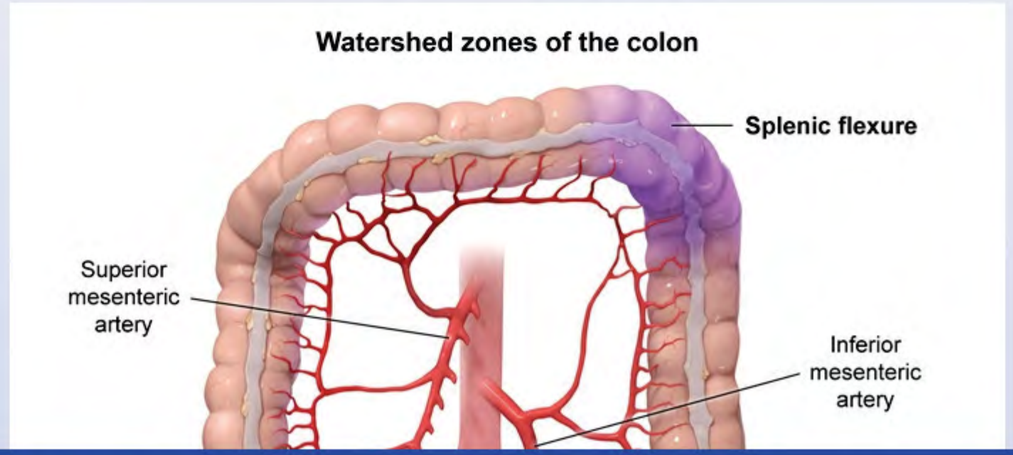
Explanation



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Item 2 of 2

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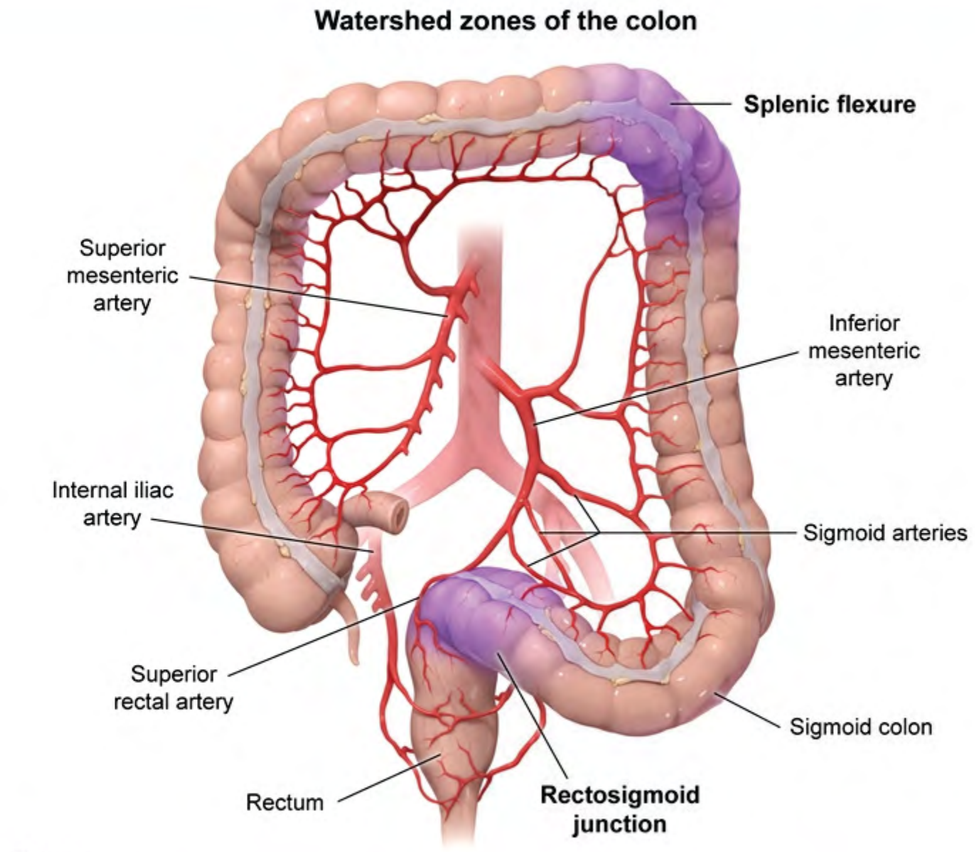

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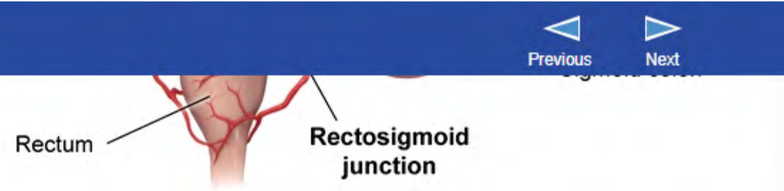

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This patient's bilateral hippocampal necrosis is characteristic of the effects of global cerebral ischemia due to **systemic hypotension**. The hippocampus is particularly vulnerable during shock because of the high metabolic demand of its CA1 pyramidal neurons. **Watershed areas** located between the distal terminal branches of 2 different vascular territories (border zones) are also susceptible to ischemia because they have a low baseline perfusion pressure and poorly tolerate sustained decreases in systemic blood pressure.

Important watershed areas in the body include:

- Gastrointestinal (colon)
  - **Splenic flexure**: the border zone supplied by branches of the superior mesenteric artery and inferior mesenteric artery
  - **Rectosigmoid junction**: the border zone supplied by the sigmoid arteries and the superior rectal arteries
- Brain: the borders of the areas supplied by the anterior cerebral artery, middle cerebral artery, and posterior cerebral artery

(Choices A, B, C, and D) The ascending colon, cecum, hepatic flexure, and ileocolic junction are well perfused by the superior mesenteric artery and do not lie in watershed areas.

Educational objective:
Severe systemic hypotension (eg, shock) is most likely to cause ischemia first in areas of high metabolic demand (eg, hippocampus) or watershed zones, which are areas that are supplied by the distal branches of two different major arteries. Commonly affected areas in the colon include the splenic flexure and rectosigmoid junction.

Pathology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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A 60-year-old man is diagnosed with exertional angina. His treatment regimen includes metoprolol, isosorbide dinitrate and aspirin. He takes isosorbide dinitrate early in the morning and again in the afternoon, but he does not take an evening dose. Such a pattern of drug administration is intended to decrease which of the following?

- A. Pharmacokinetic drug interaction
- B. Pharmacodynamic drug antagonism
- C. Effect potentiation
- D. Tolerance development
- E. Withdrawal symptoms
- F. Drug noncompliance

Submit

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A 60-year-old man is diagnosed with exertional angina. His treatment regimen includes metoprolol, isosorbide dinitrate and aspirin. He takes isosorbide dinitrate early in the morning and again in the afternoon, but he does not take an evening dose. Such a pattern of drug administration is intended to decrease which of the following?

- A. Pharmacokinetic drug interaction (4%)
- ✗ B. Pharmacodynamic drug antagonism (3%)
- C. Effect potentiation (18%)
- ✓ D. Tolerance development (65%)
- E. Withdrawal symptoms (3%)
- F. Drug noncompliance (4%)

Incorrect
Correct answer
D

65%
Answered correctly

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Explanation

Around-the-clock nitrate administration (in any form) rapidly results in development of tolerance to nitrates. This is why a nitrate-free interval must be provided every day in patients that are using daily long acting nitrates. The mechanism by which this occurs has not been fully demonstrated, but it is theorized that it is due to a decreased vascular sensitivity to nitrates and an increased sensitivity to endogenous vasoconstricting agents. Usually the nitrate-free period is timed to occur during the night when the patient is sleeping and cardiac work is the least.

(Choice A) A pharmacokinetic drug interaction refers to how drugs that are used together in a patient affect the absorption and elimination of each other. An example of this is the ability of rifampin to increase the metabolism and elimination of warfarin by inducing cytochrome P-450. This is not the reason for providing a nitrate-free interval.

(Choice B) Pharmacodynamic drug antagonism refers to the ability of a drug, as part of its mechanism of action

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Explanation

Around-the-clock nitrate administration (in any form) rapidly results in development of tolerance to nitrates. This is why a nitrate-free interval must be provided every day in patients that are using daily long acting nitrates. The mechanism by which this occurs has not been fully demonstrated, but it is theorized that it is due to a decreased vascular sensitivity to nitrates and an increased sensitivity to endogenous vasoconstricting agents. Usually the nitrate-free period is timed to occur during the night when the patient is sleeping and cardiac work is the least.

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(Choice B) Pharmacodynamic drug antagonism refers to the ability of a drug, as part of its mechanism of action, to antagonize, or prevent, the effect of another drug or endogenous substance. An example of this is the effect that labetalol has on alpha-1 and beta adrenergic receptors – it prevents the binding of catecholamines to these receptors.

(Choice C) Effect potentiation refers to one drug's ability to increase the pharmacologic effect of another agent. The drugs mentioned in this question stem do not potentiate the effects of each other, and this is not the reason for providing a nitrate-free period. Beta-adrenergic blockers are often prescribed along with nitrates to prevent reflex tachycardia as well as to provide decreased cardiac work by a mechanism different from that of nitrates.

(Choice E) Withdrawal symptoms occur when a patient forms a physiologic dependence on a drug or medication and that drug or medication is then discontinued. This is common in patients using alcohol, opiates, benzodiazepines, stimulants and some antidepressants. This is not the reason for providing a nitrate-free period in patients taking nitrates.

(Choice F) Drug noncompliance is a problem where patients do not take the medications prescribed to them properly or at all. It is frequent among patients who do not understand their disease, can not afford medications, or simply can not remember to take them. The latter becomes much more frequent when patients are taking multiple medications with many times-per-day dosing schedules. This is also not the reason for providing a nitrate-free

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(Choice A) A pharmacokinetic drug interaction refers to how drugs that are used together in a patient affect the absorption and elimination of each other. An example of this is the ability of rifampin to increase the metabolism and elimination of warfarin by inducing cytochrome P-450. This is not the reason for providing a nitrate-free interval.

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(Choice F) Drug noncompliance is a problem where patients do not take the medications prescribed to them properly or at all. It is frequent among patients who do not understand their disease, can not afford medications, or simply can not remember to take them. The latter becomes much more frequent when patients are taking multiple medications with many times-per-day dosing schedules. This is also not the reason for providing a nitrate-free period every day in patients taking long-term nitrates.

Educational objective:
Patients taking daily maintenance nitrates need to have a nitrate-free period every day to avoid tolerance to the drug.

Pharmacology	Cardiovascular System	Nitrates
Subject	System	Topic

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 65-year-old man with a long history of hypertension and type 2 diabetes mellitus comes to the office for a routine checkup. The patient is a former smoker with a 15-pack-year history. He jogs 3 miles per day. BMI is 28.5 kg/m^2. Family history is unremarkable. Auscultation findings over the cardiac apex are given below. The auscultation findings are best explained by which of the following?

Play Media

- A. Aortic root dilation
- B. Atrial septal defect
- C. Left ventricular hypertrophy
- D. Mitral valve prolapse
- E. Pericardial effusion

Submit

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Submit

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Play Media

- A. Aortic root dilation (5%)
- B. Atrial septal defect (3%)
- ✓ C. Left ventricular hypertrophy (50%)
- D. Mitral valve prolapse (35%)
- E. Pericardial effusion (5%)

Correct

50% Answered correctly

38 secs Time Spent

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Explanation

Extra (gallop) heart sounds		
	Features	Associated pathology*

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Show all

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive diastolic filling	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation
S4	• Heard just before S1 • Caused by blood striking a stiff LV wall during atrial contraction	• Concentric LV hypertrophy • Restrictive cardiomyopathy • Acute myocardial infarction
*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70). **EF** = ejection fraction; **LV** = left ventricular.		

This patient has a **low pitch**, extra heart sound at the **end of diastole** (just before S1) known as an **S4**. An S4 is thought to be caused by blood striking a stiff ventricular wall during atrial contraction. It can be a normal finding in elderly patients (eg, age >70) due to a physiologic, age-related decrease in left ventricular (LV) compliance. However, in younger patients it typically indicates pathologic LV wall stiffening, most commonly due to concentric LV hypertrophy in response to prolonged hypertension (likely the cause in this case).

In comparison, an S3 is heard in early diastole (just after S2) and is thought to result from reverberation of sound due to blood filling an enlarged and volume-overloaded ventricle (as occurs with eccentric hypertrophy in heart failure with reduced ejection fraction). Both S3 and S4 are best heard at the cardiac apex with the bell of the stethoscope.

(Choice A) Aortic root dilation can cause aortic regurgitation, which is recognized by a decrescendo early diastolic murmur. Aortic regurgitation resulting from aortic root (as opposed to valvular) pathology is typically best heard at the right upper sternal border.

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(Choice A) Aortic root dilation can cause aortic regurgitation, which is recognized by a decrescendo early diastolic murmur. Aortic regurgitation resulting from aortic root (as opposed to valvular) pathology is typically best heard at the right upper sternal border.

(Choice B) An atrial septal defect typically causes a faint, crescendo-decrescendo systolic murmur (best heard at the left upper sternal border) caused by increased blood flow through the pulmonic valve (ie, pulmonic flow murmur). Splitting of S2 is wide and fixed.

(Choice D) Mitral valve prolapse classically causes a midsystolic click followed by the late systolic murmur of mitral regurgitation (best heard at the cardiac apex). The click is relatively high pitched, and the timing varies with changes in LV blood volume.

(Choice E) Pericardial effusion commonly occurs as a complication of acute pericarditis, which may be recognized by a triphasic pericardial friction rub best heard at the left lower sternal border with the patient leaning forward.

Educational objective:
An S4 is a low frequency, extra heart sound that occurs at the end of diastole (just before S1). It is caused by blood striking a stiff ventricular wall during atrial contraction; it can be a normal finding in elderly patients (eg, age >70), but typically indicates pathologic ventricular wall stiffening in younger patients (most commonly due to concentric left ventricular hypertrophy in response to chronic hypertension).

References

- Association of the fourth heart sound with increased left ventricular end-diastolic stiffness.

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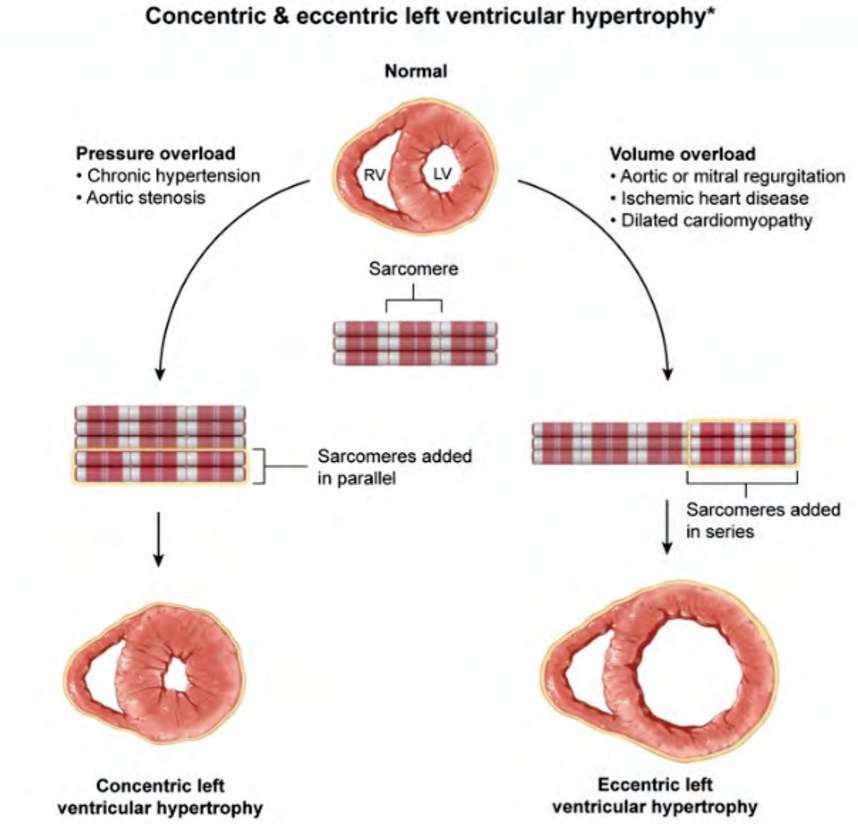

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diastolic filling

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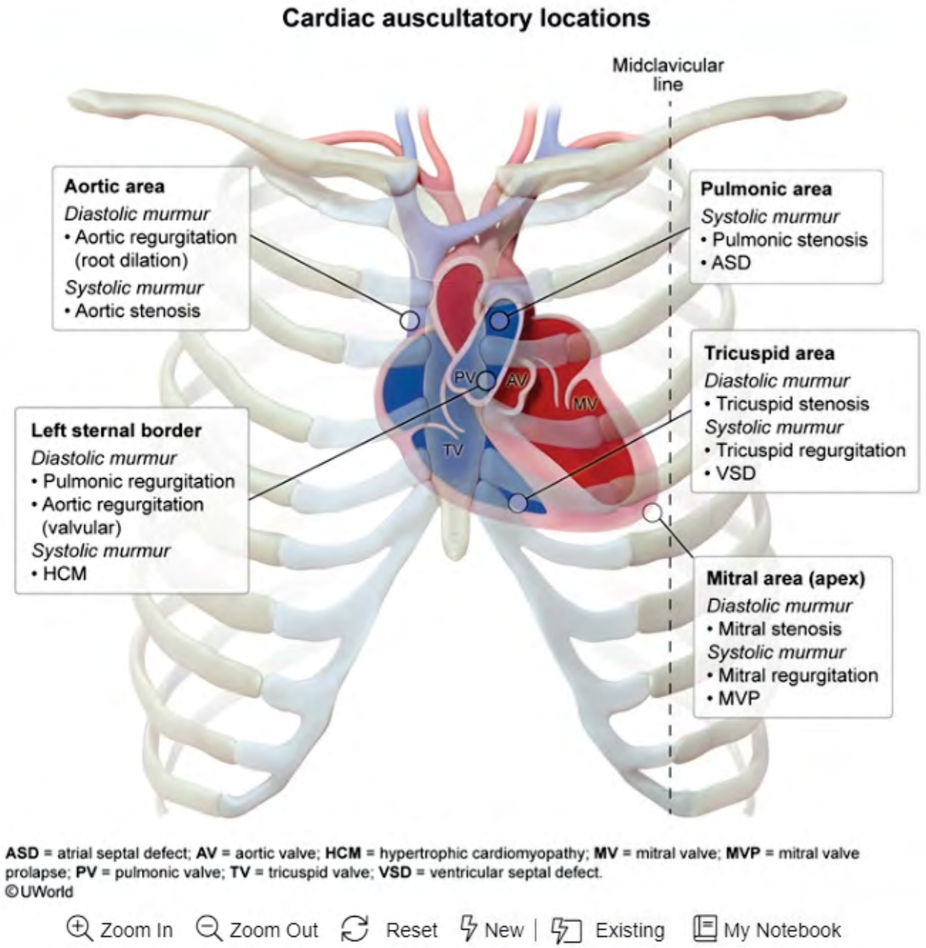

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A 57-year-old unconscious man is brought to the emergency department by his wife. Thirty minutes prior, the patient developed severe chest pain and asked to be taken to the hospital. On the way, he became unresponsive. According to his wife, the patient had been having intermittent, short-lived episodes of chest pain over the past several days. The patient's medical history is significant for hypertension, type 2 diabetes mellitus, and hypercholesterolemia. He is not adherent with medications or follow-up. In the emergency department, the patient has no spontaneous respirations or circulation and is pronounced dead. An autopsy examination is performed, and a cross-section of the mid-right coronary artery is shown in the exhibit. Which of the following is the most likely immediate cause of death in this patient?

- A. Atrial fibrillation
- B. Cardiac free wall rupture
- C. Embolic stroke
- D. Regional contractile dysfunction
- E. Ventricular fibrillation
- F. Ventricular septal rupture

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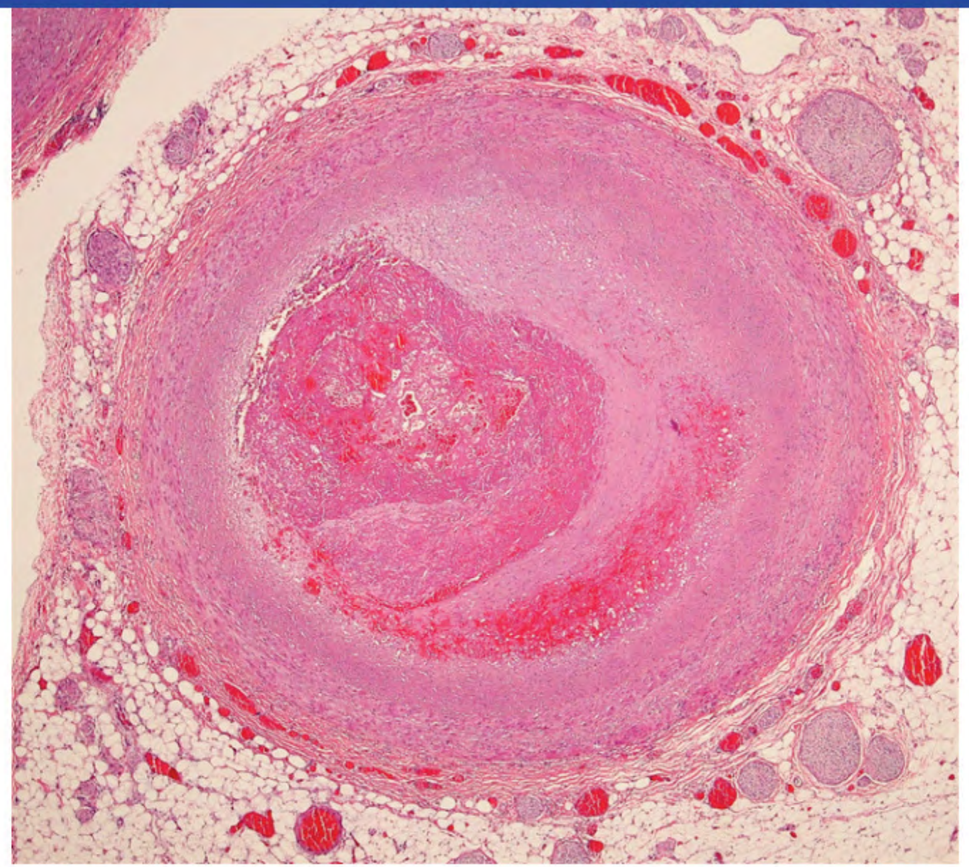

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- A. Atrial fibrillation (7%)
- B. Cardiac free wall rupture (4%)
- C. Embolic stroke (12%)
- ✗ D. Regional contractile dysfunction (20%)
- ✓ E. Ventricular fibrillation (53%)
- F. Ventricular septal rupture (1%)

Incorrect
Correct answer
E

53%
Answered correctly

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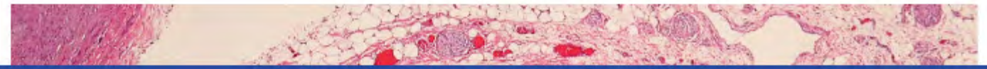
2023
Version

Explanation

Coronary artery thrombosis



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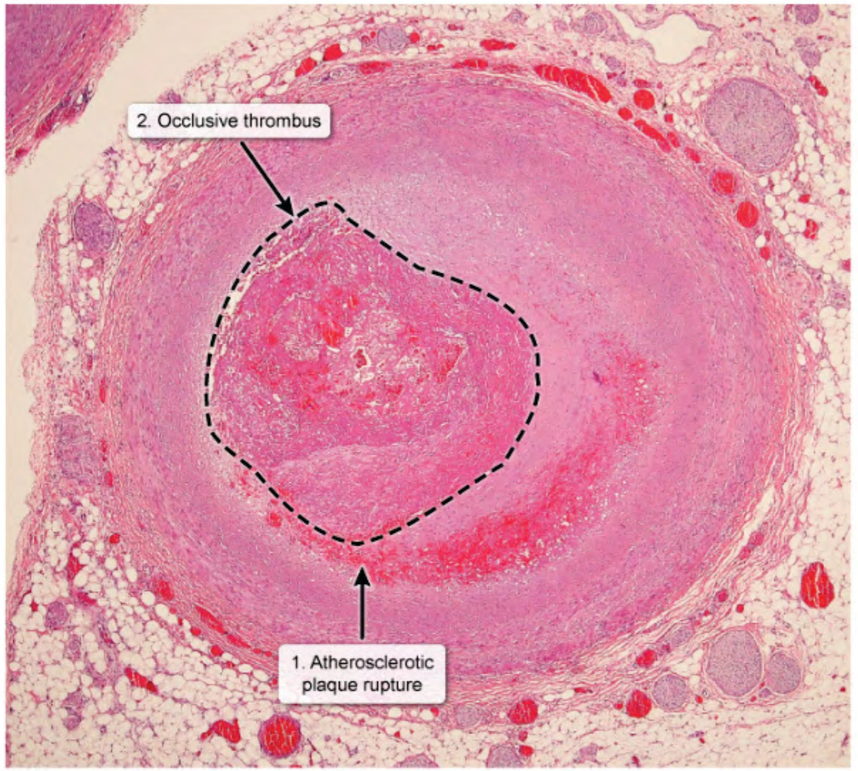
D. Regional contractile dysfunction (20%)

Exhibit Display

Coronary artery thrombosis



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This patient's clinical features are suggestive of **coronary artery disease** (chest pain and multiple risk factors) causing an **acute myocardial infarction** (MI). The diagnosis is confirmed by autopsy showing a large atheromatous plaque with superimposed thrombosis causing complete obstruction of the right coronary artery.

Acute MI is a common cause of **sudden cardiac death** (SCD) (the abrupt cessation of organized cardiac activity leading to hemodynamic collapse and inadequate tissue perfusion). SCD due to acute MI usually results from a malignant ventricular arrhythmia (ie, **ventricular fibrillation** or ventricular tachycardia degenerating to ventricular fibrillation) that is triggered by **electrical instability** in the ischemic myocardium.

(Choice A) Atrial fibrillation can sometimes occur during acute MI; however, organ perfusion is usually maintained and it is not a common cause of SCD.

(Choices B and F) Ventricular septal rupture (most common with left anterior descending artery occlusion) and cardiac free wall rupture are mechanical complications of acute MI that typically occur several days following the event. Septal rupture can sometimes occur acutely, but it is likely to cause symptomatic cardiogenic shock (eg, hypotension, respiratory distress/failure) rather than SCD.

(Choice C) Embolic stroke can result from embolization of a left ventricular (LV) mural thrombus that forms in an area of infarcted and akinetic myocardium. However, LV thrombus typically takes several days to form, and embolic stroke does not typically lead to SCD.

(Choice D) Acute MI commonly causes regional contractile dysfunction in the area of myocardium supplied by the affected artery. Infarction involving >50% of the myocardium can cause ventricular failure and cardiogenic shock, typically presenting with hypotension and respiratory distress rather than SCD.

Educational objective:
Ventricular fibrillation is the most common mechanism of sudden cardiac death due to acute myocardial infarction. It results from arrhythmogenic foci triggered by electrical instability in the ischemic myocardium.

Pathology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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This patient's clinical features are suggestive of **coronary artery disease** (chest pain and multiple risk factors)

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Mechanical complications of acute myocardial infarction

Complication	Time course	Clinical findings
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR) • Hypotension/cardiogenic shock
Interventricular septum rupture	Acute or within 3-5 days	• Chest pain • New holosystolic murmur • Hypotension/cardiogenic shock • Step up in O_2 level from RA to RV
Free wall rupture**	Within 5 days or up to 2 weeks	• Chest pain • Distant heart sounds • Shock, rapid progression to cardiac arrest
Left ventricular aneurysm**	Up to several months	• Heart failure • Angina, ventricular arrhythmias

*Usually due to right coronary artery occlusion.
**Usually due to left anterior descending artery occlusion.
MR = mitral regurgitation; **RA** = right atrium; **RV** = right ventricle.

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Pathology
Subject
Cardiovascular System
System
Myocardial Infarction
Topic

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A 54-year-old man with nonischemic cardiomyopathy comes to the office for a follow-up visit. He has been hospitalized frequently for acute decompensated heart failure, and currently has dyspnea on mild exertion. The patient also has a history of hypertension and takes multiple medications. Blood pressure is 116/70 mm Hg, and pulse is 72/min and regular. Physical examination shows elevated jugular venous pressure, bibasilar lung crackles, and lower extremity edema. Serum creatinine is 1.0 mg/dL and serum potassium is 4.2 mEq/L. Three weeks after starting digoxin therapy, the patient reports symptomatic improvement. Which of the following is the initial cellular event triggering this response to the new medication?

- A. Decreased sodium efflux from myocardial cells
- B. Increased intracellular calcium concentration
- C. Increased intracellular cyclic AMP concentration
- D. Increased potassium influx into myocardial cells
- E. Troponin sensitization to calcium

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- ✓ A. Decreased sodium efflux from myocardial cells (59%)
- B. Increased intracellular calcium concentration (31%)
- C. Increased intracellular cyclic AMP concentration (4%)
- D. Increased potassium influx into myocardial cells (4%)
- E. Troponin sensitization to calcium (0%)

Correct

59% Answered correctly

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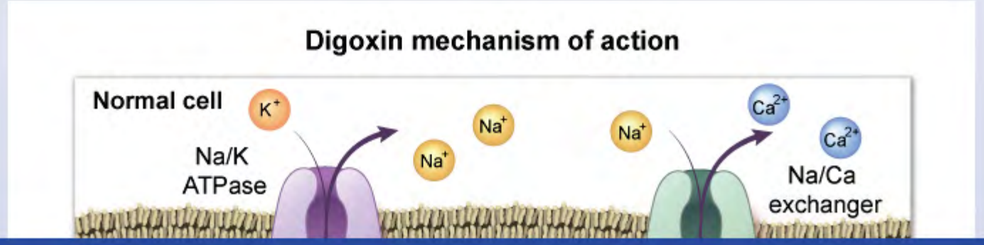
Explanation



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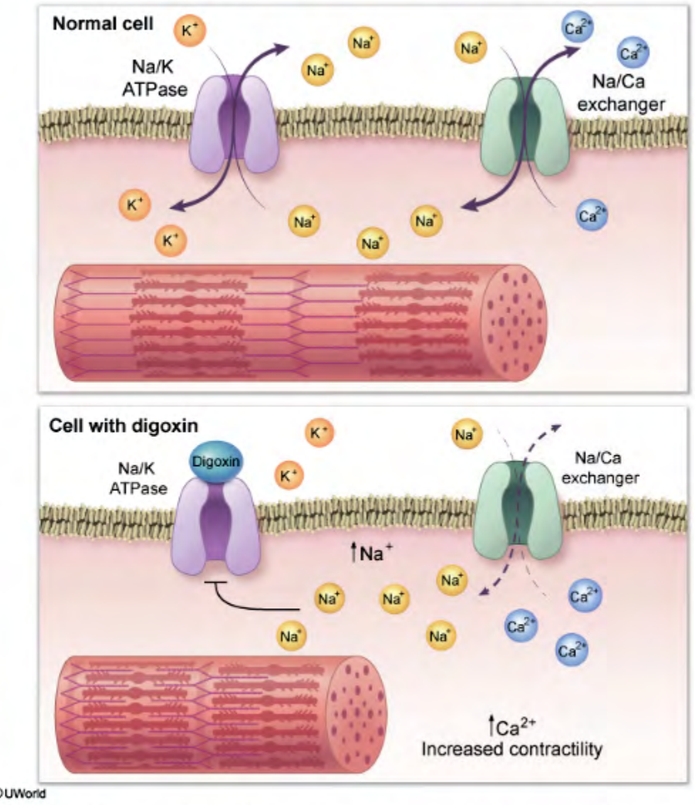
Digoxin mechanism of action



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Digoxin is a **positive inotropic agent** that provides symptomatic relief in patients with acute decompensated heart failure due to left ventricular systolic dysfunction. It also increases parasympathetic tone and slows conduction through the atrioventricular node, which can help improve cardiac function in patients with a rapid ventricular rate.

Digoxin directly **inhibits the Na-K-ATPase pump** in myocardial cells, leading to **decreased sodium efflux** and an increase in intracellular sodium levels. The decreased transmembrane sodium gradient reduces the forward activity of the sodium-calcium exchanger, leading to a secondary **decrease in calcium efflux** from the cells. Increased intracellular calcium concentration stimulates the binding of calcium to troponin C and subsequent actin-myosin cross-bridge formation, resulting in **improved myocyte contractility** and left ventricular systolic function.

(Choice B) The increase in intracellular sodium caused by digoxin reduces the forward activity of the sodium-calcium exchanger, resulting in increased intracellular calcium concentration. However, the calcium increase is a secondary effect of digoxin that occurs after the increase in intracellular sodium.

(Choice C) Cyclic AMP (cAMP), an important second messenger in excitation-contraction coupling, is formed from ATP by adenylyl cyclase. Adenylyl cyclase activity is stimulated by beta agonists and inhibited by acetylcholine. cAMP increases the conductance of the calcium channels in the sarcoplasmic reticulum. As a result, more calcium is available to bind to troponin C, strengthening the force of contraction.

(Choice D) Inhibition of the Na-K-ATPase pump by digoxin leads to a decrease in potassium influx into myocardial cells.

(Choice E) Digoxin does not cause increased troponin sensitivity to calcium; instead, it increases the amount of intracellular calcium available for troponin binding.

Educational objective:
Digoxin directly inhibits the Na-K-ATPase pump in myocardial cells, leading to a decrease in sodium efflux and an increase in intracellular sodium levels. This reduces the forward activity of the sodium-calcium exchanger, causing increased intracellular calcium concentration and improved myocyte contractility.

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A 66-year-old man comes to the office for a follow-up appointment. The patient has a history of heart failure with reduced ejection fraction and has had persistent dyspnea despite taking the maximal dosage of his heart failure medications. He has had no lightheadedness or chest pain. Blood pressure is 133/72 mm Hg and pulse is 76/min. Physical examination shows normal lung sounds and no lower extremity edema. His medical treatment is modified with the addition of a combination pill that inhibits neprilysin and blocks angiotensin II receptors. Which of the following is the most likely effect of this medication?

- A. Decreased renal free-water reabsorption
- B. Decreased venous compliance
- C. Increased myocardial contractility
- D. Increased peripheral arterial resistance
- E. Increased urinary sodium excretion

Submit

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- ✗ A. Decreased renal free-water reabsorption (19%)
- B. Decreased venous compliance (2%)
- C. Increased myocardial contractility (5%)
- D. Increased peripheral arterial resistance (3%)
- ✓ E. Increased urinary sodium excretion (68%)

Incorrect
Correct answer
E

68%
Answered correctly

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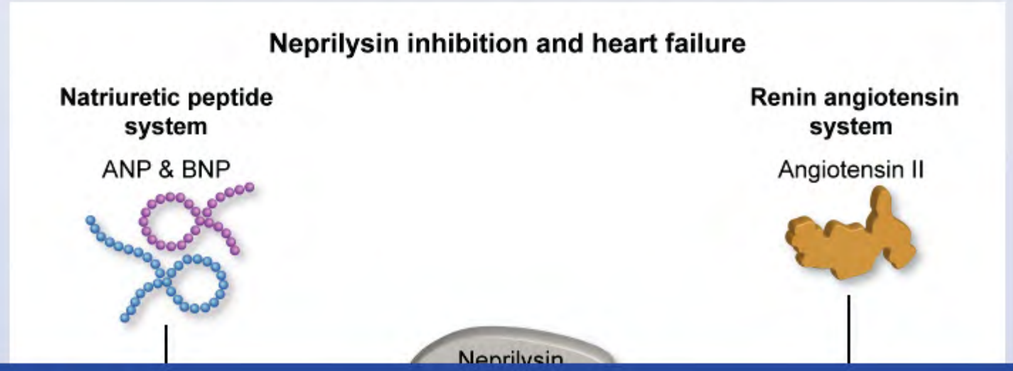
Explanation



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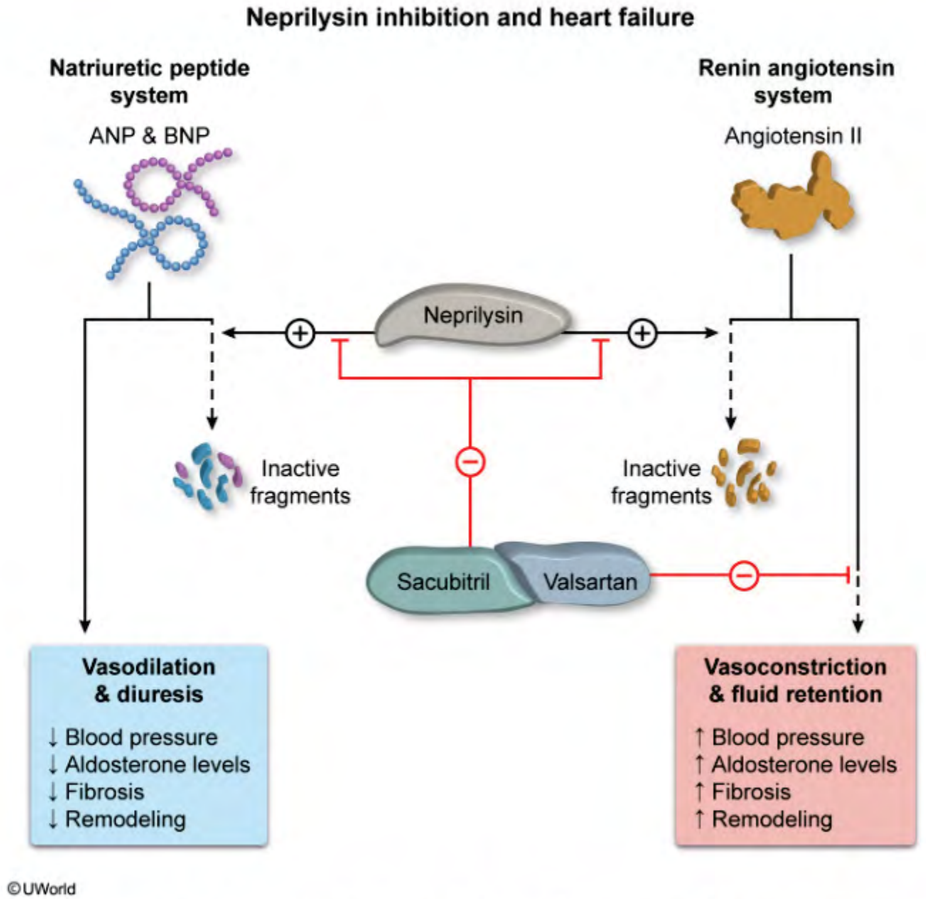

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Most pharmacologic therapies for **heart failure with reduced ejection fraction** (eg, beta blockers, ACE inhibitors, angiotensin II receptor blockers, aldosterone antagonists) are targeted toward the inhibition of detrimental neurohormonal pathways involving the sympathetic nervous system and renin-angiotensin-aldosterone system (RAAS). However, an additional strategy involves augmenting beneficial counter-regulatory mechanisms, such as those involving **natriuretic peptides**.

Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are released from the myocardium in response to high atrial and ventricular filling pressures (myocardial stretch). These peptides induce **vasodilation** and **diuresis** (excretion of salt and water) by antagonizing the actions of RAAS and also protect against the deleterious myocardial remodeling and fibrosis that occur in heart failure. Neprilysin is a metalloprotease that cleaves and inactivates both ANP and BNP. Therefore, medications that **inhibit neprilysin** (eg, sacubitril) lead to **increased levels of ANP and BNP** and promote beneficial effects in heart failure.

However, because neprilysin is also responsible for inactivating angiotensin II, inhibition of neprilysin further stimulates deleterious vasoconstriction and fluid retention via increased angiotensin II levels. Therefore, in treating heart failure, neprilysin inhibitors are **combined** with an **angiotensin II-receptor blocker** (eg, sacubitril-valsartan) to mitigate these negative effects.

(Choice A) Increased levels of ANP and BNP stimulate the excretion of both salt and water. Decreased reabsorption of free water (water without solute) can be achieved with medications that block vasopressin-2 receptors (eg, tolvaptan) on the renal collecting tubule.

(Choices B and D) The vasodilatory effects of ANP and BNP increase (rather than decrease) venous compliance and decrease (rather than increase) peripheral arterial resistance, leading to a reduction in both cardiac preload and afterload and a consequent reduction in cardiac work. These effects would be counteracted by angiotensin II-induced vasoconstriction, but angiotensin II activity is blocked by combining the neprilysin inhibitor with an angiotensin II-receptor blocker.

(Choice C) Neprilysin inhibition combined with angiotensin II receptor blockade improves cardiac output in heart failure via a reduction in the cardiac work required to attain a certain stroke volume; however, cardiac contractility is not significantly affected.

Educational objective:

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Educational objective:
Neprilysin is responsible for the breakdown of the natriuretic peptides and angiotensin II; therefore, inhibition of neprilysin increases the activity of these peptides. For treatment of heart failure, neprilysin inhibition is combined with angiotensin II receptor blockade to optimize the positive effects of the natriuretic peptides (eg, vasodilation, diuresis) while blocking the negative effects of angiotensin II (eg, vasoconstriction, fluid retention).

Physiology	Cardiovascular System	Chronic heart failure
Subject	System	Topic

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A 67-year-old man is brought to the emergency department due to progressive shortness of breath. He has had decreasing exercise tolerance for several months, and over the last week he could barely walk to the bathroom without becoming short of breath. The patient has been sleeping in a semirecumbent position recently. Medical history includes type 2 diabetes mellitus, hyperlipidemia, and a myocardial infarction 3 years ago. He takes no medications. The patient does not drink alcohol or use tobacco products. BMI is 30 kg/m^2. Oxygen saturation is 92% on room air. The apical impulse is palpated in the 6th intercostal space along the anterior axillary line. There is 3+ bilateral pitting edema in the legs. Which of the following is most likely increased in this patient?

- A. Arteriolar resistance
- B. Lung compliance
- C. Mixed venous O_2 content
- D. Renal perfusion
- E. Stroke volume

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- ✓ A. Arteriolar resistance (66%)
- B. Lung compliance (12%)
- C. Mixed venous O_2 content (7%)
- D. Renal perfusion (3%)
- E. Stroke volume (9%)

Correct

66%
Answered correctly

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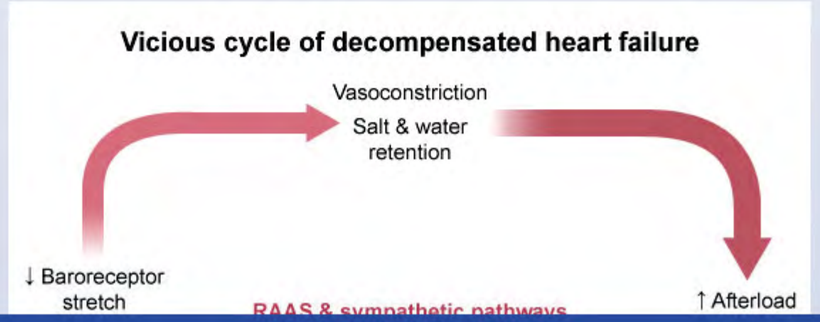
Explanation



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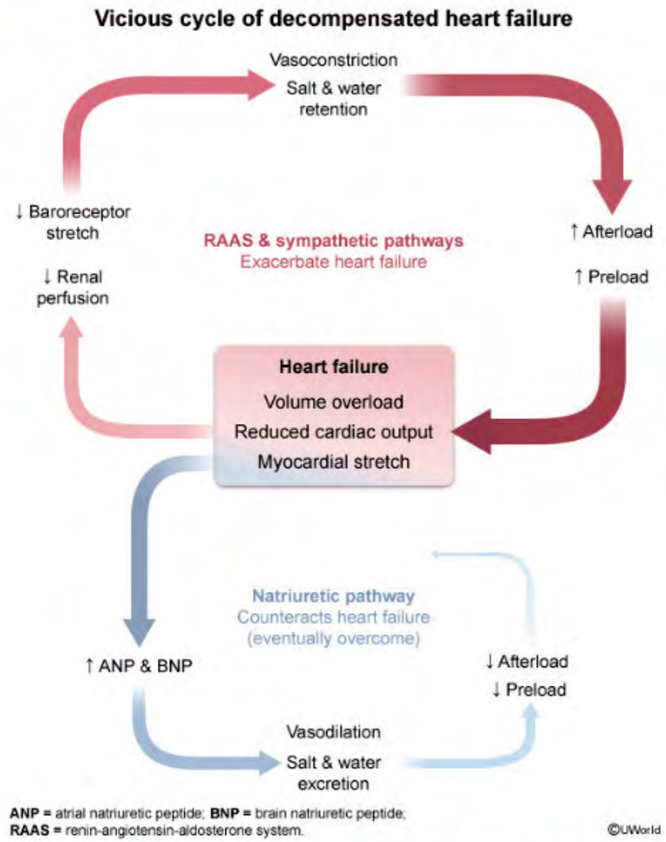

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↓ Baroreceptor stretch
RAAS & sympathetic pathways
↑ Afterload

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excretion

ANP = atrial natriuretic peptide; **BNP** = brain natriuretic peptide;
RAAS = renin-angiotensin-aldosterone system.

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This patient with progressive dyspnea, orthopnea, lower extremity swelling, and a laterally displaced point of maximal impulse has **decompensated heart failure**. Heart failure occurs due to either a progressive decline in myocardial contractility (systolic dysfunction) or impaired ability of the ventricular walls to relax during filling (diastolic dysfunction). Both etiologies eventually lead to **decreased cardiac output** and inadequate oxygen delivery to the tissues, inducing the following **neuroendocrine compensatory mechanisms** in an effort to restore organ perfusion:

- **Increased sympathetic output**: Baroreceptors sense decreased perfusion and stimulate epinephrine and norepinephrine release to increase heart rate and contractility. Vasoconstriction is also triggered, **increasing arteriolar resistance** and raising afterload.
- **Renin-angiotensin-aldosterone system (RAAS) activation**: Decreased renal perfusion **(Choice D)** leads to increased secretion of renin and increased circulating levels of angiotensin II, a potent vasoconstrictor that further increases afterload. Adrenal secretion of aldosterone also increases, which stimulates sodium and water retention and increases circulating blood volume (preload).

Although these compensatory mechanisms initially improve organ perfusion, they are overall **maladaptive**. A vicious cycle is generated as the failing heart is unable to pump against increased afterload, and volume retention creates venous congestion in the kidneys that reduces glomerular filtration rate and further impairs diuresis. Atrial and ventricular stretch triggers the release of natriuretic peptides that stimulate vasodilation and fluid diuresis to counteract the vicious cycle; however, these effects are outweighed by the downward-spiraling negative effects of sympathetic drive and RAAS activation.

(Choice B) Left ventricular failure leads to increased pulmonary venous pressure and transudation of fluid into the lung parenchyma, which stiffens the alveolar walls and decreases lung compliance.

(Choice C) With reduced cardiac output, the tissues become starved for O_2 and increase O_2 extraction from the available capillary blood. This leads to a reduction in mixed venous O_2 content.

(Choice E) Stroke volume is generally maintained early in heart failure (due to compensatory mechanisms) and

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(Choice C) With reduced cardiac output, the tissues become starved for O_2 and increase O_2 extraction from the available capillary blood. This leads to a reduction in mixed venous O_2 content.

(Choice E) Stroke volume is generally maintained early in heart failure (due to compensatory mechanisms) and decreases as decompensation takes place.

Educational objective:
Decreased cardiac output in heart failure triggers neuroendocrine compensatory mechanisms to maintain organ perfusion; however, the compensatory mechanisms are maladaptive over the long term. Increased sympathetic output and activation of the renin-angiotensin-aldosterone system stimulate vasoconstriction and volume retention, compounding the hemodynamic stress on an already failing heart and creating a vicious cycle of decompensation.

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↑ ANP & BNP

↓ Afterload
↓ Preload

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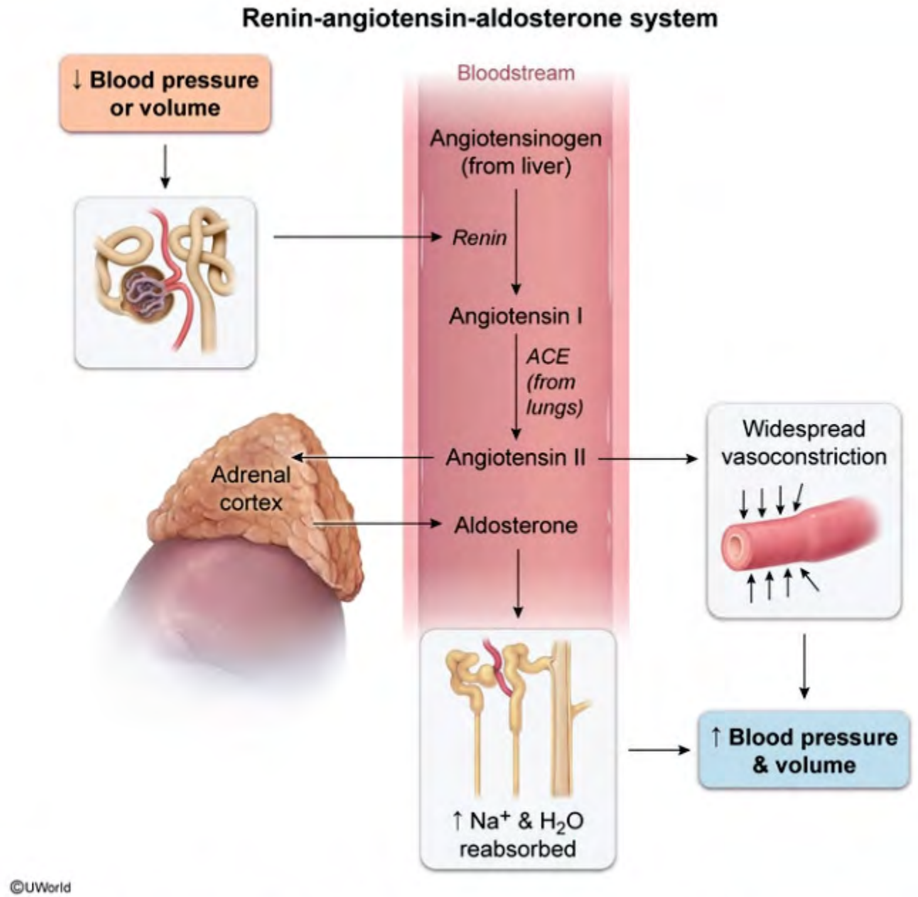

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lung parenchyma, which stiffens the alveolar walls and decreases lung compliance.

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A 74-year-old man is brought to the emergency department due to acute-onset lower abdominal and back pain that was followed by an episode of syncope 2 hours ago. The pain is sharp and constant. He has had no changes in bowel or bladder function. Other medical conditions include hypertension and hyperlipidemia. The patient has a 50-pack-year history of smoking cigarettes. Temperature is 36.9 C (98.4 F), blood pressure is 90/60 mm Hg, pulse is 108/min and regular, and respirations are 18/min. BMI is 34 kg/m^2. The patient is diaphoretic and appears pale. Heart and lung sounds are normal. Abdominal examination reveals moderate tenderness to deep palpation in the lower abdomen; there is no guarding or rebound tenderness. Bowel sounds are normal. Which of the following is the most likely diagnosis?

- A. Acute mesenteric ischemia
- B. Colonic perforation
- C. Renal colic
- D. Ruptured abdominal aortic aneurysm
- E. Vertebral compression fracture

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- ✗ A. Acute mesenteric ischemia (18%)
- B. Colonic perforation (2%)
- C. Renal colic (1%)
- ✓ D. Ruptured abdominal aortic aneurysm (75%)
- E. Vertebral compression fracture (1%)

Incorrect
Correct answer
D

75%
Answered correctly

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Explanation

Abdominal aortic aneurysm	
Risk factors	• Advanced age (>60) • Smoking, male sex, hypertension • History of atherosclerosis or CTD
	• Asymptomatic until rupture or rapid expansion

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Explanation

Abdominal aortic aneurysm	
Risk factors	• Advanced age (>60) • Smoking, male sex, hypertension • History of atherosclerosis or CTD

Clinical presentation	• Asymptomatic until rupture or rapid expansion
	• Rupture
	  ◦ Sudden, severe abdominal/back pain ± shock
	  ◦ Umbilical/flank hematoma

Management	• Smoking cessation • Periodic imaging to assess for expansion • Elective repair for large, asymptomatic aneurysm
CTD = connective tissue disease.	

Ruptured abdominal aortic aneurysm (AAA) is a surgical emergency involving full-thickness compromise of the aortic wall with extravasation of blood into surrounding tissues and spaces. The **acute onset** of **severe abdominal and back pain** is the most common presenting symptom. In general, anterior rupture into the **peritoneal cavity** is quickly accompanied by **syncope**, hypotension, and **shock**, whereas posterior rupture into the retroperitoneum may be temporarily contained, resulting in delayed onset of hemodynamic instability. Other signs suggesting AAA rupture include abdominal distension, a pulsatile abdominal mass, and umbilical or flank hematoma (indicators of retroperitoneal hemorrhage).

Early detection of AAA can be difficult because patients frequently have few symptoms until the AAA either ruptures or markedly expands. Therefore, ruptured AAA should be suspected in patients with consistent symptoms and known **risk factors**, which include advanced age (>60), **smoking**, male sex, and a history of atherosclerosis or connective tissue disease. CT imaging can confirm the diagnosis, but hemodynamically unstable patients with suspected ruptured AAA should go straight to emergent surgical repair.

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(Choice A) Acute mesenteric ischemia has similar risk factors to AAA. Patients typically describe severe abdominal pain but have few findings on physical examination (eg, minimal tenderness) and hypotension and syncope are not usually seen.

(Choice B) Colonic perforation can be due to diverticulitis, malignancy, or complications from colonoscopy. Patients can develop hypotension due to sepsis, but signs of infection (eg, fever) and peritonitis (eg, rebound, guarding) are also expected.

(Choice C) Renal colic is characterized by episodic (eg, every 30 min) flank or groin pain due to capsular stretch from nephrolithiasis-associated obstruction. Hypotension and syncope are not typical features.

(Choice E) Vertebral compression fractures can cause acute back pain exacerbated by minimal movement (eg, coughing, bending). Although the pain can occasionally refer to the abdomen, it is most commonly at the midline over the spinous processes. There should not be associated hypotension.

Educational objective:
Ruptured abdominal aortic aneurysm is a surgical emergency that usually presents with the acute onset of severe abdominal and back pain in patients with appropriate risk factors (eg, advanced age, smoking, atherosclerosis). Accompanying syncope, hypotension, and shock may occur quickly (intraperitoneal rupture) or may be delayed

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peritoneal cavity is quickly accompanied by **syncope**, hypotension, and **shock**, whereas posterior rupture into the retroperitoneum may be temporarily contained, resulting in delayed onset of hemodynamic instability. Other signs suggesting AAA rupture include abdominal distension, a pulsatile abdominal mass, and umbilical or flank hematoma (indicators of retroperitoneal hemorrhage).

Early detection of AAA can be difficult because patients frequently have few symptoms until the AAA either ruptures or markedly expands. Therefore, ruptured AAA should be suspected in patients with consistent symptoms and known **risk factors**, which include advanced age (>60), **smoking**, male sex, and a history of atherosclerosis or connective tissue disease. CT imaging can confirm the diagnosis, but hemodynamically unstable patients with suspected ruptured AAA should go straight to emergent surgical repair.

(Choice A) Acute mesenteric ischemia has similar risk factors to AAA. Patients typically describe severe abdominal pain but have few findings on physical examination (eg, minimal tenderness) and hypotension and syncope are not usually seen.

(Choice B) Colonic perforation can be due to diverticulitis, malignancy, or complications from colonoscopy. Patients can develop hypotension due to sepsis, but signs of infection (eg, fever) and peritonitis (eg, rebound, guarding) are also expected.

(Choice C) Renal colic is characterized by episodic (eg, every 30 min) flank or groin pain due to capsular stretch from nephrolithiasis-associated obstruction. Hypotension and syncope are not typical features.

(Choice E) Vertebral compression fractures can cause acute back pain exacerbated by minimal movement (eg, coughing, bending). Although the pain can occasionally refer to the abdomen, it is most commonly at the midline over the spinous processes. There should not be associated hypotension.

Educational objective:
Ruptured abdominal aortic aneurysm is a surgical emergency that usually presents with the acute onset of severe abdominal and back pain in patients with appropriate risk factors (eg, advanced age, smoking, atherosclerosis). Accompanying syncope, hypotension, and shock may occur quickly (intraperitoneal rupture) or may be delayed (retroperitoneal rupture).

References

- Abdominal aortic aneurysm

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Ruptured abdominal aortic aneurysm (AAA) is a surgical emergency involving full-thickness compromise of the aortic wall with extravasation of blood into surrounding tissues and spaces. The **acute onset of severe abdo**
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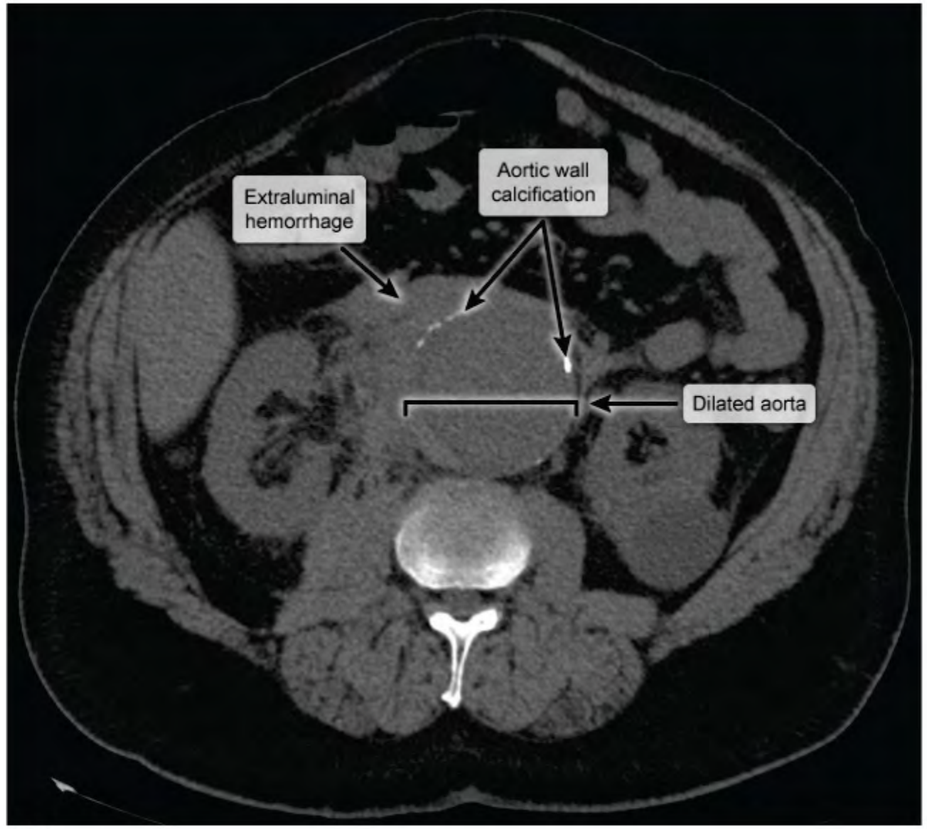
Ruptured abdominal aortic aneurysm



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A 40-year-old female with a history of depression and hypertension is brought to the ER after being found obtunded in her apartment. She is hypotensive and bradycardic on physical examination. Intravenous glucagon is administered, and her condition improves. Which of the following intracellular changes is most likely responsible for the improvement in her condition?

- A. Increased synaptic release of glutamate
- B. Decreased cAMP in vascular smooth muscle
- C. Decreased DAG in vascular smooth muscle
- D. Increased cAMP in cardiac myocytes
- E. Increased IP_3 in cardiac myocytes

Submit

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- A. Increased synaptic release of glutamate (3%)
- ✗ B. Decreased cAMP in vascular smooth muscle (12%)
- C. Decreased DAG in vascular smooth muscle (3%)
- ✓ D. Increased cAMP in cardiac myocytes (67%)
- E. Increased IP_3 in cardiac myocytes (12%)

Incorrect
Correct answer
D

67%
Answered correctly

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Explanation

This patient has most likely overdosed on beta blocker medications. Beta blocker overdose causes diffuse non-selective blockade of peripheral beta adrenergic receptors, causing depression of myocardial contractility, bradycardia, and varying degrees of AV block. The result is a low cardiac output state.

Glucagon is the drug of choice for beta blocker overdose. Glucagon acts on G protein-coupled receptors, increasing intracellular cAMP and thus increasing the release of intracellular calcium during muscle contraction. This increases heart rate and cardiac contractility. Improvements in heart rate and blood pressure may be observed within minutes.

Educational Objective:
Patients who have overdosed on beta blockers should be treated with glucagon, which increases heart rate and contractility independent of adrenergic receptors. Glucagon activates G-protein-coupled receptors on cardiac

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- B. Decreased cAMP in vascular smooth muscle (12%)
- C. Decreased DAG in vascular smooth muscle (3%)
- D. Increased cAMP in cardiac myocytes (67%)
- E. Increased IP_3 in cardiac myocytes (12%)

Incorrect
Correct answer
D

67%
Answered correctly

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Educational Objective:
Patients who have overdosed on beta blockers should be treated with glucagon, which increases heart rate and contractility independent of adrenergic receptors. Glucagon activates G-protein-coupled receptors on cardiac myocytes, causing activation of adenylate cyclase and raising intracellular cAMP. The result is calcium release from intracellular stores and increased sinoatrial node firing.

Pharmacology	Cardiovascular System	Beta blocker poisoning
Subject	System	Topic

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A 60-year-old man comes to the emergency department with dizziness and palpitations. He woke up this morning with an intermittent pounding sensation in his chest. The patient has occasionally experienced a similar sensation in the past and did not think it was serious. Later, while mowing the lawn, he became lightheaded and had to sit down to avoid passing out. After receiving the appropriate workup, the patient is diagnosed with a cardiac arrhythmia and started on a medication known to prolong the QT interval. However, the drug is associated with a lower incidence of torsade de pointes than other QT-prolonging agents. Which of the following medications was most likely used in this patient?

- A. Adenosine
- B. Amiodarone
- C. Digoxin
- D. Esmolol
- E. Lidocaine
- F. Procainamide
- G. Verapamil

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- A. Adenosine (5%)
- ✓ B. Amiodarone (48%)
- C. Digoxin (3%)
- D. Esmolol (5%)
- E. Lidocaine (7%)
- F. Procainamide (20%)
- G. Verapamil (7%)

Correct

48%
Answered correctly

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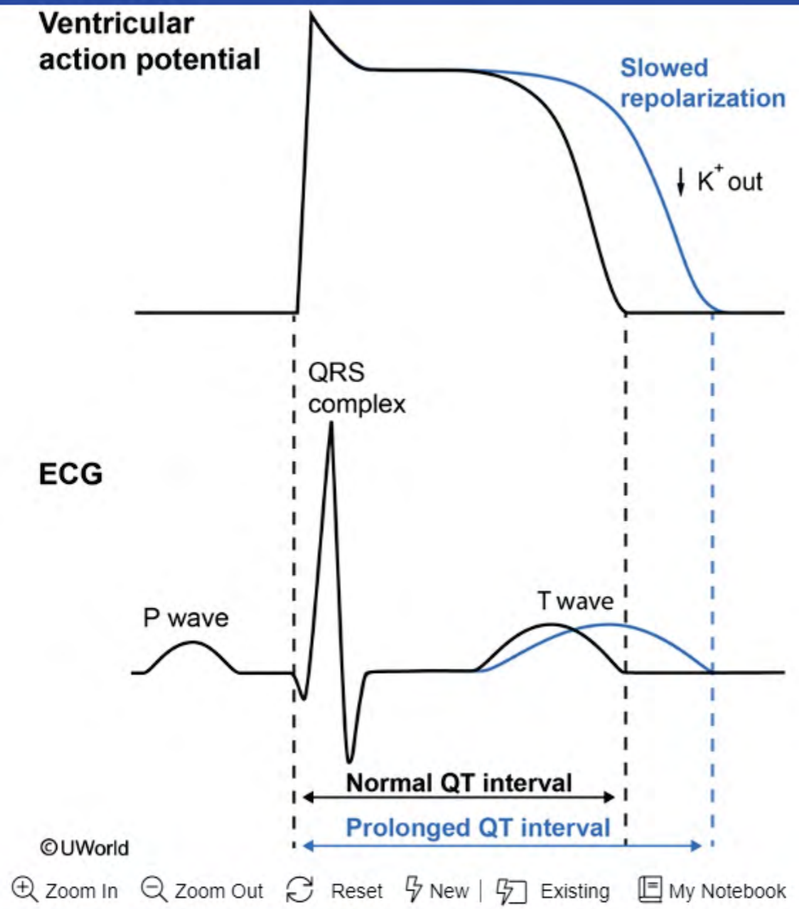

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Amiodarone is a class III antiarrhythmic drug used for the management of a variety of supraventricular and ventricular arrhythmias. Class III drugs predominantly block **potassium channels** and inhibit the outward potassium currents during phase 3 of the cardiac action potential, prolonging repolarization and total action potential duration (APD).

The **QT interval** represents the time required for ventricular depolarization and repolarization; it can be regarded as a rough estimate of the APD. Drugs that significantly prolong the cardiac action potential (eg, class IA and III antiarrhythmics) cause prolongation of the QT interval. Prolonged QT is associated with a form of ventricular tachycardia called torsades de pointes (polymorphic ventricular tachycardia). However, unlike other drugs that cause QT prolongation, amiodarone has **very little risk** of inducing torsades de pointes. This effect is thought to be due to it having a more homogeneous effect on ventricular repolarization compared to other drugs (ie, less QT dispersion).

(Choice A) Adenosine interacts with A1 receptors on cardiac cells and activates potassium channels, increasing potassium conductance. It causes a transient conduction delay through the atrioventricular (AV) node and is used for acute termination of paroxysmal supraventricular tachycardia (PSVT). Adenosine is rapidly metabolized in the blood and tissues and has a very brief duration of action.

(Choice C) Digoxin slows AV nodal conduction speed by augmenting vagal parasympathetic tone and increases cardiac contractility by inhibiting Na^+/K^+-ATPase (increases intracellular calcium). It also decreases the APD and can cause QT interval shortening.

(Choice D) Esmolol is a rapidly acting, short-duration beta blocker (class II antiarrhythmic) that slows the rate of discharge of sinus or ectopic pacemakers and increases the refractory period of the AV node. It has no effect on QT interval.

(Choice E) Lidocaine is a class IB antiarrhythmic drug that acts by blocking sodium channels and inhibiting phase 0 depolarization. It does not prolong the QT interval.

(Choice F) Procainamide is a class IA antiarrhythmic drug that inhibits sodium-dependent (phase 0) depolarization. It also has moderate potassium channel blocking activity, leading to prolongation of APD and the QT interval with an increased risk of torsades de pointes.

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(Choice F) Procainamide is a class IA antiarrhythmic drug that inhibits sodium-dependent (phase 0) depolarization. It also has moderate potassium channel blocking activity, leading to prolongation of APD and the QT interval with an increased risk of torsades de pointes.

(Choice G) Verapamil is a cardio-selective calcium channel blocker (class IV antiarrhythmic drug). It slows the sinus rate and prolongs the PR interval by slowing conduction through the AV node. It does not prolong the QT interval.

Educational objective:
Amiodarone (and other class III and class IA antiarrhythmic agents) causes lengthening of the cardiac action potential, which manifests as QT interval prolongation on ECG. QT prolongation caused by amiodarone, in contrast to other drugs, is associated with a very low risk of torsades de pointes.

References

- Chronic amiodarone evokes no torsade de pointes arrhythmias despite QT lengthening in an animal model of

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Antiarrhythmic drugs – Vaughan Williams classification

Class	Drugs	Predominant actions
IA	Sodium channel blockers • Disopyramide • Procainamide • Quinidine	• Slows AP conduction velocity • Prolongs APD
IB	• Lidocaine • Mexiletine • Phenytoin	• No effect on AP conduction velocity • Shortens APD
IC	• Flecainide • Propafenone	• Slows AP conduction velocity • Minimal effect on APD
II	Beta receptor blockers • Atenolol • Metoprolol • Esmolol • Carvedilol	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness
III	Potassium channel blockers • Amiodarone • Dronedarone • Dofetilide • Sotalol (also class II)	• No effect on AP conduction velocity • Prolongs APD
IV	Non-dihydropyridine calcium channel blockers • Verapamil • Diltiazem	• Slows sinus node discharge rate • Slows AV nodal conduction & prolongs refractoriness

AP = action potential; **APD** = action potential duration; **AV** = atrioventricular.

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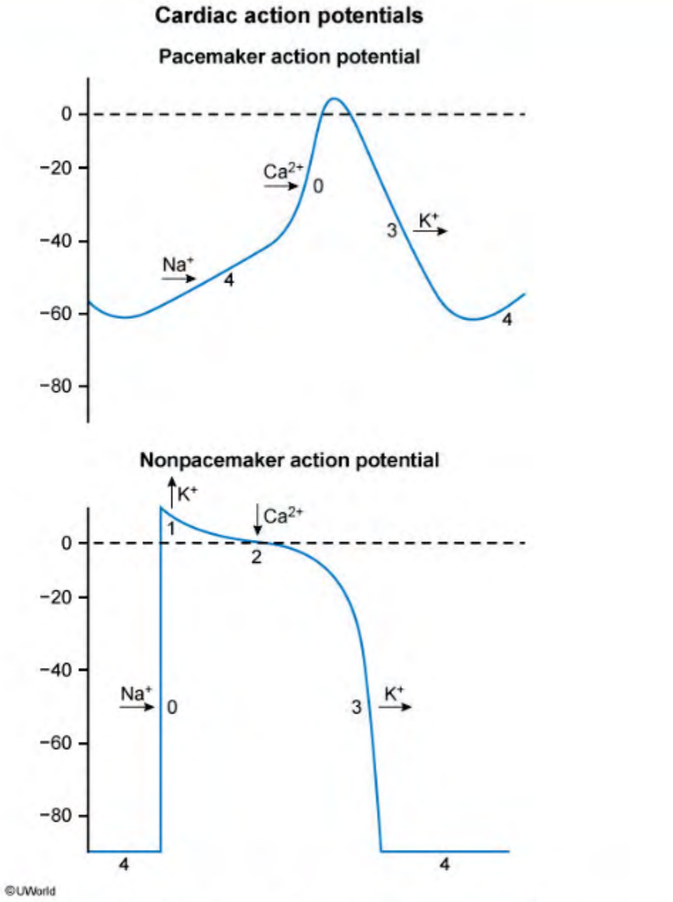

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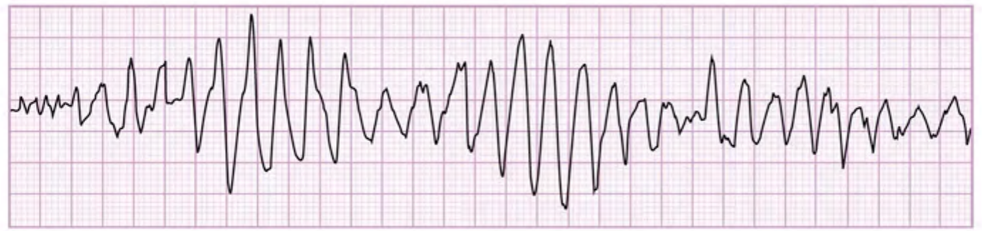

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A 34-year-old woman is brought to the emergency department due to chest pain. The patient reports that she went to bed feeling well and woke up in the middle of the night with severe, crushing chest pain that radiated to her left arm. She has had similar episodes over the past year that typically occur in early morning and spontaneously resolve after several minutes. The patient has a history of occasional migraine that responds to acetaminophen. She does not use tobacco, alcohol, or illicit drugs. Blood pressure is 150/100 mm Hg, pulse is 110/min, and respirations are 14/min. On physical examination, the patient appears distressed and is diaphoretic. ECG reveals ST-segment elevations in leads I, aVL, and V4-V6. The patient is given nitroglycerin, which improves her symptoms. Percutaneous coronary angiography is performed, and no obstructive lesions are observed. Which of the following is most likely to be involved in the pathogenesis of this patient's chest pain?

- A. Decreased endothelial expression of endothelin-1
- B. Granulomatous inflammation of arterial media
- C. Hyperreactivity of vascular smooth muscle
- D. Increased endothelial expression of prostacyclin
- E. Ulceration of thin atheromatous fibrous cap

Submit

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- ✗ A. Decreased endothelial expression of endothelin-1 (3%)
- B. Granulomatous inflammation of arterial media (4%)
- ✓ C. Hyperreactivity of vascular smooth muscle (83%)
- D. Increased endothelial expression of prostacyclin (3%)
- E. Ulceration of thin atheromatous fibrous cap (5%)

Incorrect
Correct answer
C

83%
Answered correctly

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Time Spent

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Explanation

Vasospastic angina	
Pathogenesis	• Hyperreactivity of coronary smooth muscle due to endothelial dysfunction & autonomic imbalance

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Vasospastic angina	
Pathogenesis	• Hyperreactivity of coronary smooth muscle due to endothelial dysfunction & autonomic imbalance

Clinical presentation	• Young age (<50)
	• Minimal CAD risk factors (other than smoking)
	• Recurrent chest discomfort
	  ◦ At rest or during sleep
	  ◦ Spontaneous resolution ≤15 min

Diagnosis	• ECG: ST-segment elevation during episode • Coronary angiography: no CAD
CAD = coronary artery disease.	

This patient most likely has **vasospastic angina** (formerly Prinzmetal angina), which involves **intermittent coronary artery vasospasm** leading to myocardial ischemia. The condition likely results from hyperreactivity of arterial smooth muscle due to a combination of **endothelial dysfunction** and **autonomic imbalance**.

The major underlying defect in vasospastic angina is likely a deficiency of vasodilatory nitric oxide in the coronary artery endothelium, causing acetylcholine released from vagus nerve terminals to trigger vasoconstriction rather than vasodilation. Patients typically experience episodes of **recurrent chest discomfort** that spontaneously **resolve within 15 minutes**. The episodes often occur at rest or **during sleep**, when vagal tone is at a peak. As with typical angina, there may be associated diaphoresis, nausea, and dyspnea, but unlike typical angina, patients are usually young (age <50) and lack most risk factors for atherosclerotic coronary artery disease (CAD) (eg, hypertension, diabetes). However, smoking is a strong risk factor, likely due to its contribution to endothelial dysfunction.

Vasospastic angina is usually diagnosed by ambulatory ECG (Holter monitor) that shows **ST elevation** during an

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Vasospastic angina is usually diagnosed by ambulatory ECG (Holter monitor) that shows **ST elevation** during an episode of chest discomfort, followed by coronary angiography to rule out atherosclerotic CAD. Sublingual nitroglycerin is effective in relieving an active episode of vasospastic angina, and calcium channel blockers (eg, diltiazem, amlodipine) are effective in preventing episodes.

(Choices A and D) Endothelin-1 is a potent vasoconstrictor and prostacyclin is a vasodilator. Therefore, decreased expression of endothelin-1 or increased expression of prostacyclin would stimulate coronary vasodilation rather than vasospasm.

(Choice B) Granulomatous inflammation of arterial media occurs in Takayasu arteritis, a large-vessel vasculitis affecting the aorta and its major branches. Young women of Asian descent are most commonly affected and sometimes develop angina due to coronary artery involvement; however, limb claudication (due to subclavian artery involvement) accompanied by constitutional symptoms (eg, fever, fatigue) is a more common presentation.

(Choice E) Ulceration of the thin fibrous cap overlying an atherosclerotic plaque can lead to unstable angina, characterized by intermittent chest discomfort similar to that of vasospastic angina. However, atherosclerotic CAD

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(Choice E) Ulceration of the thin fibrous cap overlying an atherosclerotic plaque can lead to unstable angina, characterized by intermittent chest discomfort similar to that of vasospastic angina. However, atherosclerotic CAD is unlikely in this young woman without significant CAD risk factors.

Educational objective:
Vasospastic angina involves hyperreactivity of coronary artery smooth muscle. Patients are usually young (age <50) and without significant risk factors for coronary artery disease; they experience recurrent episodes of chest discomfort that typically occur during rest or sleep and resolve within 15 minutes.

Pathophysiology Cardiovascular System Vasospastic angina

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A 51-year-old man is brought to the emergency department due to chest tightness that started 30 minutes prior to arrival. His chest discomfort is associated with shortness of breath and nausea. The patient was shoveling snow off his driveway when his symptoms began. He has a history of hypertension and type 2 diabetes mellitus. Initial ECG shows ST elevation in leads I and aVL. Cardiac enzymes are elevated. Emergent cardiac catheterization in this patient will most likely show occlusion of which of the following arteries?

- A. Distal left anterior descending artery
- B. Left circumflex artery
- C. Left main coronary artery
- D. Proximal left anterior descending artery
- E. Right coronary artery

Submit

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- ✗ A. Distal left anterior descending artery (13%)
- ✓ B. Left circumflex artery (55%)
- C. Left main coronary artery (5%)
- D. Proximal left anterior descending artery (14%)
- E. Right coronary artery (11%)

Incorrect
Correct answer
B

55%
Answered correctly

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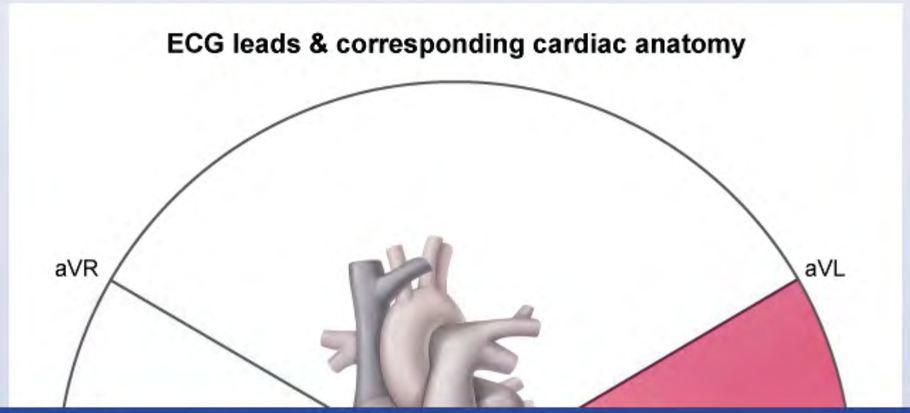
Explanation



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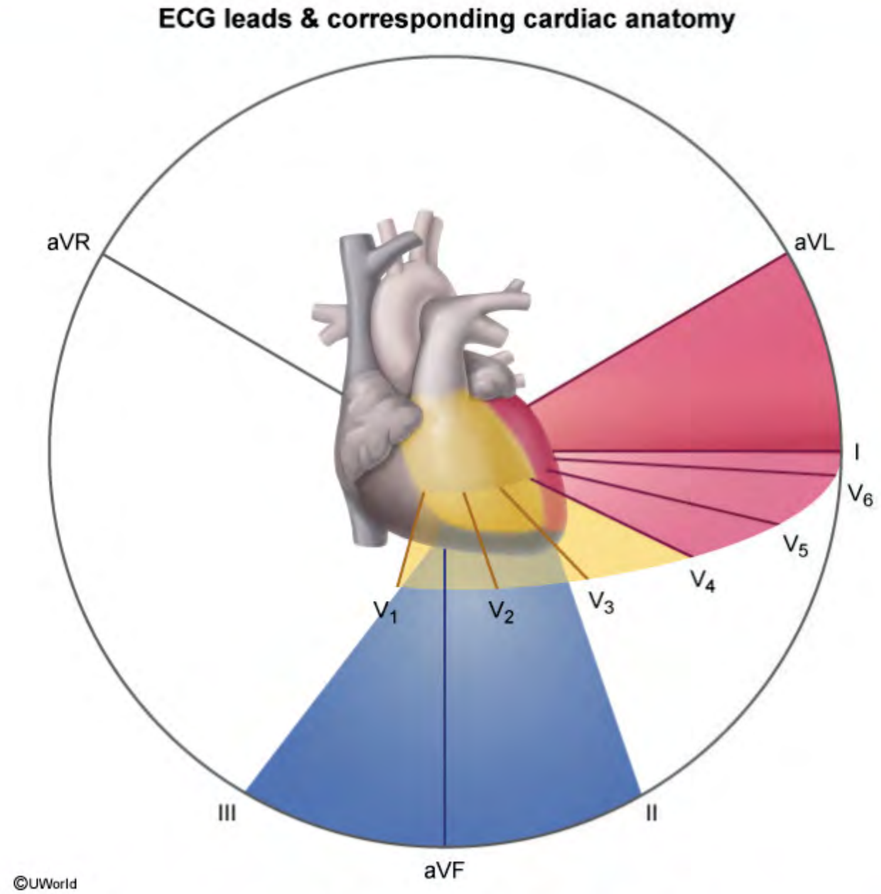

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aVF

This patient with **ST elevations** in leads I and aVL most likely has an acute lateral **myocardial infarction** (MI) due to occlusion of the left circumflex artery. ECGs are useful in the diagnosis of MI and may help localize the area of infarction. ST elevations typically represent acute MI, whereas prominent Q waves are suggestive of old MI.

Leads **I and aVL** correspond to the **lateral limb leads** on ECG; therefore, ST elevation or Q waves in these leads are indicative of infarction involving the lateral aspect of the left ventricle. Because the chest leads V5-V6 are also laterally placed, they may also show ST elevation during a lateral infarction. The lateral aspect of the left ventricle is supplied by the **left circumflex artery**, which originates from the left main coronary artery.

(Choices A and D) The left anterior descending artery (LAD) primarily supplies the anterior aspect of the left ventricle and interventricular septum (septal branches), which corresponds to the anterior chest leads (V1-V4). Proximal occlusion of the LAD may involve all of these leads; however, distal LAD occlusion typically spares the septal leads (V1-V2).

(Choice C) The left main coronary artery gives rise to the LAD and left circumflex arteries. Therefore, left main coronary artery occlusion typically results in anterolateral infarction, which corresponds to ST elevation in the anterior (eg, V1-V4) and lateral (V5-V6, I, and aVL) leads.

(Choice E) The right coronary artery typically supplies the right ventricle and inferior aspect of the left ventricle. Occlusion of this vessel therefore results in inferior MI, which corresponds to ST elevation in the inferior leads (II, III, and aVF).

Educational objective:
Leads I and aVL correspond to the lateral limb leads on ECG. Therefore, ST elevation or Q waves in these leads are indicative of infarction involving the lateral aspect of the left ventricle, which is supplied by the left circumflex artery.

References

- The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis.
- Electrocardiographic localization of coronary artery narrowings: studies during myocardial ischemia and infarction in patients with one vessel disease

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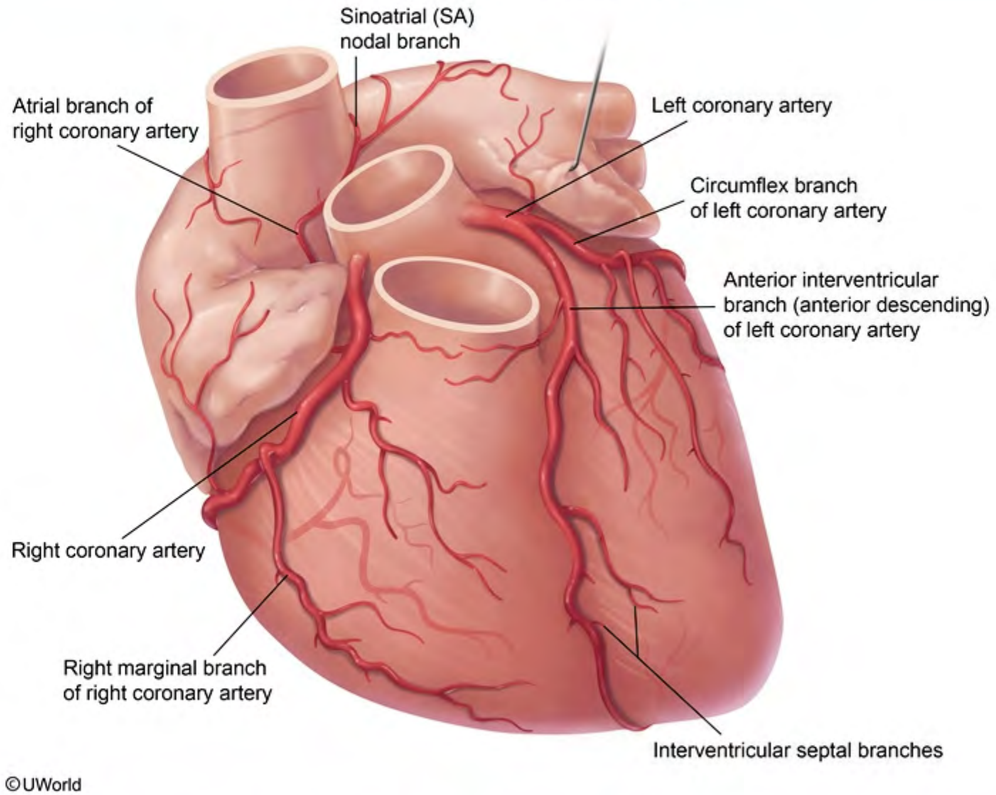
Coronary arteries, anterior view | Coronary Arteries

Coronary arteries: anterior view



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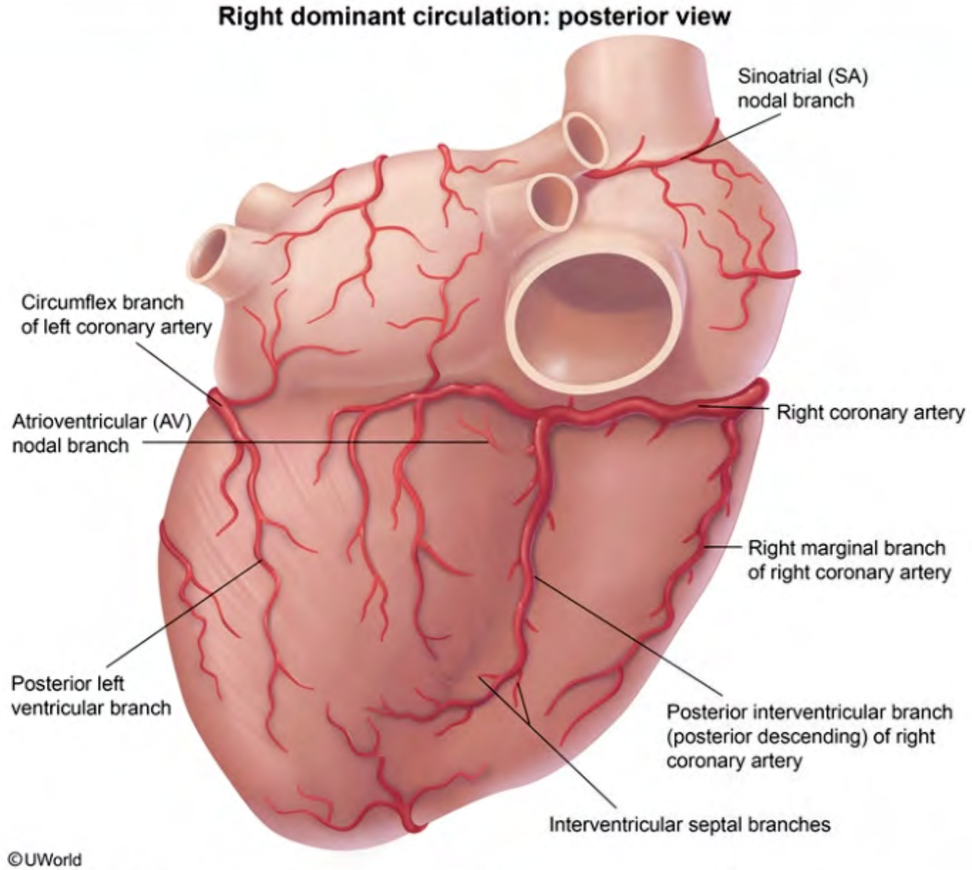

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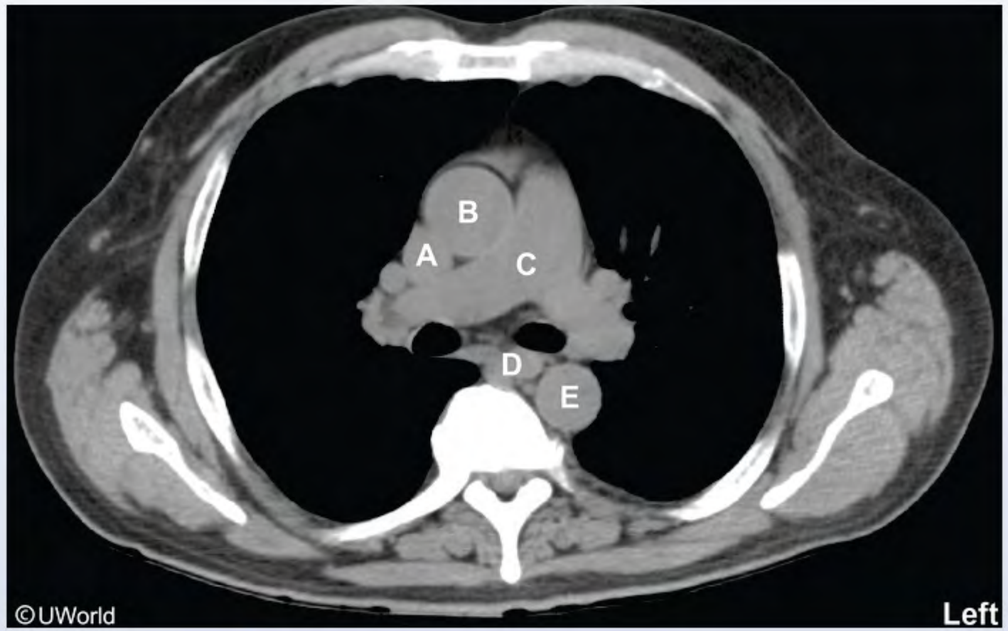
A 55-year-old man is brought to the emergency department due to shortness of breath, productive cough, and confusion. His wife mentions that last week he was diagnosed with the flu and his symptoms have progressively worsened. Temperature is 39.4 C (103 F), blood pressure is 80/50 mm Hg, pulse is 120/min, and respiratory rate is 22/min. Pulse oximetry shows 86% on room air. On physical examination, the patient is obtunded and in respiratory distress. He is intubated, given a 2-L bolus of normal saline, and started on broad spectrum antibiotics after blood cultures are obtained. A central line is placed to access a structure embryonically derived from the common cardinal veins. This structure is represented by which of the following labels shown in the chest CT scan below?



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- B. B
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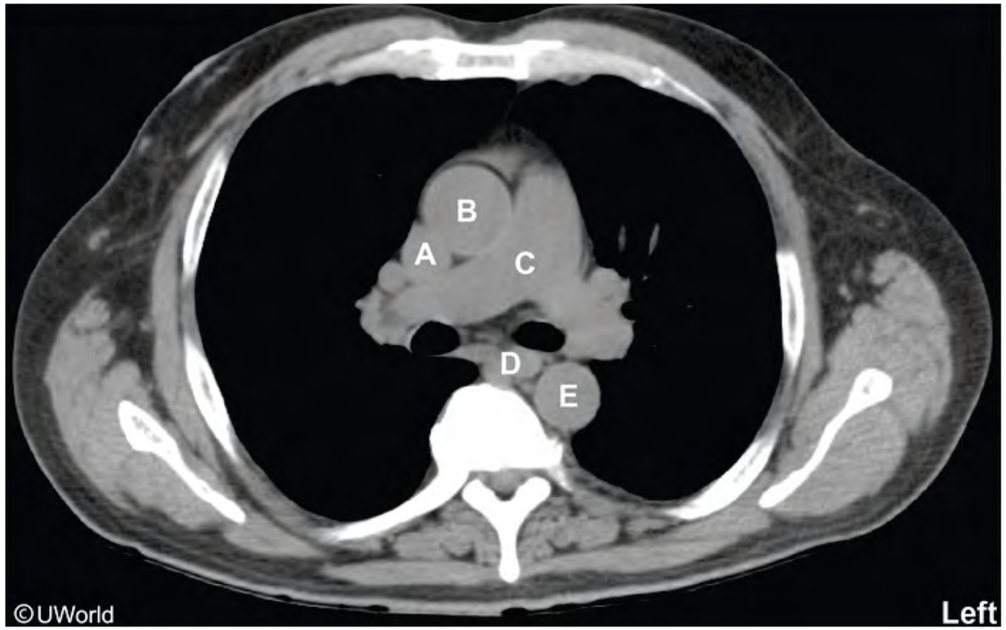

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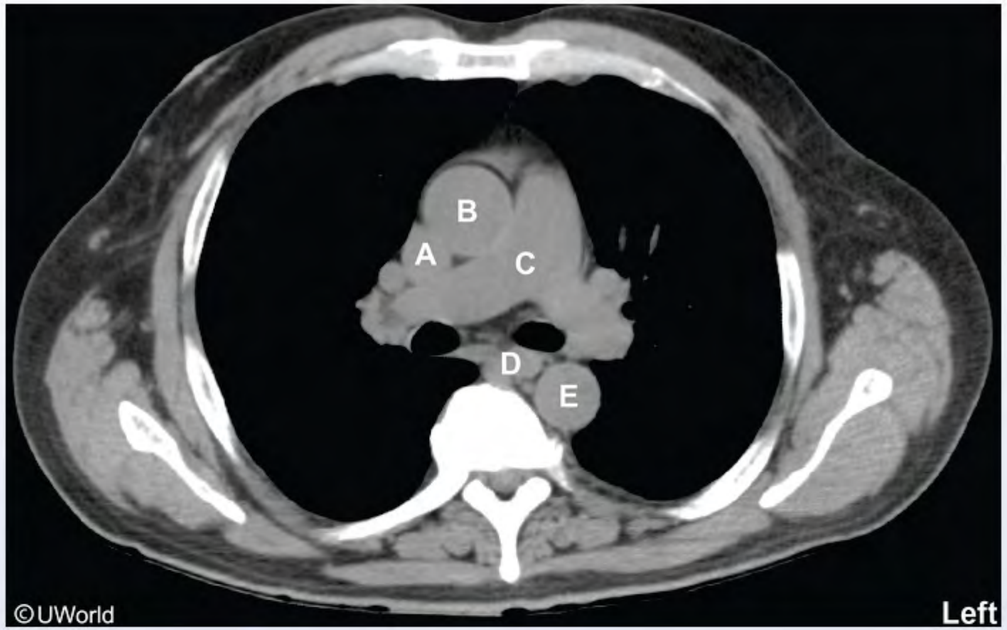

- A. A
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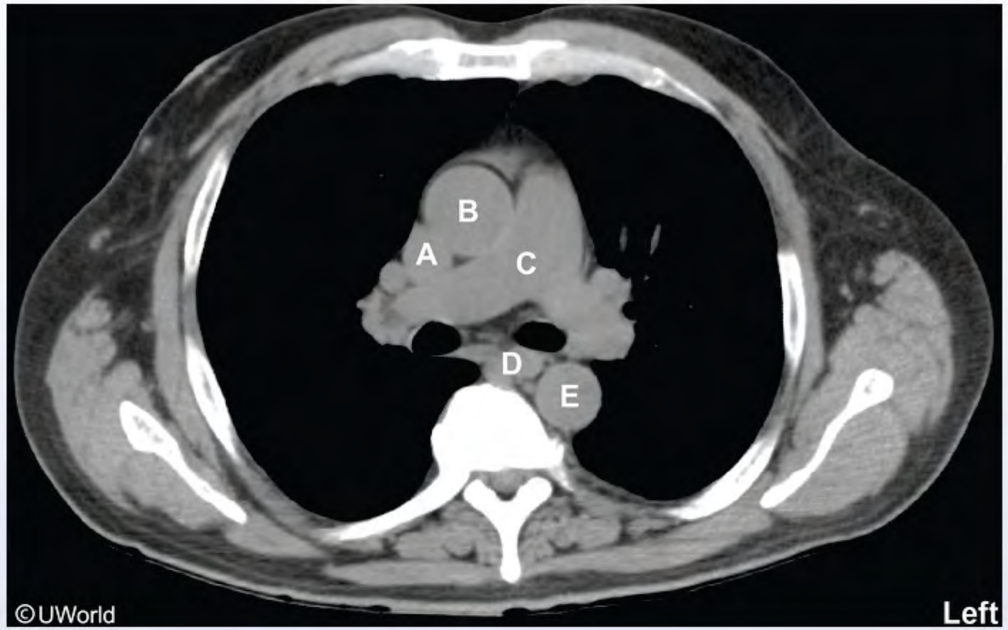

- ✓ A. A (51%)
- B. B (8%)
- C. C (12%)
- D. D (13%)
- E. E (13%)

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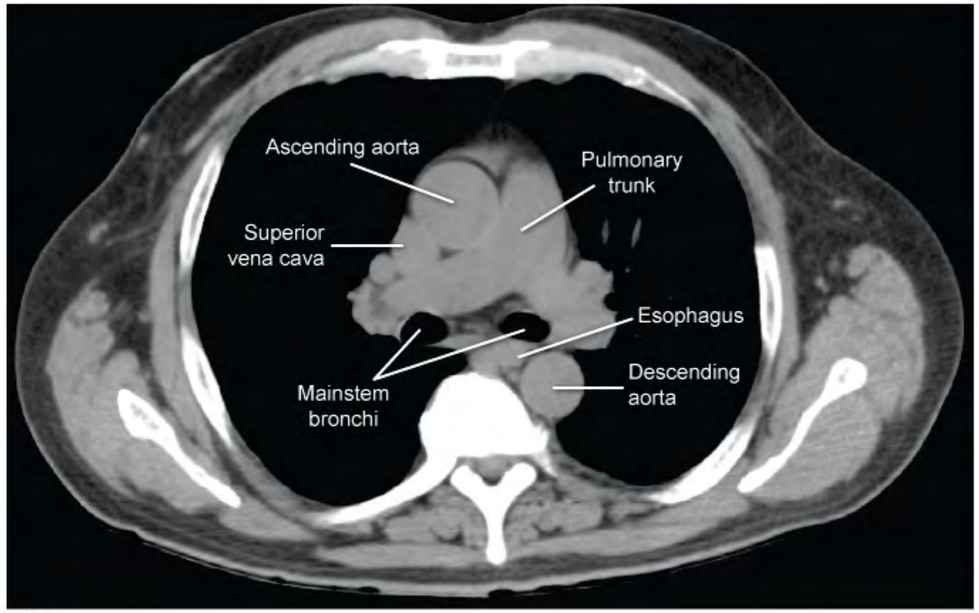
Normal chest CT



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Patients with hemodynamic instability or shock often have **central lines** placed into the central venous system to allow rapid infusion of intravenous fluids, blood, and vasopressors. The catheter is usually inserted into the neck (internal jugular vein) or chest (subclavian vein), and is advanced until the catheter tip enters the **superior vena cava** (SVC). The SVC is derived from the common cardinal veins. On CT scan imaging of the upper thorax, the SVC can be identified to the right of the heart, posterolateral to the ascending aorta, anterior to the right pulmonary artery, and just below the level of the carina.

All veins in the developing embryo ultimately drain into the sinus venosus, which drains into the primitive atrium of the developing heart. In early embryonic development, the body's veins fall into 3 main groups: umbilical, vitelline, and **cardinal veins**. The umbilical vein degenerates, the vitelline veins form the portal system, and the cardinal veins form constituents of the **systemic venous circulation**.

(Choice B) The ascending aorta develops from the truncus arteriosus, which also gives rise to the pulmonary trunk. A portion of the aortic arch develops from the fourth branchial (pharyngeal) arch.

(Choice C) The pulmonary trunk is derived from the truncus arteriosus. Neural crest cell migration helps partition the truncus arteriosus into the 2 great arteries (aorta and pulmonary artery) by causing fusion and twisting of the truncal and bulbar ridges. This process results in the normal spiral relation between the aorta and pulmonary artery. Failure of this process results in transposition of the great vessels and tetralogy of Fallot.

(Choice D) The esophagus is embryologically derived from the endodermal foregut. In the thoracic cavity, it descends posterior to the trachea and anterior to the vertebrae.

(Choice E) The descending aorta is derived from fusion of the embryonic right and left dorsal aortas. In the thoracic cavity, it starts left of the vertebral column and approaches the midline as it descends toward the diaphragm.

Educational objective:
The common cardinal veins of the developing embryo drain directly into the sinus venosus. These cardinal veins ultimately give rise to the superior vena cava and other constituents of the systemic venous circulation.

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References

- Misplaced central venous catheters: Applied anatomy and practical management.

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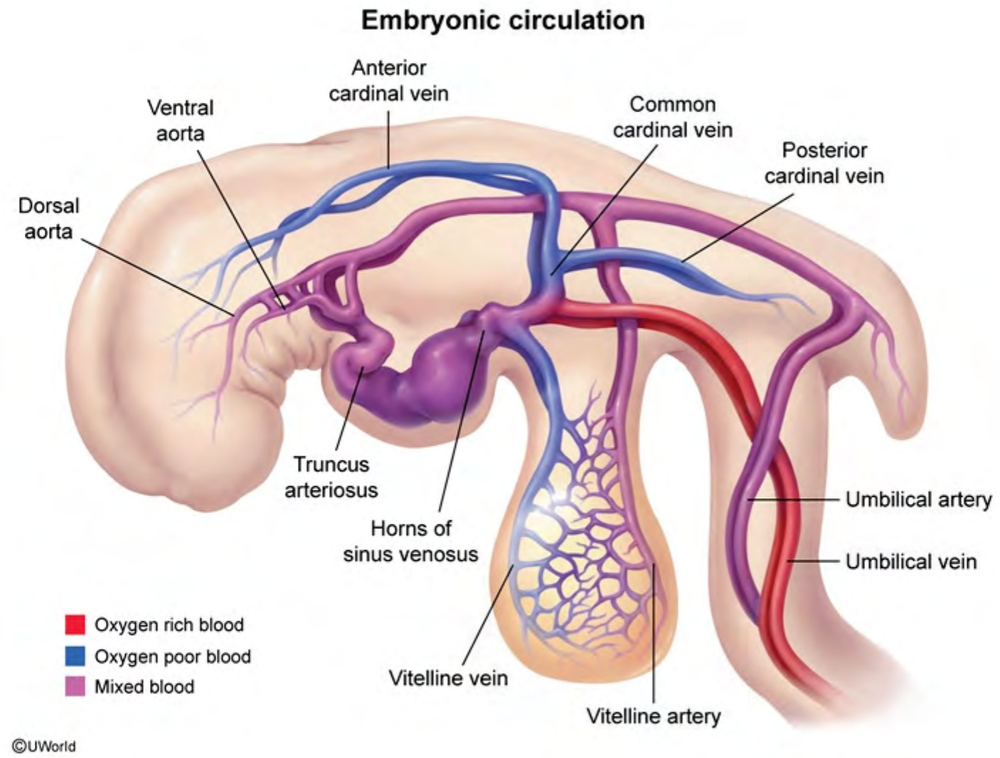

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A 68-year-old man comes to the office due to exertional shortness of breath and fatigue, which have progressed over the past year. The patient has a history of hypertension, but medical history is otherwise unremarkable. He is a lifetime nonsmoker. His father died of a "heart attack" at age 70. Blood pressure is 144/74 mm Hg, and pulse is 72/min and regular. Cardiac auscultation reveals a 3/6 ejection-type, late-peaking systolic murmur and a barely audible S2. The murmur diminishes in intensity during the straining phase of the Valsalva maneuver. Which of the following processes underlies this patient's current condition?

- A. Cardiomyocyte hypertrophy and disarray causing asymmetric septal thickening
- B. Differentiation of valve fibroblasts into osteoblast-like cells
- C. Myxomatous valve thickening and elastin fragmentation
- D. Subendocardial granulomatous lesions with fibrinoid necrosis and subsequent fibrosis
- E. Thrombus formation on inflamed valvular endothelium

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- ✗ A. Cardiomyocyte hypertrophy and disarray causing asymmetric septal thickening (27%)
- ✓ B. Differentiation of valve fibroblasts into osteoblast-like cells (23%)
- C. Myxomatous valve thickening and elastin fragmentation (35%)
- D. Subendocardial granulomatous lesions with fibrinoid necrosis and subsequent fibrosis (7%)
- E. Thrombus formation on inflamed valvular endothelium (5%)

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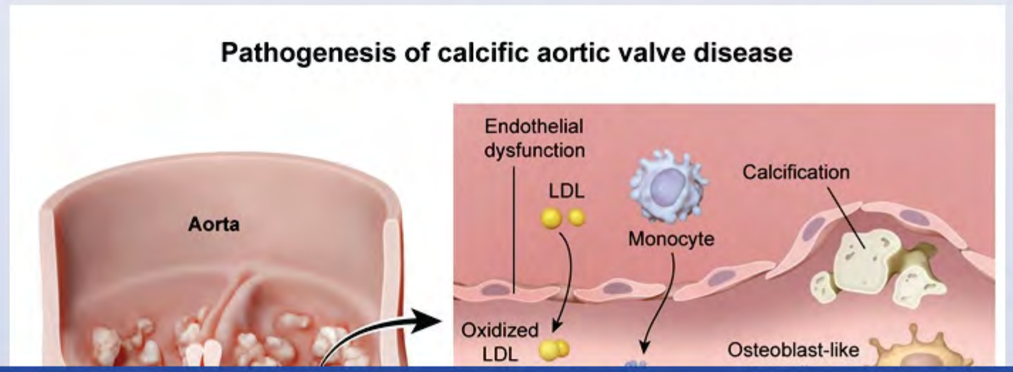

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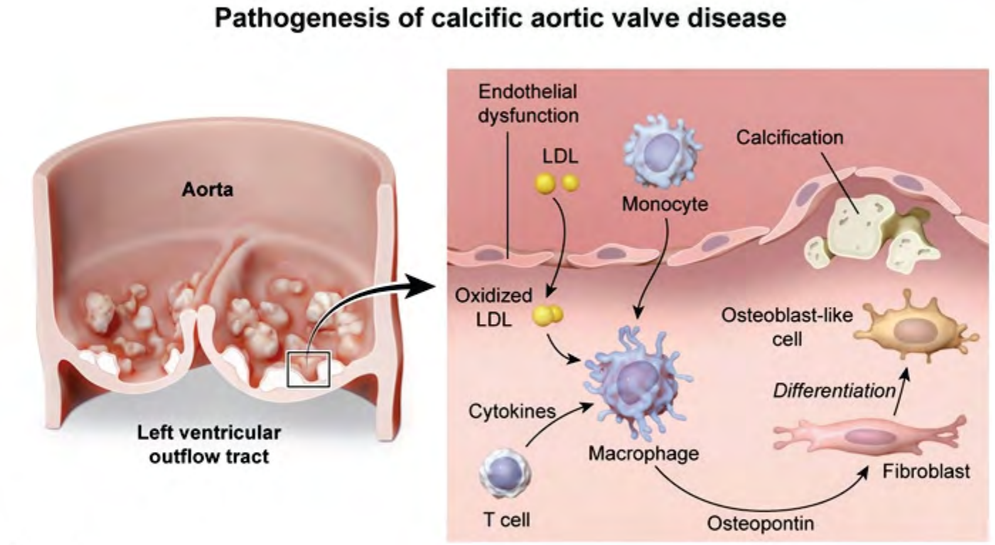

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This patient's soft S2 and late-peaking systolic ejection murmur that decreases in intensity with maneuvers that decrease left ventricular blood volume (eg, abrupt standing, Valsalva straining phase) are consistent with **aortic stenosis**, which most commonly occurs due to age-related **calcific aortic valve disease** (CAVD).

The early pathogenesis of CAVD is analogous to that of **arterial atherosclerosis**. The endothelium lining the aortic side of the aortic valve cusps is exposed to the same high pressure and turbulent blood flow as the aortic vascular endothelium. As occurs with atheroma development in the vascular endothelium, these mechanical forces (along with smoking, hyperglycemia, and hyperlipidemia) over time cause damage to the **aortic valve cusp endothelium**, triggering **endothelial dysfunction** and the onset of a similar atherosclerotic process. There is subendothelial lipid deposition and infiltration of inflammatory cells (ie, macrophages, T lymphocytes) followed by the release of inflammatory mediators (eg, interleukin-1-beta, transforming growth factor beta-1).

Subsequently, there is increased production of proteins involved in tissue calcification (eg, osteopontin). Fibroblasts differentiate into **osteoblast-like cells**, leading to aberrant bone matrix deposition with **progressive valvular calcification** and stenosis.

(Choice A) Cardiomyocyte hypertrophy and disarray with asymmetric septal thickening occurs in hypertrophic cardiomyopathy. As with aortic stenosis, patients have a harsh crescendo-decrescendo systolic murmur; however, the murmur increases (rather than decreases) in intensity with maneuvers that reduce left ventricular blood volume.

(Choice C) Myxomatous valve thickening and elastin fragmentation is typically seen in mitral valve prolapse. Patients have a nonejection systolic click and a mid-to-late systolic murmur that occurs earlier in systole with decreases in left ventricular blood volume.

(Choice D) Subendocardial granulomatous lesions with fibrinoid necrosis and subsequent fibrosis are seen in patients with rheumatic heart disease. Patients with rheumatic aortic stenosis typically have mitral valve involvement as well, which would be evidenced by a murmur of mitral stenosis or regurgitation.

(Choice E) Inflammation of valvular endothelium with deposition of sterile platelet thrombi is seen in nonbacterial thrombotic endocarditis (also known as marantic, verrucous, or Libman-Sacks endocarditis). The mitral or aortic valve is typically involved, usually leading to regurgitation rather than stenosis.

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Educational objective:
Aortic stenosis most commonly results from age-related calcific aortic valve disease (CAVD). The early pathogenesis of CAVD closely mimics that of arterial atherosclerosis. In the later stages, fibroblasts differentiate into osteoblast-like cells and deposit bone matrix, leading to progressive valvular calcification and stenosis.

References

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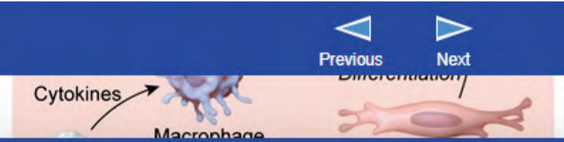

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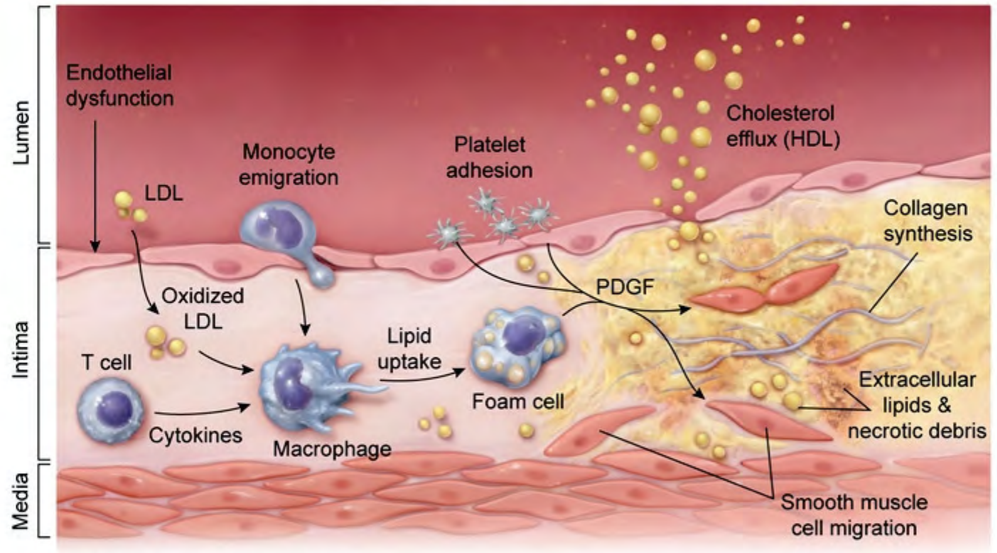

PDGF = platelet-derived growth factor.

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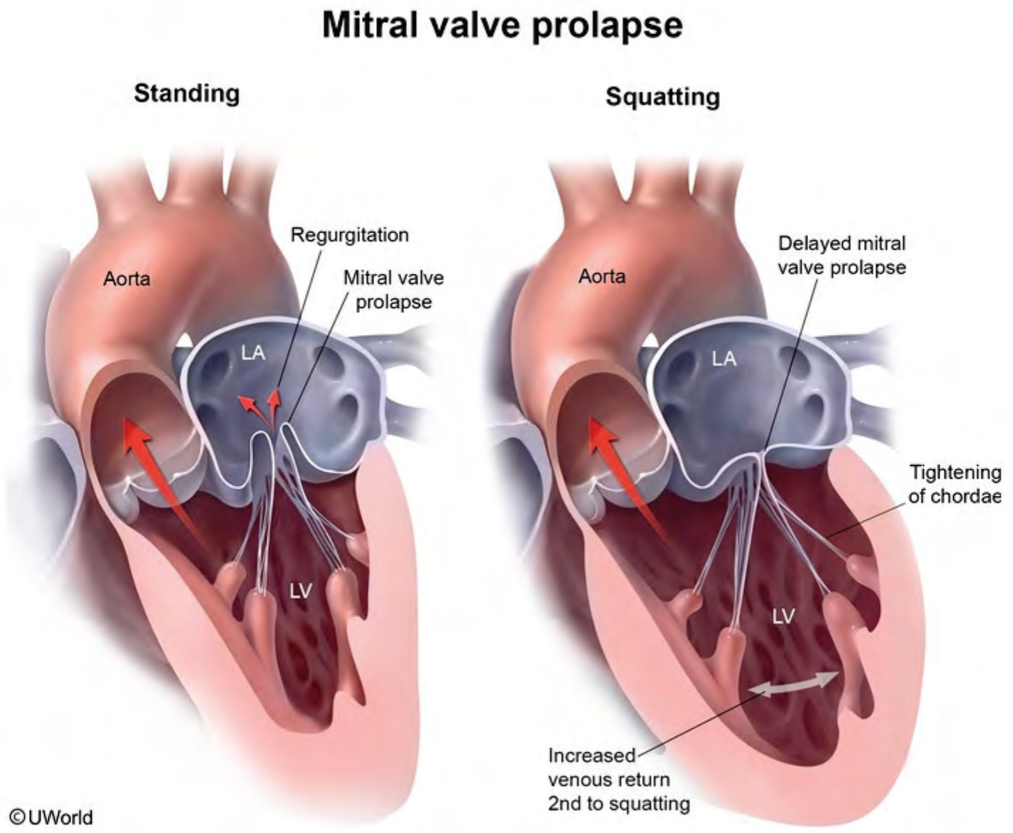

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thrombotic endocarditis (also known as marantic, verrucous, or Libman-Sacks endocarditis). The mitral or aortic

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A 21-year-old man comes to the office due to multiple episodes of syncope. The patient has no chest discomfort or dyspnea. He has no known medical problems and does not use tobacco, alcohol, or illicit drugs. The patient is a computer analyst and leads a mostly sedentary lifestyle. He reports that several family members have died of sudden cardiac death. Genetic analysis reveals an ion channel defect. Due to the defect, cardiac cells show decreased outward potassium flow and resultant prolongation of the action potential. Which of the following is the most likely consequence of this patient's disease?

- A. Abnormal anatomic communication between cardiac chambers
- B. Asymmetric hypertrophy of the left ventricle
- C. Ischemic myocardial necrosis followed by scarring
- D. Left ventricular dilation and systolic dysfunction
- E. Ventricular tachycardia and sudden death

Submit

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- ✗ A. Abnormal anatomic communication between cardiac chambers (6%)
- B. Asymmetric hypertrophy of the left ventricle (7%)
- C. Ischemic myocardial necrosis followed by scarring (1%)
- D. Left ventricular dilation and systolic dysfunction (9%)
- ✓ E. Ventricular tachycardia and sudden death (74%)

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Correct answer
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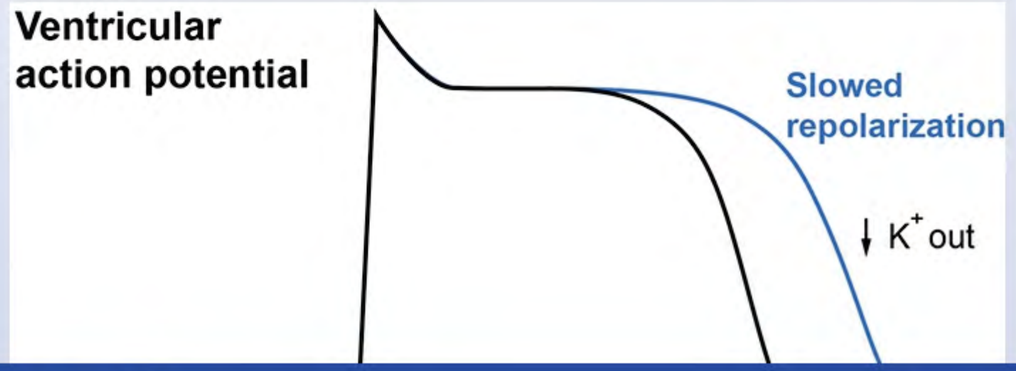

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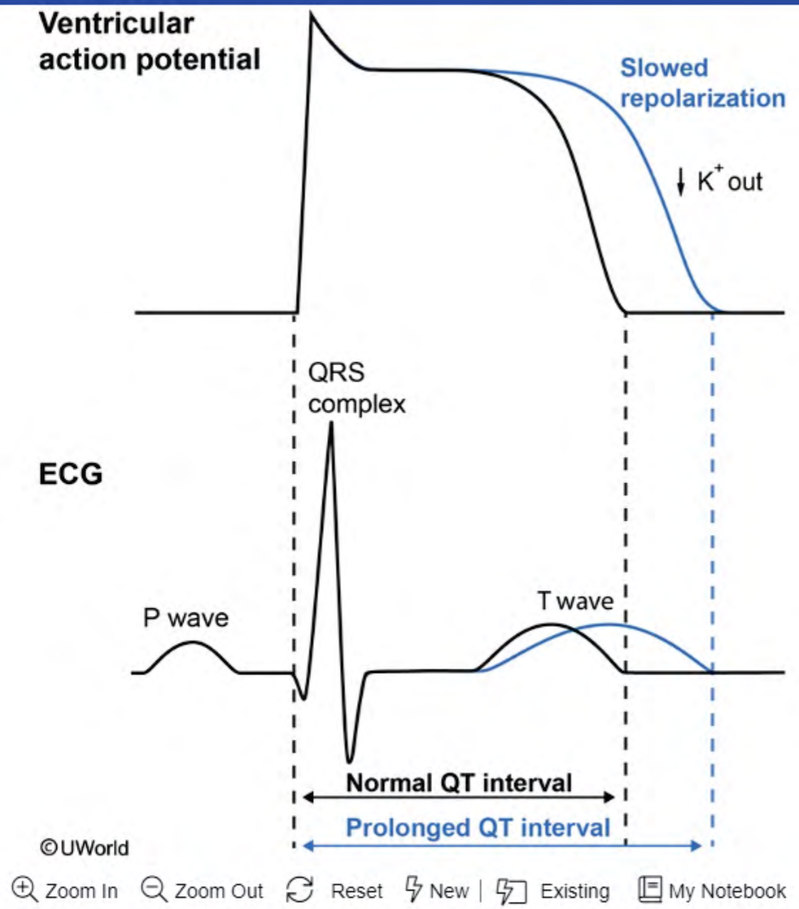

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This patient's recurrent syncope, family history of sudden death, and ion channel defect (decreased outward potassium flow with prolonged action potential) is suggestive of **congenital long QT syndrome** (LQTS). There are several genetic mutations described in patients with congenital LQTS. The 2 most common forms (LQT type 1 and LQT type 2) are due to genetic **mutations in K+ channel** proteins that contribute to the outward-rectifying potassium current.

Phase 3 (repolarization) of the myocardial action potential occurs due to time-dependent inactivation of the L-type (long-lasting, large-conductance) Ca^{2+} channels (which reduces inward current) along with activation of the voltage-gated delayed rectifier K^+ channels (which increases outward current). This net increase in outward transmembrane current is responsible for ventricular repolarization. **Decreased outward potassium** flow due to K^+ channel mutations leads to prolongation of action potential duration and QT interval. Prolongation of QT interval predisposes to the development of life-threatening ventricular arrhythmias (eg, torsades de pointes), which can present as recurrent palpitations, **syncope**, **seizures**, or **sudden cardiac death**.

(Choice A) Abnormal anatomic communication between cardiac chambers presents in patients with atrial septal defects, ventricular septal defects, or patent ductus arteriosus. These disorders are not associated with any genetic defects in ion channels.

(Choice B) Asymmetric hypertrophy of the left ventricle occurs in patients with hypertrophic cardiomyopathy, an autosomal dominant disorder caused by mutations in sarcomere genes. The 2 most common mutations involve the cardiac myosin-binding protein C gene and cardiac beta-myosin heavy chain gene.

(Choice C) Ischemic myocardial necrosis and scarring occur in myocardial infarction and often lead to left ventricular systolic dysfunction. Patients with scarred myocardium are also at increased risk for developing ventricular arrhythmias; however, these arrhythmias would not be associated with an ion channel defect.

(Choice D) Dilated cardiomyopathy is characterized by left ventricle dilation and systolic dysfunction. Several genetic mutations involving the sarcomere structure and function have been identified in patients with familial dilated cardiomyopathy. However, abnormal transmembrane K^+ currents are not known to be directly involved in the pathophysiology.

Educational objective:

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Educational objective:
Congenital long QT syndrome is most often caused by genetic mutations in a K^+ channel protein that contributes to the outward-rectifying potassium current. A decrease in the outward K^+ current leads to prolongation of action potential duration and QT interval. This prolongation predisposes to the development of life-threatening ventricular arrhythmias (eg, torsades de pointes) that can cause palpitations, syncope, seizures, or sudden cardiac death.

Pathology	Cardiovascular System	Long QT syndrome
Subject	System	Topic

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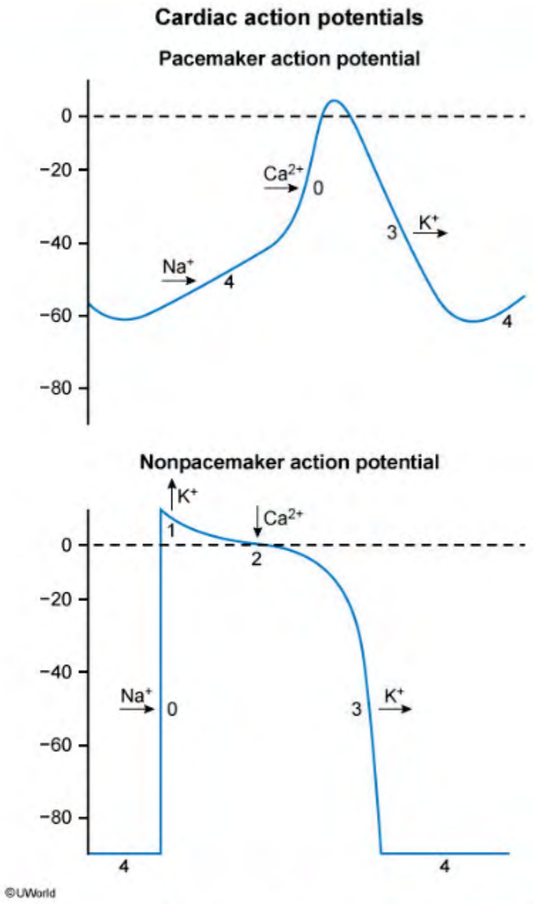

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Normal QT interval

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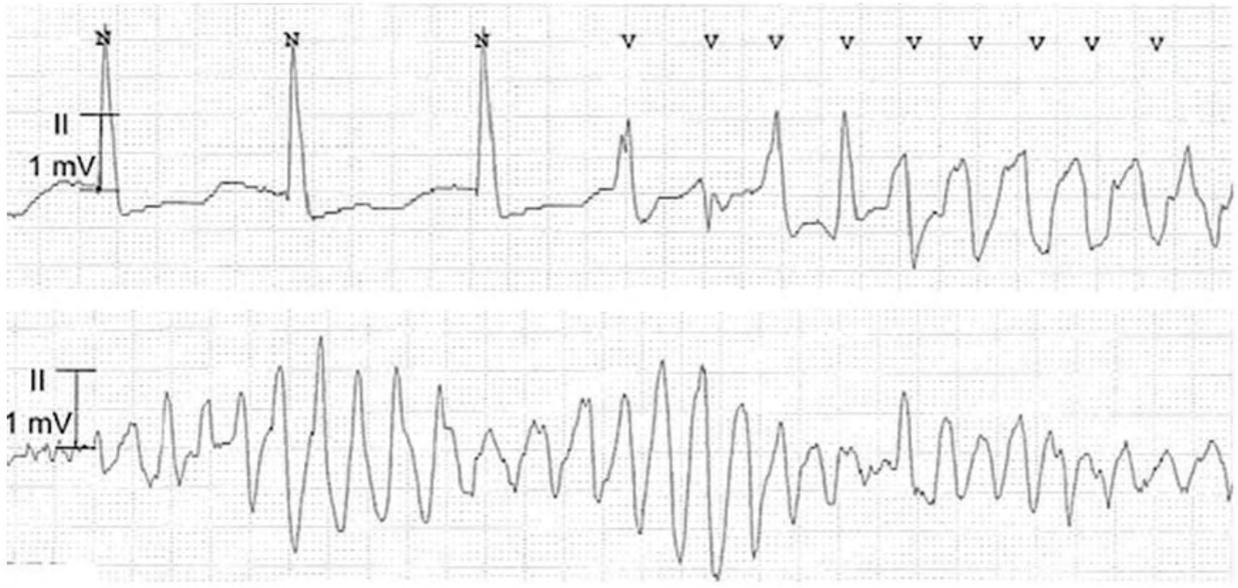

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(Choice D) Dilated cardiomyopathy is characterized by left ventricle dilation and systolic dysfunction. Several genetic mutations involving the sarcomere structure and function have been identified in patients with familial

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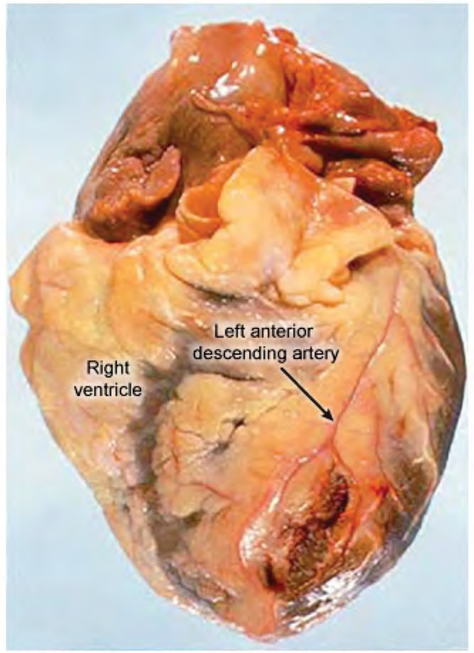
A 64-year-old woman who was discharged from the hospital 4 days ago comes to the emergency department due to chest tightness and severe shortness of breath. During evaluation, the patient becomes unresponsive and goes into cardiac arrest. Despite appropriate lifesaving measures, there is no return of spontaneous circulation and she is pronounced dead. Autopsy examination is performed, and the myocardial findings are shown in the image below.



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Which of the following most likely predisposed this patient to the observed cardiac findings?

- A. Coronary atherosclerosis
- B. Prolonged alcohol consumption
- C. Recent viral infection

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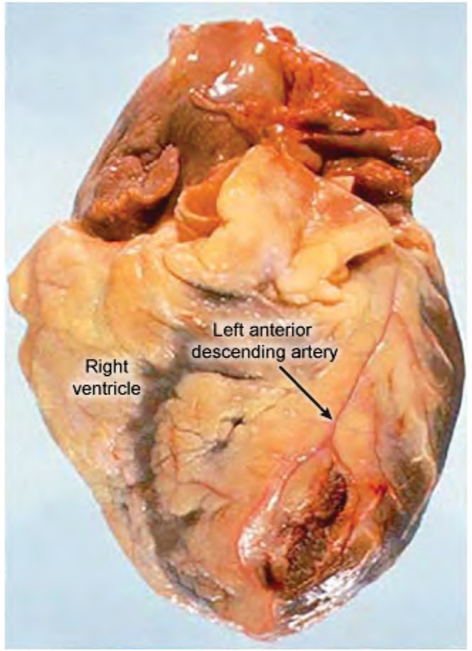

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Which

C. Recent viral infection

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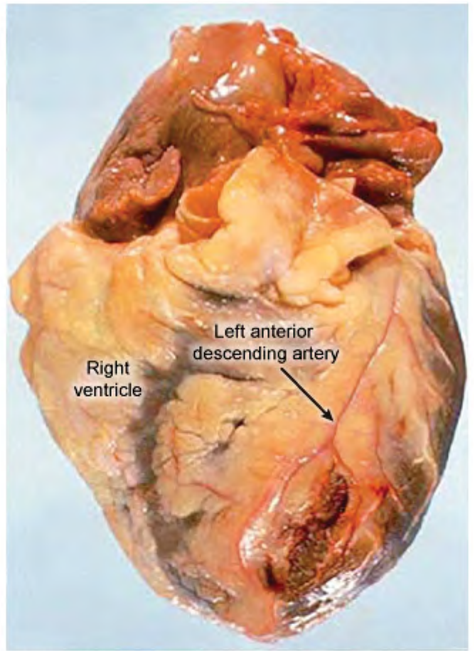

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Which of the following most likely predisposed this patient to the observed cardiac findings?

- A. Coronary atherosclerosis
- B. Prolonged alcohol consumption
- C. Recent viral infection
- D. Recurrent bacterial pharyngitis
- E. Repeated blood transfusions

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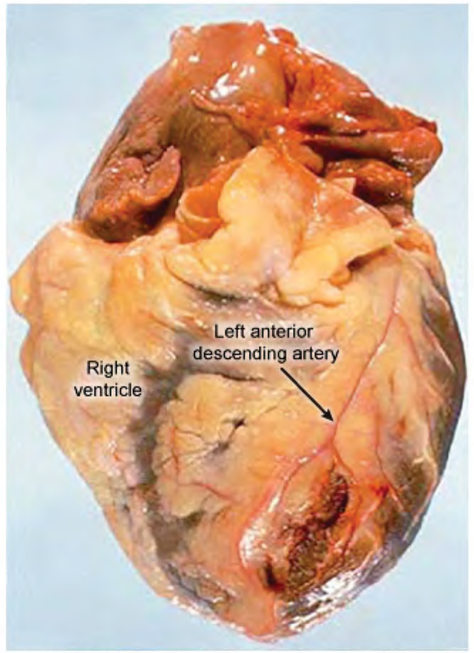

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Which of the following most likely predisposed this patient to the observed cardiac findings?

- ✓ A. Coronary atherosclerosis (90%)
- B. Prolonged alcohol consumption (1%)
- C. Recent viral infection (4%)
- D. Recurrent bacterial pharyngitis (1%)
- E. Repeated blood transfusions (1%)

Correct

90% Answered correctly

29 secs Time Spent

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Explanation

Mechanical complications of acute myocardial infarction		
Complication	**Time course**	**Clinical findings**
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR) • Hypotension/cardiogenic shock
Interventricular septum rupture	Acute or within 3-5 days	• Chest pain • New holosystolic murmur • Hypotension/cardiogenic shock • Step up in O_2 level from RA to RV
Free wall rupture**	Within 5 days or up to 2 weeks	• Chest pain • Distant heart sounds • Shock, rapid progression to cardiac arrest
Left ventricular aneurysm**	Up to several months	• Heart failure • Angina, ventricular arrhythmias
*Usually due to right coronary artery occlusion. **Usually due to left anterior descending artery occlusion. **MR** = mitral regurgitation; **RA** = right atrium; **RV** = right ventricle.		

This patient's autopsy reveals a slitlike tear in the anterior left ventricular (LV) wall that is consistent with **LV free-wall rupture**, an uncommon but devastating mechanical complication of **transmural myocardial infarction** (MI) that occurs **up to 2 weeks** following the event (frequently within 5 days). She likely had significant **coronary atherosclerosis** and experienced a transmural (ST-segment elevation) MI during her previous hospitalization.

Following MI, the infarcted myocardium becomes akinetic and does not move with the remaining healthy myocardium, creating additional shear stress on the myocardial wall. Coagulative necrosis and enzymatic lysis of

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This patient's autopsy reveals a slitlike tear in the anterior left ventricular (LV) wall that is consistent with **LV free-wall rupture**, an uncommon but devastating mechanical complication of **transmural myocardial infarction** (MI) that occurs **up to 2 weeks** following the event (frequently within 5 days). She likely had significant **coronary atherosclerosis** and experienced a transmural (ST-segment elevation) MI during her previous hospitalization.

Following MI, the infarcted myocardium becomes akinetic and does not move with the remaining healthy myocardium, creating additional shear stress on the myocardial wall. Coagulative necrosis and enzymatic lysis of connective tissue also weaken the myocardial wall to the extent that it may be unable to contain the high LV pressures. If rupture occurs, it leads to abrupt filling of the pericardial space with blood and rapid development of **cardiac tamponade**. Patients can experience chest pain and shortness of breath followed by hypotension and shock that can rapidly progress to **cardiac arrest**.

(Choices B, C, and E) Prolonged alcohol consumption, recent viral infection, and repeated blood transfusions (ie, iron overload) are all potential causes of dilated cardiomyopathy. Dilated cardiomyopathy can lead to cardiac arrest and death via generation of a malignant arrhythmia (ie, ventricular tachycardia or fibrillation); however, autopsy is expected to reveal an enlarged heart with eccentric hypertrophy of the LV and no tears in the myocardium.

(Choice D) Recurrent bacterial pharyngitis can lead to recurrent episodes of rheumatic fever, which can cause inflammatory damage to both the myocardium and valvular endocardium. Death most commonly results from decompensated heart failure caused by valvular dysfunction; gross findings on autopsy are expected to show an enlarged heart with degenerated valves.

Educational objective:
Left ventricular (LV) free-wall rupture is an uncommon but devastating mechanical complication of transmural myocardial infarction that occurs up to 2 weeks following the event (frequently within 5 days). Rupture leads to cardiac tamponade that causes hypotension and shock with rapid progression to cardiac arrest. Autopsy typically reveals a slitlike tear at the site of infarction in the LV wall.

Pathophysiology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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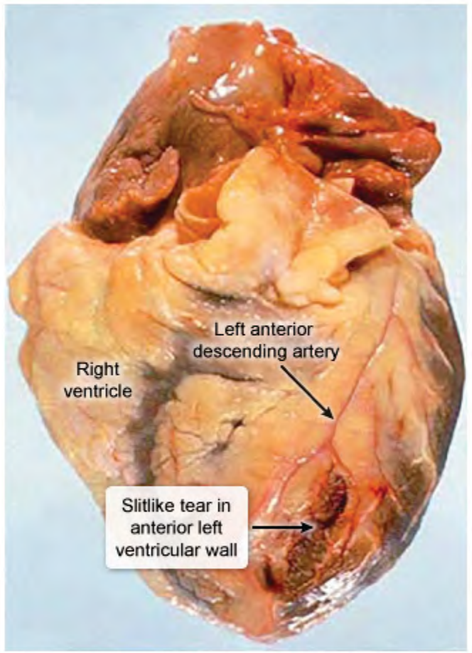
Left ventricular	Up to several months	• Heart failure

Exhibit Display

Left ventricular free-wall rupture



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Educational objective:
Left ventricular (LV) free-wall rupture is an uncommon but devastating mechanical complication of transmural

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A 2-day-old boy is being evaluated in the nursery for irritability. He was born to a 22-year-old primigravida who received limited prenatal care. Delivery was uncomplicated, and Apgar scores were 7 and 8 at 1 and 5 minutes, respectively. Temperature is 36.7 C (98 F), blood pressure is 70/30 mm Hg, pulse is 148/min, and respirations are 68/min. On physical examination, the patient is cyanotic and irritable. Auscultation reveals a continuous, machinery-like murmur appreciated between the scapulae. Serum lactate level is elevated. Echocardiogram shows the origin of the aorta lying anterior and to the right of the pulmonary artery. Failure of which of the following processes during embryonic development is most likely responsible for this patient's findings on imaging?

- A. Fusion
- B. Obliteration
- C. Proliferation
- D. Septation
- E. Spiraling

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- ✗ A. Fusion (4%)
- B. Obliteration (13%)
- C. Proliferation (1%)
- D. Septation (11%)
- ✓ E. Spiraling (67%)

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Correct answer
E

67%
Answered correctly

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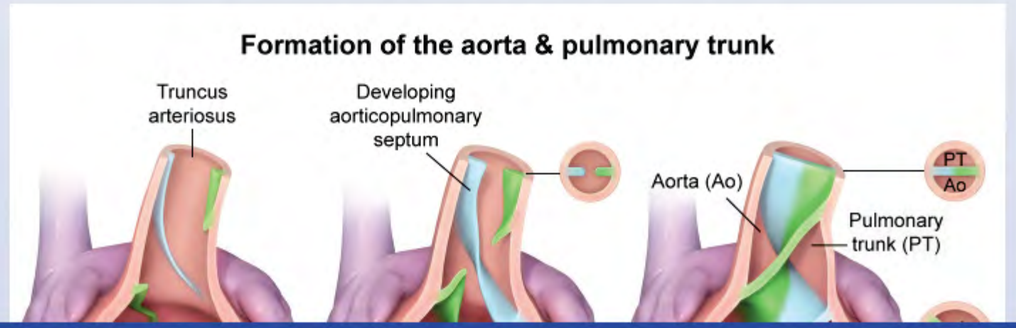
Explanation



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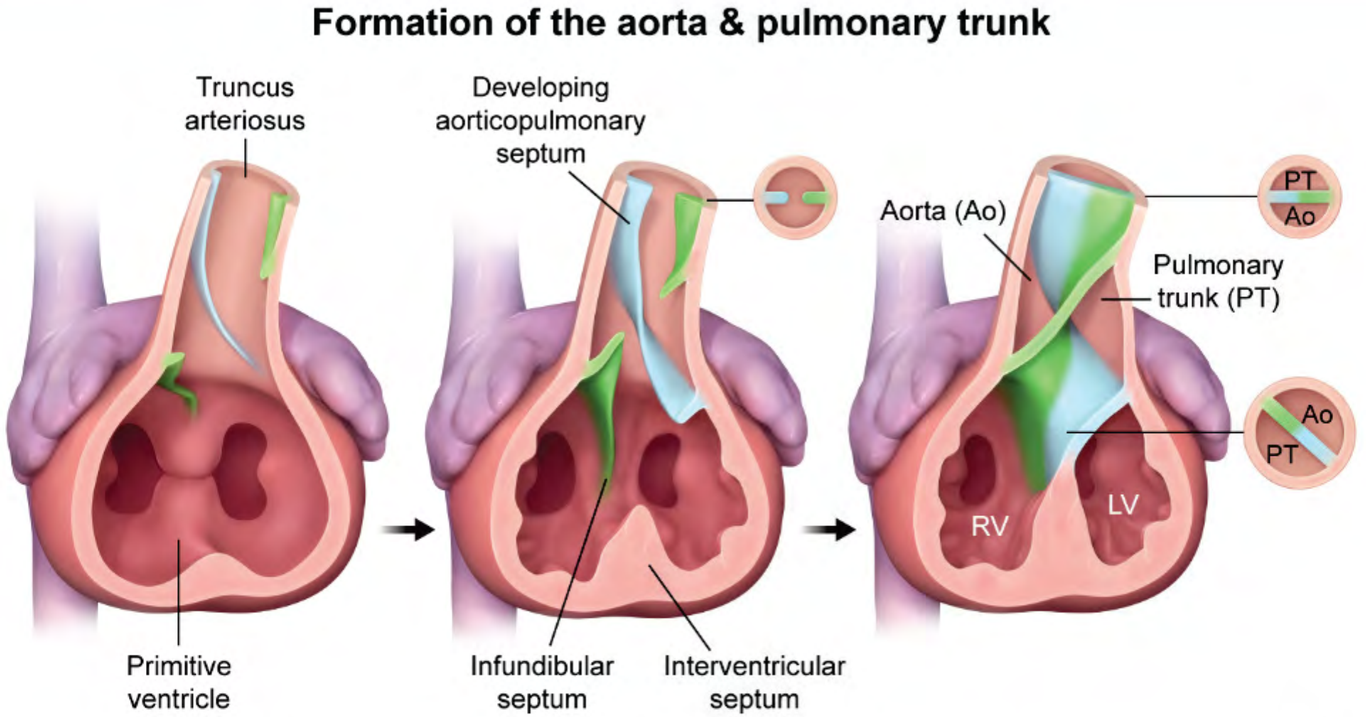

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This newborn's echocardiogram is indicative of **transposition of the great arteries** (TGA), a cyanotic congenital heart defect characterized by abnormal origination of the aorta/pulmonary artery. In early gestation, there is a single outflow tract from the rudimentary heart that is subsequently divided into the aorta and pulmonary artery by the **aorticopulmonary septum**. This septum normally spirals during outflow tract division to create an anteriorly located pulmonary artery arising from the right ventricle and a posteriorly located aorta arising from the left ventricle.

TGA occurs due to **failed spiraling** of the aorticopulmonary septum, resulting in an **anterior aorta** that originates from the right ventricle and a **posterior pulmonary artery** that originates from the left ventricle. Parallel pulmonary and systemic circuits are created, and **deoxygenated blood** is delivered from the right ventricle to the **systemic circulation** via the aorta.

Neonates with TGA often have cyanosis, tachypnea, and tachycardia immediately after birth; however, some may have normal examination findings initially due to shunting of oxygenated blood to the systemic circulation via the ductus arteriosus, with cyanosis developing after 24-48 hours as the ductus begins to close. Other mixing lesions, such as a large ventricular septal defect, can also mask the development of clinically-apparent cyanosis. In this patient, elevated lactate is caused by a high degree of anaerobic metabolism due to poor oxygen delivery to the tissues.

(Choices A and C) Failed fusion of the septum primum with the endocardial cushions leads to a primum-type atrial septal defect (ASD), whereas failed proliferation of the septum secundum leads to a secundum-type ASD. An ASD would not cause neonatal cyanosis.

(Choice B) Failed obliteration of the ductus arteriosus results in a patent ductus arteriosus (PDA), which by itself would not explain neonatal cyanosis. PDA in a patient with TGA tends to delay the development of cyanosis.

(Choice D) Failed conotruncal septation results in persistent truncus arteriosus. Although affected patients typically develop neonatal cyanosis, an echocardiogram would reveal a single arterial trunk. This patient's distinct aorta and pulmonary artery indicate conotruncal septation has occurred.

Educational objective:
Transposition of the great arteries is a cyanotic congenital heart condition resulting from failed spiraling of the

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the **aorticopulmonary septum**. This septum normally spirals during outflow tract division to create an anteriorly located pulmonary artery arising from the right ventricle and a posteriorly located aorta arising from the left ventricle.

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(Choice D) Failed conotruncal septation results in persistent truncus arteriosus. Although affected patients typically develop neonatal cyanosis, an echocardiogram would reveal a single arterial trunk. This patient's distinct aorta and pulmonary artery indicate conotruncal septation has occurred.

Educational objective:
Transposition of the great arteries is a cyanotic congenital heart condition resulting from failed spiraling of the aorticopulmonary septum during embryonic development. The aorta aberrantly originates from the right ventricle, lies anterior to the pulmonary artery, and delivers deoxygenated blood to the systemic circulation.

References

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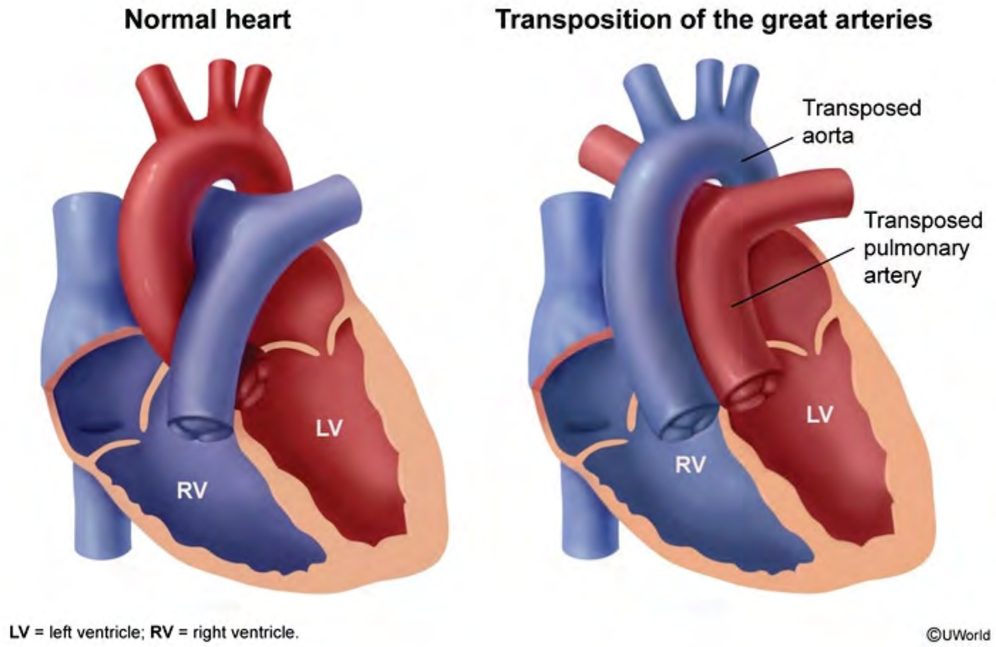

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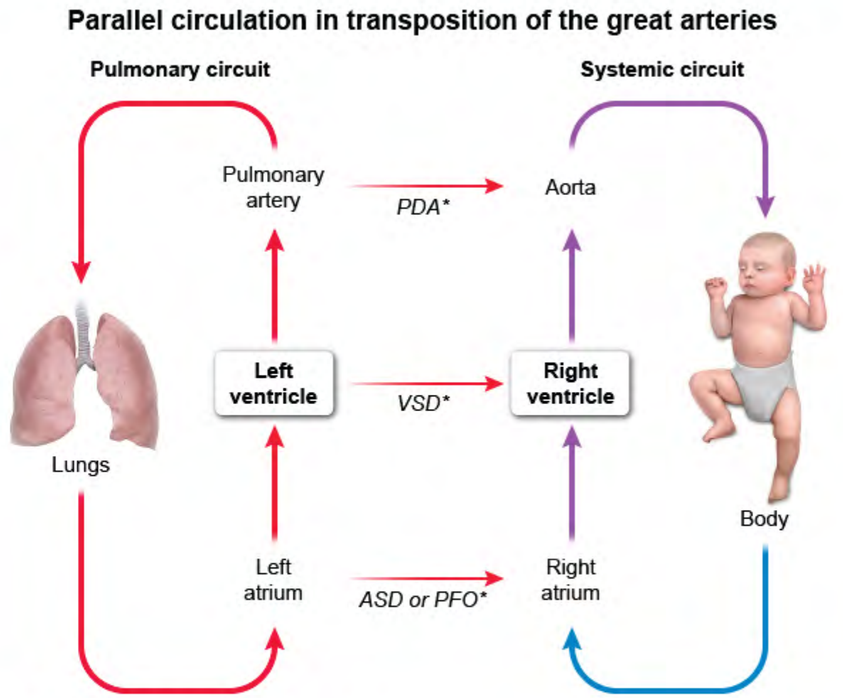

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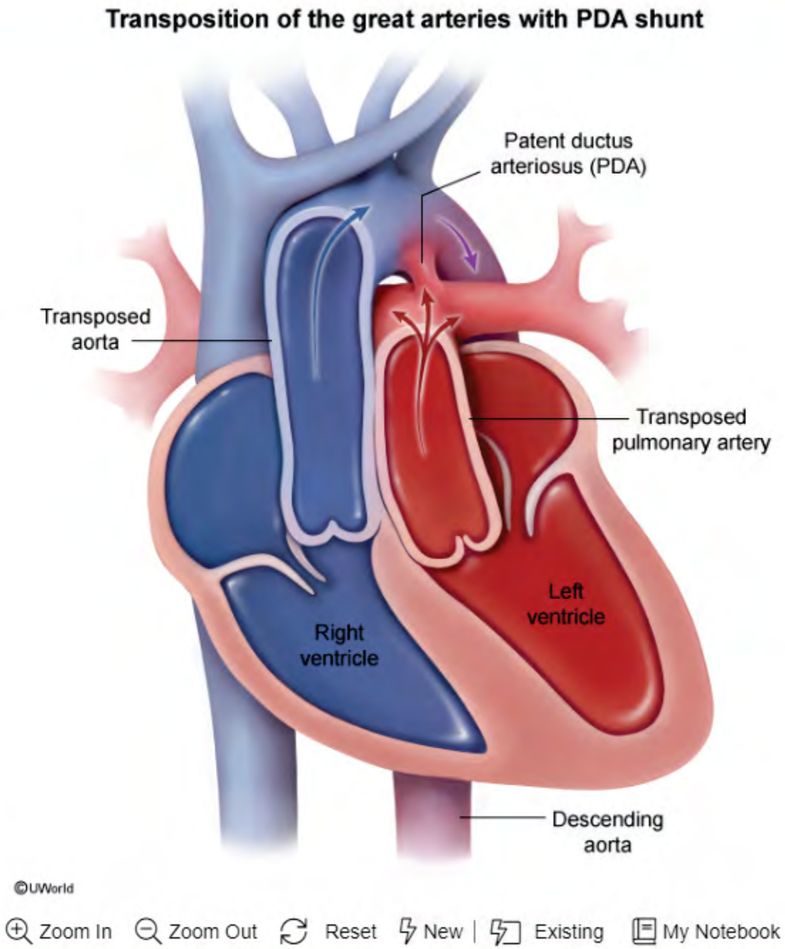

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This newborn's echocardiogram is indicative of **transposition of the great arteries** (TGA), a cyanotic congenital heart
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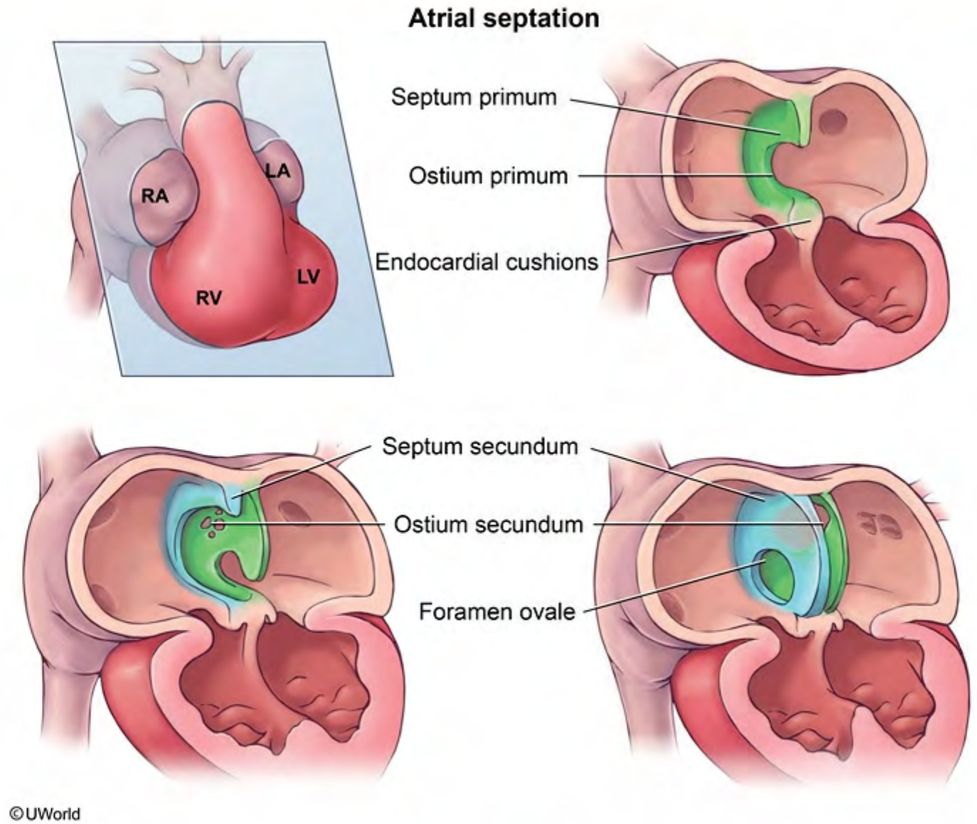

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Educational objective:

Transposition of the great arteries is a cyanotic congenital heart condition resulting from failed spiraling of the

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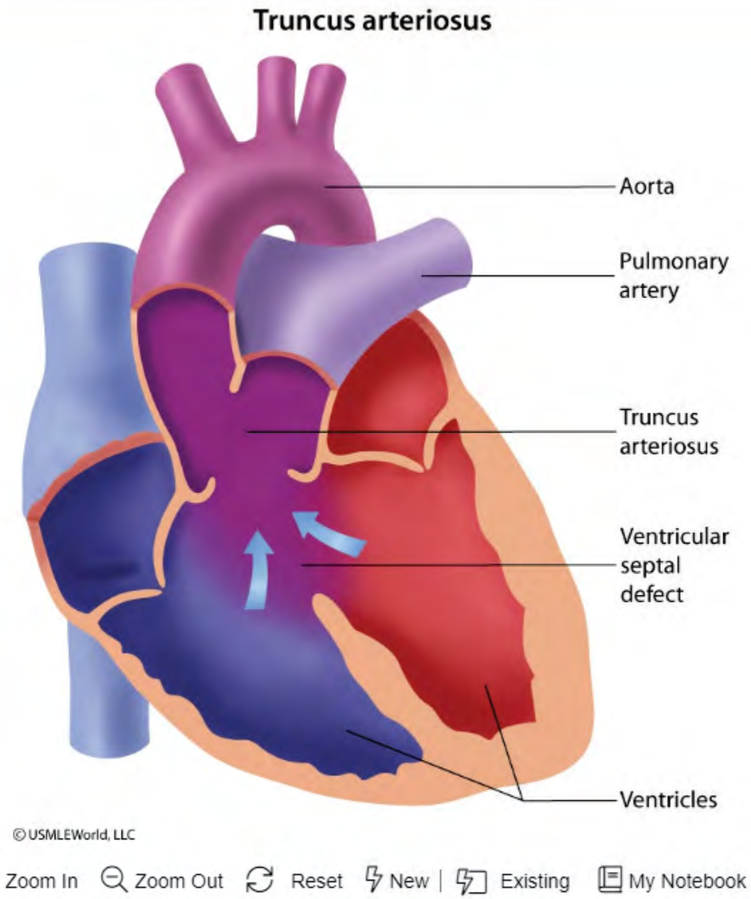

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A 71-year-old woman comes to the emergency department due to progressive dyspnea and orthopnea for the last week. She has a history of myocardial infarction and systolic heart failure with a left ventricular ejection fraction of 15%. The patient is compliant with her medications, which include carvedilol, furosemide, aspirin, lisinopril, and atorvastatin. Blood pressure is 118/74 mm Hg and pulse is 72/min. Examination reveals elevated jugular venous pressure, bibasilar crackles, an S3 heart sound, and bilateral lower extremity pitting edema. The patient is started on dobutamine infusion, which provides significant symptom relief. Echocardiogram shows mild improvement in cardiac contractility. Which of the following is the likely mechanism underlying this patient's clinical improvement?

- A. Cyclic nucleotide phosphodiesterase inhibition
- B. G_s protein-adenylate cyclase activation
- C. Late-phase inward sodium channel inhibition
- D. Sodium-potassium exchange pump inhibition
- E. Soluble guanylate cyclase activation
- F. Voltage-dependent calcium channel inhibition

Submit

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- A. Cyclic nucleotide phosphodiesterase inhibition (2%)
- ✓ B. G_s protein-adenylate cyclase activation (79%)
- C. Late-phase inward sodium channel inhibition (3%)
- D. Sodium-potassium exchange pump inhibition (5%)
- E. Soluble guanylate cyclase activation (3%)
- F. Voltage-dependent calcium channel inhibition (4%)

Correct

79%
Answered correctly

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Time Spent

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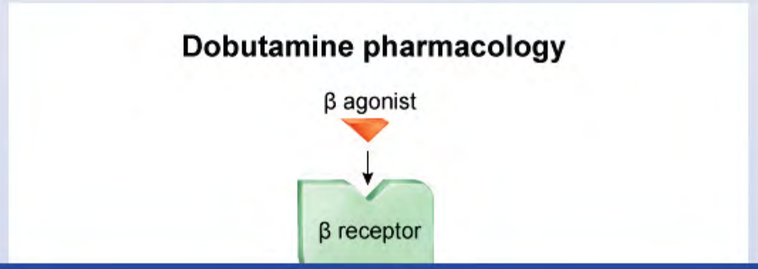
Explanation



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E. Soluble guanylate cyclase activation (3%)

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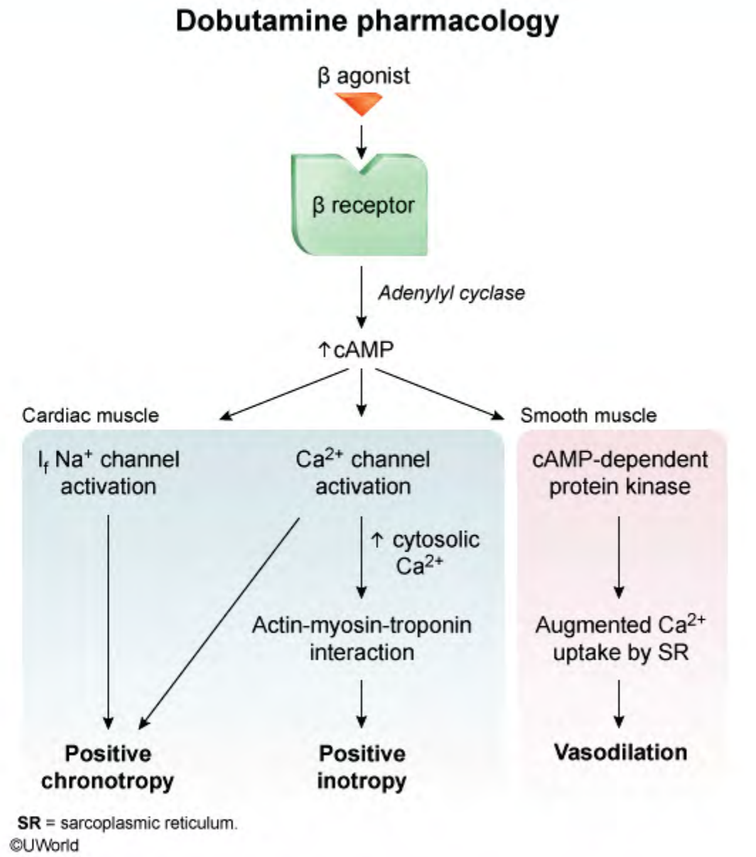

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Positive chronotropy | Positive inotropy | Vasodilation

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Dobutamine is a β-adrenergic agonist with predominant activity on β_1 receptors and weak activity on β_2 and α_1 receptors. Stimulation of β_1-adrenergic receptors causes **G_s protein** GTP binding, leading to activation of adenylyl cyclase and **increased production of cAMP** in the target cells. In cardiac myocytes, increased cAMP causes calcium channel activation and increased cytosolic Ca^{2+} concentration. This causes a conformational change in the troponin complex, facilitating actin-myosin binding and increasing myocardial contractility (**positive inotropy**). Increased cAMP also causes a rise in heart rate by increasing Na^+ and Ca^{2+} channel activation in pacemaker cells (**positive chronotropy**).

In the vasculature, α_1 agonist activity (vasoconstriction) balances the β_2 agonist effect (vasodilation) with resultant mild vasodilation. The net hemodynamic effects are an increase in cardiac contractility and a decrease in systemic vascular resistance without significant change in arterial blood pressure. This increase in cardiac output can improve symptoms and end-organ perfusion in patients with severe left ventricular systolic dysfunction and cardiogenic shock.

(Choice A) Phosphodiesterase inhibitors (eg, milrinone) increase the intracellular levels of cAMP by preventing its enzymatic breakdown in cardiac and vascular tissues, and result in vasodilation and positive inotropic effects.

(Choice C) Ranolazine is an anti-anginal drug that inhibits late-phase inward sodium channels in ischemic myocardial cells during cardiac repolarization. A decrease in intracellular sodium concentration enhances calcium efflux via the sodium-calcium exchanger, which reduces myocardial oxygen consumption.

(Choice D) Digoxin acts by inhibiting the sodium-potassium ATPase pump. This increases intracellular sodium concentration, which in turn decreases activity of the sodium-calcium exchanger that exports calcium from myocardial cells. The corresponding rise in intracellular Ca^{2+} concentration leads to improved myocardial contractility.

(Choice E) Nitrates provide free radical nitric oxide, which activates guanylate cyclase and increases cGMP levels. This in turn leads to dephosphorylation of myosin light chains and causes vascular smooth muscle relaxation.

(Choice F) Calcium channel blockers (eg, verapamil, diltiazem) prevent calcium ions from traversing voltage-dependent calcium channels in vascular smooth muscle and myocardium, leading to vasodilation and a decrease

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(**positive chronotropy**).

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(Choice F) Calcium channel blockers (eg, verapamil, diltiazem) prevent calcium ions from traversing voltage-dependent calcium channels in vascular smooth muscle and myocardium, leading to vasodilation and a decrease in cardiac contractility.

Educational objective:
Dobutamine is a β-adrenergic agonist with predominant activity on β_1 receptors and weak activity on β_2 and α_1 receptors. Stimulation of β_1 receptors leads to an increased production of cAMP and increased cytosolic Ca^{2+} concentration. This facilitates the interaction between actin and myosin, resulting in increased myocardial contractility.

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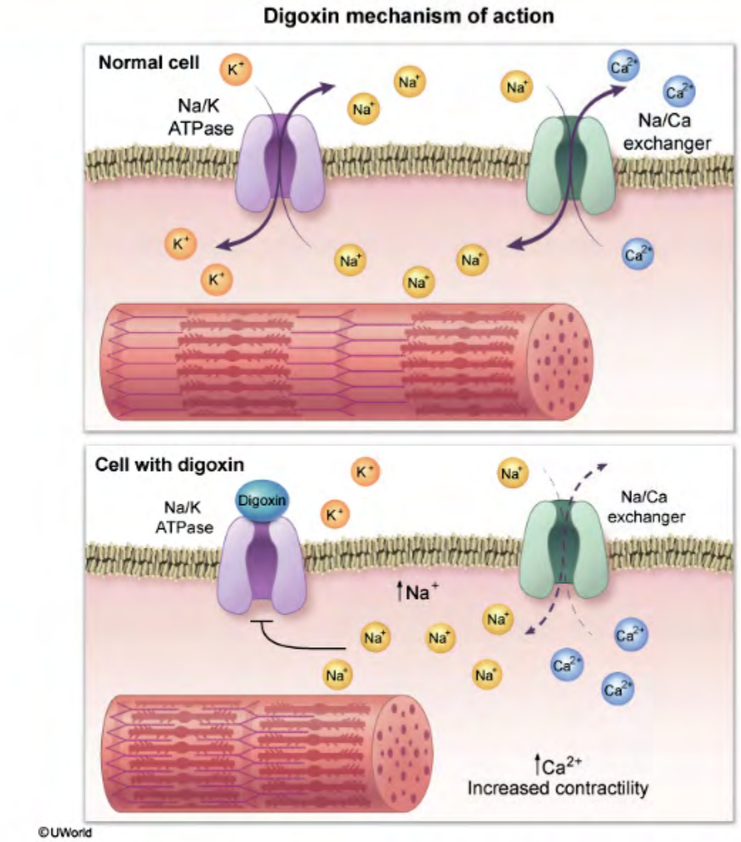

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Educational objective:

Dobutamine is a β-adrenergic agonist with predominant activity on β_1 receptors and weak activity on β_2 and α_1

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(**positive chronotropy**).

In the vasculature, α_1 agonist activity (vasoconstriction) balances the β_2 agonist effect (vasodilation) with resultant mild vasodilation. The net hemodynamic effects are an increase in cardiac contractility and a decrease in systemic vascular resistance without significant change in arterial blood pressure. This increase in cardiac output can improve symptoms and end-organ perfusion in patients with severe left ventricular systolic dysfunction and cardiogenic shock.

(Choice A) Phosphodiesterase inhibitors (eg, milrinone) increase the intracellular levels of cAMP by preventing its enzymatic breakdown in cardiac and vascular tissues, and result in vasodilation and positive inotropic effects.

(Choice C) Ranolazine is an anti-anginal drug that inhibits late-phase inward sodium channels in ischemic myocardial cells during cardiac repolarization. A decrease in intracellular sodium concentration enhances calcium efflux via the sodium-calcium exchanger, which reduces myocardial oxygen consumption.

(Choice D) Digoxin acts by inhibiting the sodium-potassium ATPase pump. This increases intracellular sodium concentration, which in turn decreases activity of the sodium-calcium exchanger that exports calcium from myocardial cells. The corresponding rise in intracellular Ca^{2+} concentration leads to improved myocardial contractility.

(Choice E) Nitrates provide free radical nitric oxide, which activates guanylate cyclase and increases cGMP levels. This in turn leads to dephosphorylation of myosin light chains and causes vascular smooth muscle relaxation.

(Choice F) Calcium channel blockers (eg, verapamil, diltiazem) prevent calcium ions from traversing voltage-dependent calcium channels in vascular smooth muscle and myocardium, leading to vasodilation and a decrease in cardiac contractility.

Educational objective:
Dobutamine is a β-adrenergic agonist with predominant activity on β_1 receptors and weak activity on β_2 and α_1 receptors. Stimulation of β_1 receptors leads to an increased production of cAMP and increased cytosolic Ca^{2+} concentration. This facilitates the interaction between actin and myosin, resulting in increased myocardial contractility.

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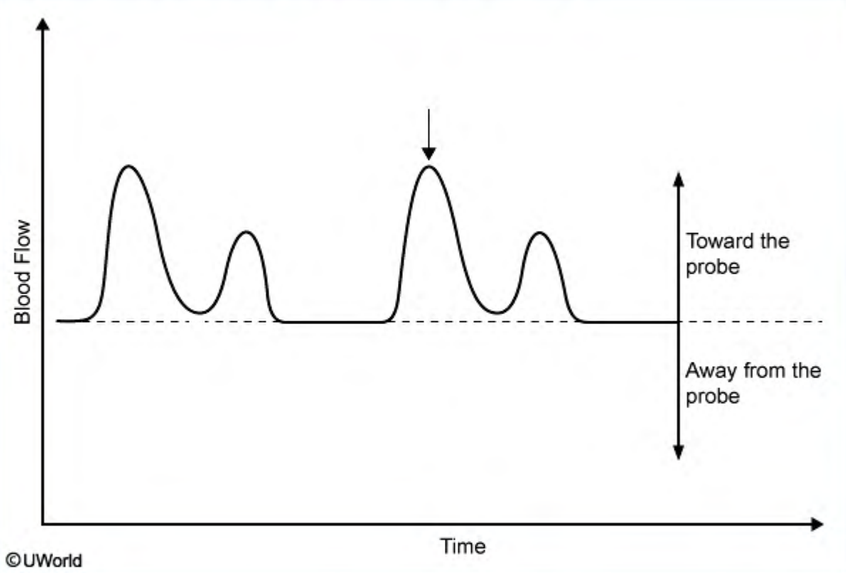
The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 52-year-old man comes to the office with a 2-week history of shortness of breath and dry cough. The patient has noticed decreased exercise tolerance that limits his daily activities. A Doppler probe placed at the cardiac apex reveals the following blood flow pattern across the patient's mitral valve.



Item 1 of 2

The point marked by the arrow would best correspond to which of the following auscultation findings?

- A. Fourth heart sound
- B. Midsystolic click
- C. Second heart sound
- D. Third heart sound

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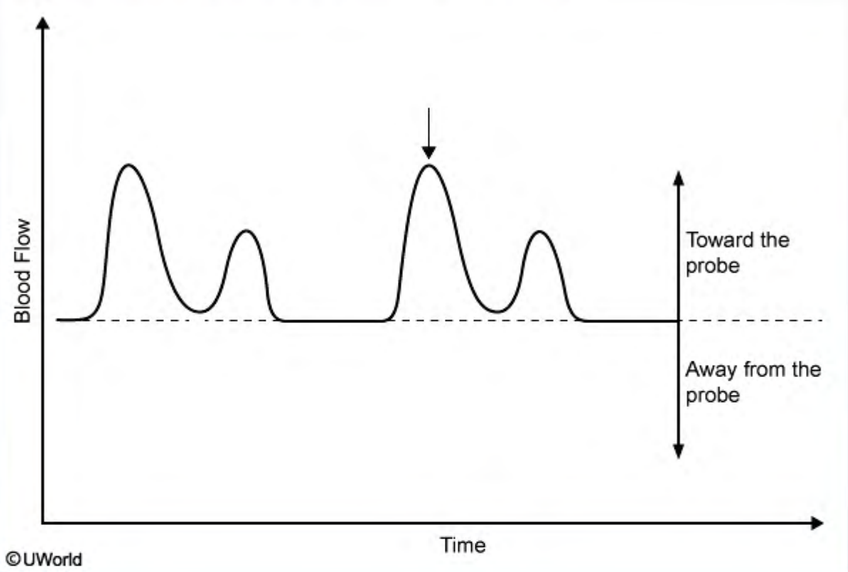

Item 1 of 2

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- B. Midsystolic click
- C. Second heart sound
- D. Third heart sound

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Item 2 of 2

On physical examination, jugular venous pressure is elevated, breath sounds are decreased at the right lung base with dullness to percussion, and 2+ bilateral lower extremity pitting edema is present. Chest x-ray shows cephalization of the blood vessels, Kerley B lines, and a right pleural effusion. Thoracentesis is performed for pleural fluid analysis. In comparison to plasma fluid, which of the following pleural fluid findings is most likely to be observed?

- A. High amylase content
- B. High protein content
- C. High white blood cell count
- D. Low glucose content
- E. Low lactate dehydrogenase content

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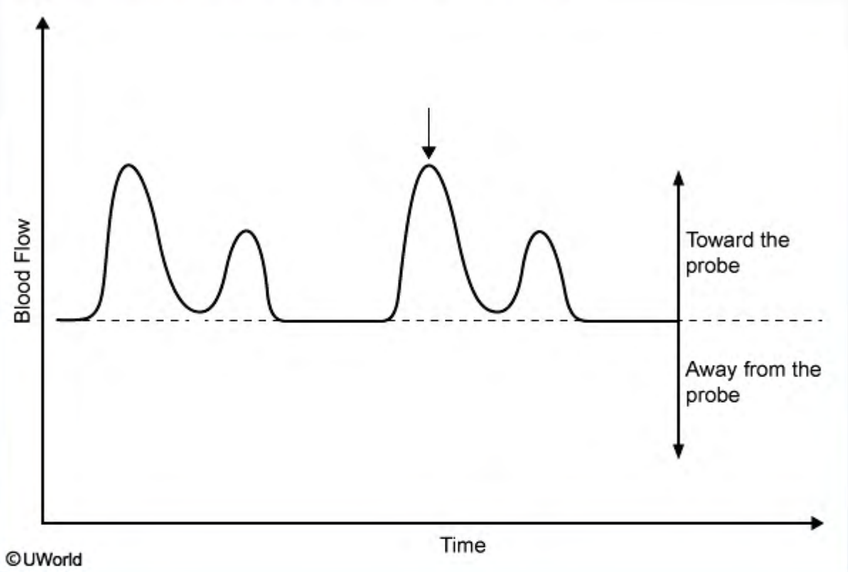

Item 1 of 2

The point marked by the arrow would best correspond to which of the following auscultation findings?

- ✗ A. Fourth heart sound (14%)
- B. Midsystolic click (19%)
- C. Second heart sound (26%)
- ✓ D. Third heart sound (39%)

Incorrect
Correct answer
D

39%
Answered correctly

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Incorrect
Correct answer
D

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Answered correctly

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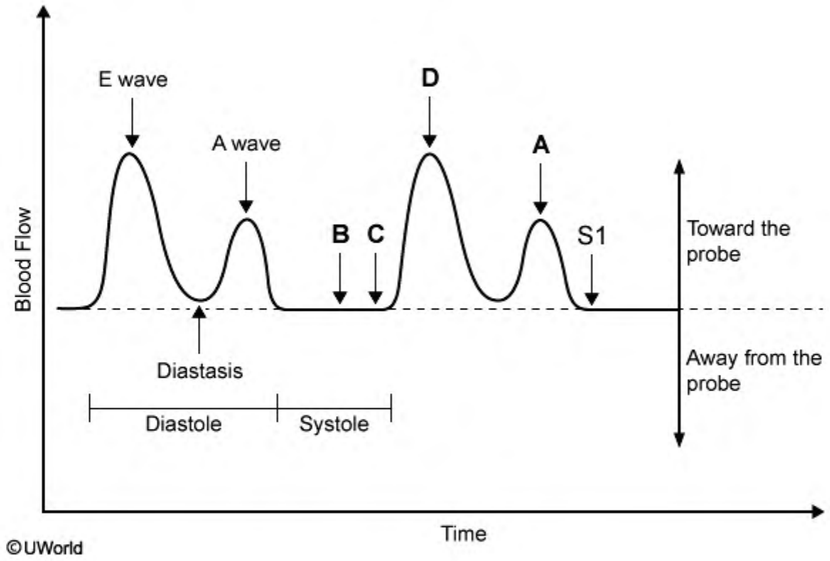

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ventricular movement **during passive ventricular filling**. An S3 may be a normal finding in healthy young adults;

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The graph shows the normal pattern of blood flow across the **mitral valve** from the left atrium to left ventricle during **diastole**. There is an initial phase of rapid transmitral blood flow after the mitral valve opens, which represents **early passive ventricular filling** (E wave). This is followed by a period of minimal blood flow (diastasis), which occurs due to equalization in pressure between the left atrium and ventricle. Finally, there is a second peak corresponding to increased transmitral blood flow due to **atrial contraction** (A wave). The A wave ends with the first heart sound (S1) caused by mitral (and tricuspid) valve closure.

When present, a **third heart sound** (S3) occurs during diastole and is best heard with the bell of the stethoscope over the cardiac apex with the patient in the left lateral decubitus position. It is due to sudden limitation of ventricular movement **during passive ventricular filling**. An S3 may be a normal finding in healthy young adults; however, in patients age >40 it suggests abnormal ventricular cavity enlargement (ie, eccentric hypertrophy). An S3 is often associated with chronic severe mitral regurgitation, chronic aortic regurgitation, or dilated cardiomyopathy; occasionally, it may be present in high cardiac output states such as pregnancy or thyrotoxicosis.

(Choice A) A fourth heart sound is a low-frequency, late diastolic sound coinciding with atrial systole. It is caused by a sudden rise in end-diastolic pressure as blood strikes a stiff left ventricular wall, and most commonly suggests concentric left ventricular hypertrophy such as occurs in prolonged hypertension or severe aortic stenosis.

(Choice B) A midsystolic click is associated with mitral valve prolapse. In patients with mitral valve prolapse and mitral regurgitation, there will be blood flow away from an apical Doppler probe during systole.

(Choice C) The second heart sound results from closure of the semilunar valves (aortic and pulmonic) at the end of systole.

Educational objective:
A third heart sound (S3) occurs due to sudden limitation of ventricular movement during passive ventricular filling in diastole. An S3 in patients age >40 suggests abnormal ventricular cavity enlargement such as occurs in chronic severe mitral regurgitation, chronic aortic regurgitation, or dilated cardiomyopathy.

Physiology	Cardiovascular System	Mitral regurgitation
Subject	System	Topic

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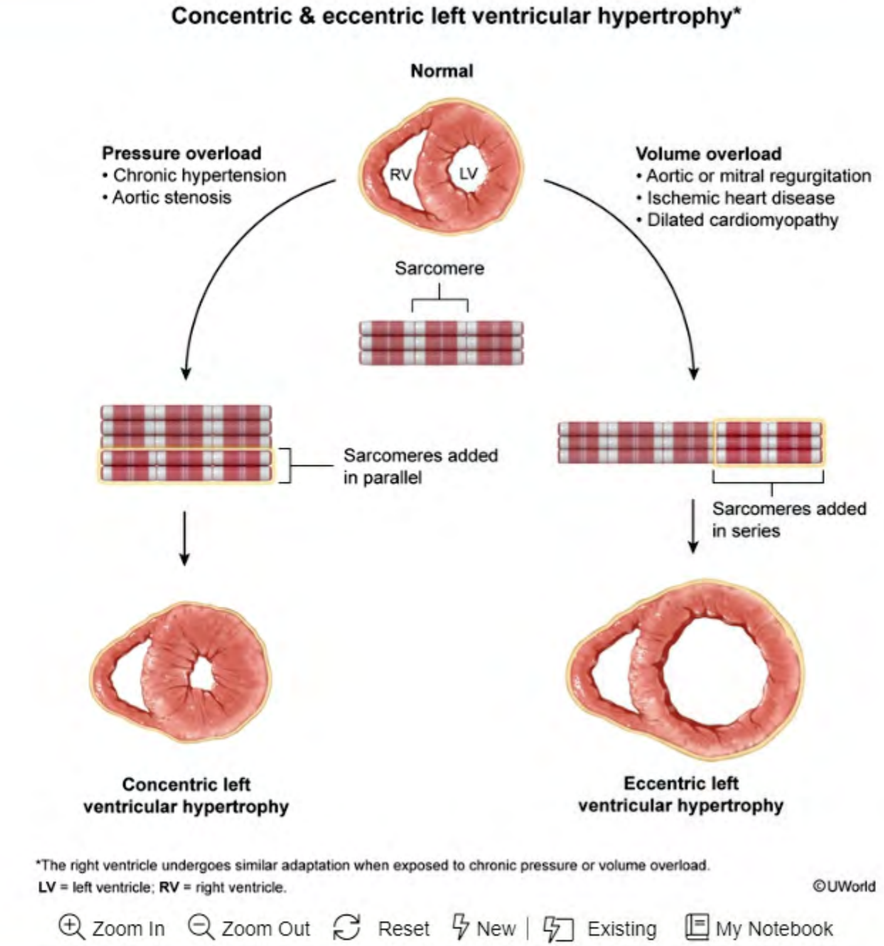

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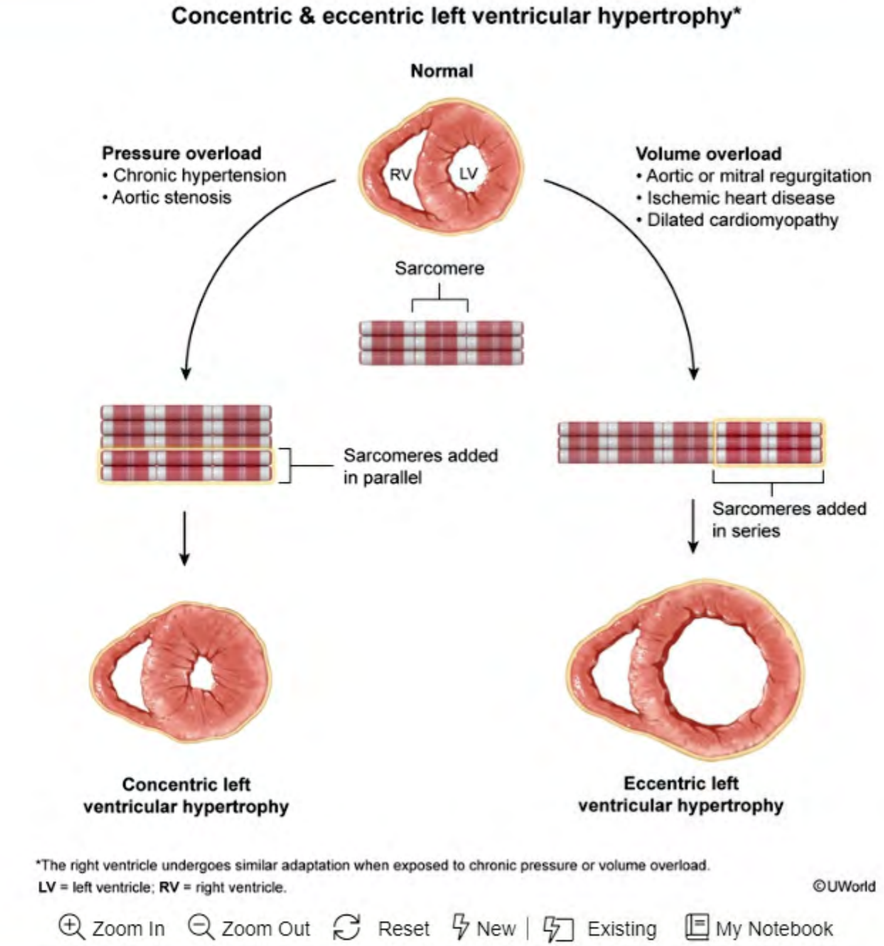

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Item 2 of 2

On physical examination, jugular venous pressure is elevated, breath sounds are decreased at the right lung base with dullness to percussion, and 2+ bilateral lower extremity pitting edema is present. Chest x-ray shows cephalization of the blood vessels, Kerley B lines, and a right pleural effusion. Thoracentesis is performed for pleural fluid analysis. In comparison to plasma fluid, which of the following pleural fluid findings is most likely to be observed?

- ✗ A. High amylase content (1%)
- B. High protein content (20%)
- C. High white blood cell count (3%)
- D. Low glucose content (13%)
- ✓ E. Low lactate dehydrogenase content (61%)

Incorrect
Correct answer
E

61%
Answered correctly

06 secs
Time Spent

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Explanation

Exudative & transudative pleural effusions		
	Exudate	**Transudate**
Light criteria	Pleural protein/serum protein >0.5 **OR** Pleural LDH/serum LDH >0.6 **OR**	Exudate criteria not met

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Exudative & transudative pleural effusions		
	Exudate	**Transudate**
Light criteria	Pleural protein/serum protein >0.5 **OR** Pleural LDH/serum LDH >0.6 **OR** Pleural LDH greater than two-thirds upper limit of normal of serum LDH	Exudate criteria not met
Pathophysiology	Inflammatory increase in membrane permeability	Change in hydrostatic or oncotic pressure
Common causes	• Infection (eg, pneumonia, TB) • Malignancy • Rheumatologic disease	• Heart failure • Cirrhosis (hepatic hydrothorax) • Nephrotic syndrome
LDH = lactate dehydrogenase; **TB** = tuberculosis.		

In determining the etiology of a **pleural effusion**, differentiating whether the effusion is transudative or exudative is an important step. The **Light criteria** allows differentiation of these two types of effusion via analysis of the protein and lactate dehydrogenase levels of the fluid.

Transudative pleural effusions form due to an imbalance in hydrostatic or oncotic pressure. **Heart failure** causes increased hydrostatic pressure in the pulmonary circulation due to backup of blood flow from the failing left ventricle. This increased pressure leads to leakage of fluid from the pulmonary capillaries and causes a **transudative pleural effusion**, which is characterized by **low protein** and **low lactate dehydrogenase** levels

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LDH = lactate dehydrogenase; **TB** = tuberculosis.

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Transudative pleural effusions form due to an imbalance in hydrostatic or oncotic pressure. **Heart failure** causes increased hydrostatic pressure in the pulmonary circulation due to backup of blood flow from the failing left ventricle. This increased pressure leads to leakage of fluid from the pulmonary capillaries and causes a **transudative pleural effusion**, which is characterized by **low protein** and **low lactate dehydrogenase** levels compared to serum values.

In contrast, exudative effusions result from inflammatory disruption of vascular permeability (eg, infection, malignancy) and demonstrate high protein levels **(Choice B)** due to increased capillary permeability and reduced sieving of proteins as fluid traverses the capillary wall. Lactate dehydrogenase levels also tend to be high in exudative effusions.

(Choice A) High amylase content is suggestive of a pleural effusion due to pancreatitis, pancreatic pseudocyst, or esophageal rupture.

(Choice C) High leukocyte count is typical of a complicated parapneumonic effusion or some types of malignant pleural effusion. Transudative effusions do not demonstrate high leukocyte counts.

(Choice D) Low glucose content is seen in the setting of malignant or infectious pleural effusion due to consumption of glucose by bacteria, neutrophils, and/or malignant cells.

Educational objective:
Heart failure can cause transudative pleural effusion due to an increase in pulmonary capillary hydrostatic pressure. Light criteria are useful to differentiate transudative and exudative pleural effusions; transudative effusions are characterized by low protein and lactate dehydrogenase content compared to serum values.

References

- The Light criteria: the beginning and why they are useful 40 years later.

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A 25-year-old man comes to the office due to shortness of breath. He states that even moderate exertion forces him to stop to catch his breath. The patient has no family history of sudden cardiac death. He does not use tobacco or illicit drugs. On examination, the patient has a faint systolic murmur at the left sternal border in the supine position that increases to 3/6 in intensity with Valsalva maneuver. Echocardiography shows interventricular septal hypertrophy and increased left ventricular mass. The patient is started on high-dose beta blocker therapy. On follow-up 2 weeks later, he reports significant improvement in his shortness of breath. Physical examination reveals no murmurs supine or with Valsalva. Which of the following best accounts for improvement of this patient's symptoms?

- A. Dilation of epicardial coronary arteries
- B. Increase in left ventricular outflow tract flow velocity
- C. Reduction in left ventricular contractility
- D. Reduction in left ventricular mass
- E. Reduction in left ventricular preload

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- ✗ A. Dilation of epicardial coronary arteries (2%)
- B. Increase in left ventricular outflow tract flow velocity (17%)
- ✓ C. Reduction in left ventricular contractility (63%)
- D. Reduction in left ventricular mass (3%)
- E. Reduction in left ventricular preload (12%)

Incorrect
Correct answer
C

63%
Answered correctly

03 secs
Time Spent

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Explanation

This patient's echocardiogram shows increased left ventricular (LV) mass with predominant interventricular **septal hypertrophy**, which are classic findings for **hypertrophic cardiomyopathy** (HCM). Many patients with HCM have LV outflow tract obstruction that can lead to symptoms of poor cardiac output (eg, dyspnea, syncope). The outflow tract **obstruction worsens** with **decreased LV blood volume** (as can occur during exercise due to decreased LV filling time and increased LV contractility); on physical examination, this effect is evidenced by a systolic murmur that increases in intensity with maneuvers that decrease LV blood volume (eg, Valsalva strain phase, abrupt standing)

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Explanation

This patient's echocardiogram shows increased left ventricular (LV) mass with predominant interventricular **septal hypertrophy**, which are classic findings for **hypertrophic cardiomyopathy** (HCM). Many patients with HCM have LV outflow tract obstruction that can lead to symptoms of poor cardiac output (eg, dyspnea, syncope). The outflow tract **obstruction worsens** with **decreased LV blood volume** (as can occur during exercise due to decreased LV filling time and increased LV contractility); on physical examination, this effect is evidenced by a systolic murmur that increases in intensity with maneuvers that decrease LV blood volume (eg, Valsalva strain phase, abrupt standing).

In patients with HCM and symptomatic LV outflow tract obstruction, **beta blockers** (eg, metoprolol, nadolol) improve symptoms mostly via an **increase in LV volume** that reduces LV outflow tract obstruction. This occurs via the following 2 mechanisms:

- Beta blockers **reduce heart rate**, which lengthens diastolic filling time to increase LV end-diastolic volume (ie, preload)
- Beta blockers **reduce LV contractility**, which decreases the amount of blood ejected during ventricular systole (ie, increased end-systolic volume)

Nondihydropyridine calcium channel blockers (eg, verapamil) have similar effects and can be used in patients with a contraindication to beta blockers or as additional therapy for persistent symptoms.

(Choice A) Some patients with HCM may experience anginal symptoms due to inadequate blood supply to the increased myocardial mass. Beta blockers may help alleviate these symptoms by decreasing LV wall stress to reduce myocardial oxygen demand and improve the coronary artery perfusion gradient; however, they have minimal dilatory effect on the coronary arteries.

(Choice B) By increasing the cross-sectional area of the LV outflow tract, beta blockers tend to decrease flow velocity (flow velocity = volumetric flow rate / cross-sectional area). Consequently, the intensity of the HCM murmur is also decreased.

(Choice D) Over time, beta blockers may reduce the compensatory LV hypertrophy that can occur in HCM due to

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(Choice B) By increasing the cross-sectional area of the LV outflow tract, beta blockers tend to decrease flow velocity (flow velocity = volumetric flow rate / cross-sectional area). Consequently, the intensity of the HCM murmur is also decreased.

(Choice D) Over time, beta blockers may reduce the compensatory LV hypertrophy that can occur in HCM due to prolonged contraction against the outflow tract obstruction (similar to aortic stenosis); however, this effect would not be evident within 2 weeks.

(Choice E) Beta blockers increase LV preload. Medications that reduce LV preload (eg, nitrates, diuretics) can worsen LV outflow tract obstruction and should be avoided in patients with HCM.

Educational objective:
Many patients with hypertrophic cardiomyopathy have exercise intolerance due to left ventricular (LV) outflow tract obstruction. This outflow obstruction is worsened by decreased LV blood volume. Beta blockers decrease heart rate and LV contractility to increase LV blood volume, reduce LV outflow tract obstruction, and improve symptoms.

Pharmacology Cardiovascular System Hypertrophic cardiomyopathy

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A full-term newborn is evaluated in the delivery room due to mild tachypnea and cyanosis immediately after birth. Fetal ultrasonography performed during pregnancy revealed transposition of the great arteries. Heart rate and blood pressure are normal. Oxygen saturation is 72%. Cyanosis is apparent in the hands, feet, lips, and tongue. Peripheral pulses are strong. Bedside echocardiography confirms the prenatal diagnosis; there is also a small patent foramen ovale and a small patent ductus arteriosus. Definitive surgical correction of the main congenital abnormality is planned. Pending definitive repair, which of the following temporary measures would be most likely to improve this patient's condition?

- A. Ablating the sinoatrial node
- B. Administering 100% oxygen
- C. Administering digoxin
- D. Enlarging the patent foramen ovale
- E. Ligating the patent ductus arteriosus

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- A. Ablating the sinoatrial node (0%)
- B. Administering 100% oxygen (15%)
- ✗ C. Administering digoxin (1%)
- ✓ D. Enlarging the patent foramen ovale (74%)
- E. Ligating the patent ductus arteriosus (8%)

Incorrect
Correct answer
D

74%
Answered correctly

03 secs
Time Spent

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Explanation

Transposition of the great arteries	
Anatomy	• Parallel (separate) systemic & pulmonary circuits ◦ Right ventricle → aorta ◦ Left ventricle → pulmonary artery • Deoxygenated blood is delivered to the systemic circulation

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Transposition of the great arteries	

Anatomy	• Parallel (separate) systemic & pulmonary circuits
	  ◦ Right ventricle → aorta
	  ◦ Left ventricle → pulmonary artery
	• Deoxygenated blood is delivered to the systemic circulation
	• Mixing shunt (eg, VSD, PDA) must be present for viability

Clinical features	• Cyanosis at or shortly after birth • Tachypnea & tachycardia • Murmur typically absent at birth due to low velocity shunting
Diagnosis	• Chest x-ray: narrow mediastinum ± cardiomegaly ("egg on a string") • Hyperoxia test: little to no ↑ in arterial oxygenation with 100% O_2 • Echocardiography typically confirmatory

Initial management	• Promote mixing of oxygenated & deoxygenated blood
	  ◦ Prostaglandin infusion (maintains PDA)
	  ◦ Atrial septostomy (maintains PFO)

PDA = patent ductus arteriosus; **PFO** = patent foramen ovale; **VSD** = ventricular septal defect.	

Transposition of the great arteries (TGA) is caused by **impaired spiraling** of the conotruncal septum (divides the truncus arteriosus into the aorta and the pulmonary artery), resulting in **reversed connections** between the ventricles and great arteries:

- The right ventricle empties into the aorta, causing deoxygenated mixed venous blood to be pumped directly into the systemic circulation (bypassing the lungs), leading to **neonatal cyanosis**
- The left ventricle ejects blood into the pulmonary artery, causing the pulmonary venous return to be pumped

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- The left ventricle ejects blood into the pulmonary artery, causing the pulmonary venous return to be pumped back into the lungs, **trapping oxygenated blood in the pulmonary circulation**

The parallel (ie, separate) circulations in TGA are incompatible with life unless a mixing shunt (eg, patent ductus arteriosus, patent foramen ovale, ventricular septal defect) is present to provide a path for partially oxygenated blood to enter the systemic circulation. Therefore, initial management of TGA involves **ensuring adequate mixing** of oxygenated and deoxygenated blood. For example, administration of prostaglandin E_1 to maintain patency of the ductus arteriosus or surgical procedures to open or **widen the foramen ovale**.

(Choice A) Sinoatrial node ablation is sometimes used for treating tachyarrhythmias involving the sinoatrial node.

(Choice B) Because deoxygenated blood bypasses the pulmonary circulation and is delivered directly to the systemic circulation in TGA, supplemental oxygen administration does not significantly improve hypoxemia or cyanosis.

(Choice C) Digoxin is a positive inotropic agent that can treat heart failure due to left ventricular systolic dysfunction (a finding not present on this patient's echocardiogram). Increased myocyte contractility would not improve this patient's oxygenation.

(Choice E) Ligating a patent ductus arteriosus in a patient with TGA would decrease mixing of blood between the parallel systemic and pulmonary circulations, worsening hypoxemia and cyanosis.

Educational objective:
Transposition of the great arteries is characterized by parallel circulations of deoxygenated and oxygenated blood. Initial management includes maintaining a mixing shunt (eg, patent ductus arteriosus, patent foramen ovale) to

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Educational objective:
Transposition of the great arteries is characterized by parallel circulations of deoxygenated and oxygenated blood. Initial management includes maintaining a mixing shunt (eg, patent ductus arteriosus, patent foramen ovale) to provide the systemic circulation with partially oxygenated blood.

References

- Transposition of the great arteries.

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- Atrial septostomy (maintains PFO)

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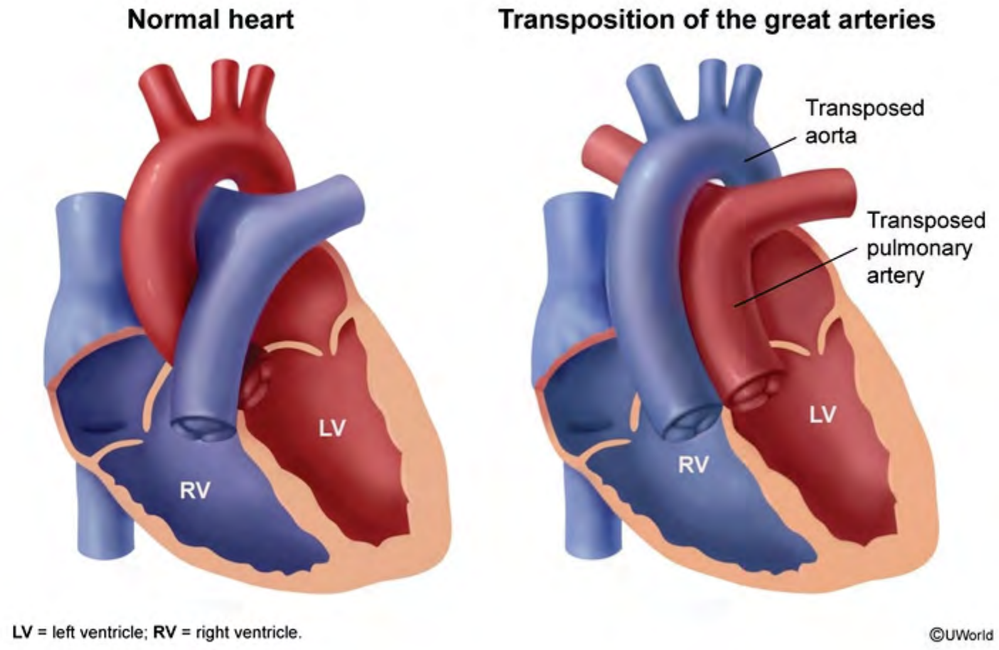

LV = left ventricle; **RV** = right ventricle.

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- Atrial septostomy (maintains PFO)

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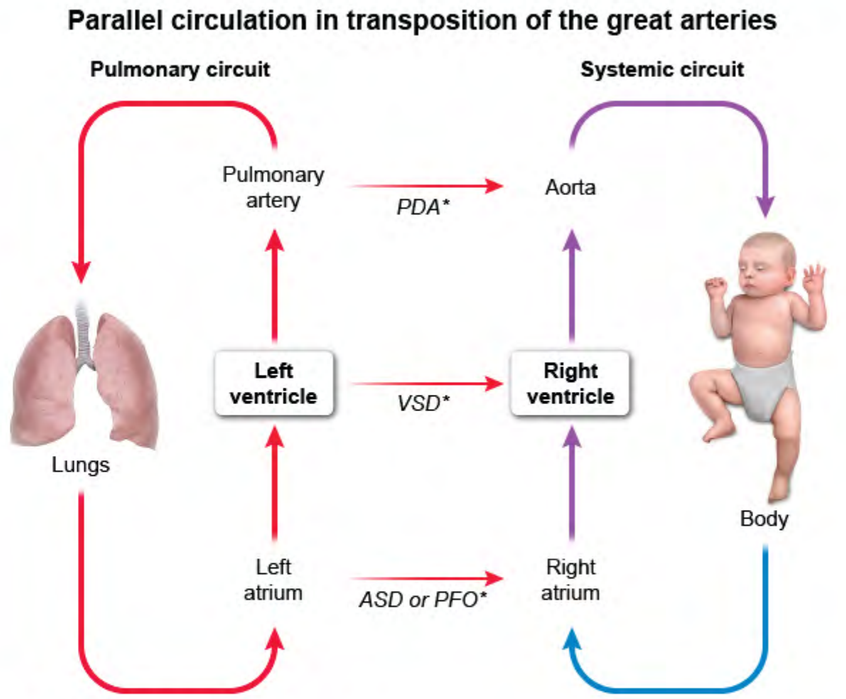

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A 35-year-old woman is brought to the emergency department due to severe right leg pain. She is a concert pianist with no known medical issues. During rehearsal, she began experiencing cramping of the right foot that did not improve with stretching or rest. The pain quickly spread to her calf, and the patient now rates the pain as 9 out of 10 in intensity. On examination, the right foot and calf appear pale and cool compared with the left, and sensation is diminished. Right-sided dorsalis pedis and posterior tibial pulses are absent, and the popliteal pulse is barely palpable. Emergency embolectomy is performed and a gelatinous mass causing arterial occlusion is successfully removed. Histopathologic section of the mass is shown in the exhibit. Which of the following is the most likely origin of this patient's embolus?

- A. Aortic valve
- B. Descending aorta
- C. Left atrium
- D. Left ventricle
- E. Mitral valve

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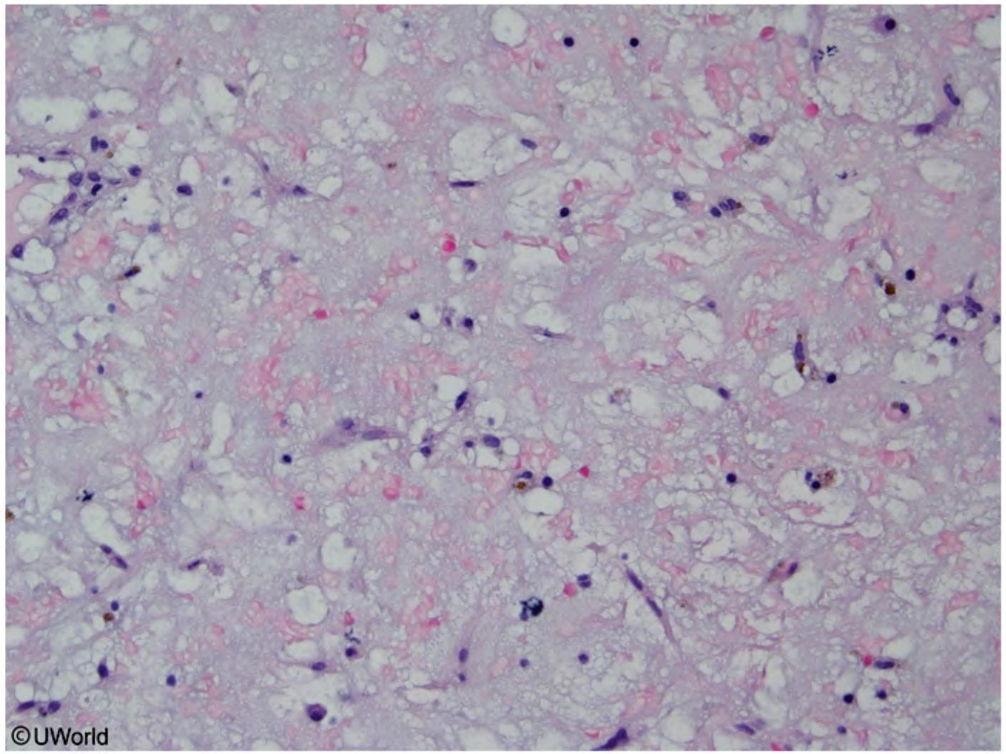

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- A. Aortic valve (3%)
- B. Descending aorta (14%)
- ✓ C. Left atrium (62%)
- D. Left ventricle (2%)
- E. Mitral valve (17%)

Correct

62% Answered correctly

01 min, 25 secs Time Spent

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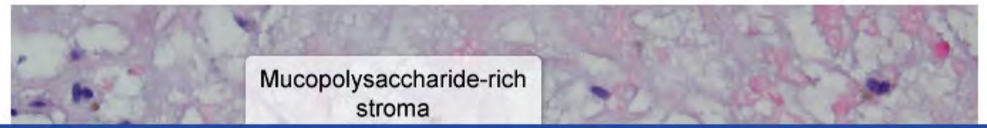
Explanation

Cardiac myxoma



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D. Left ventricle (2%)

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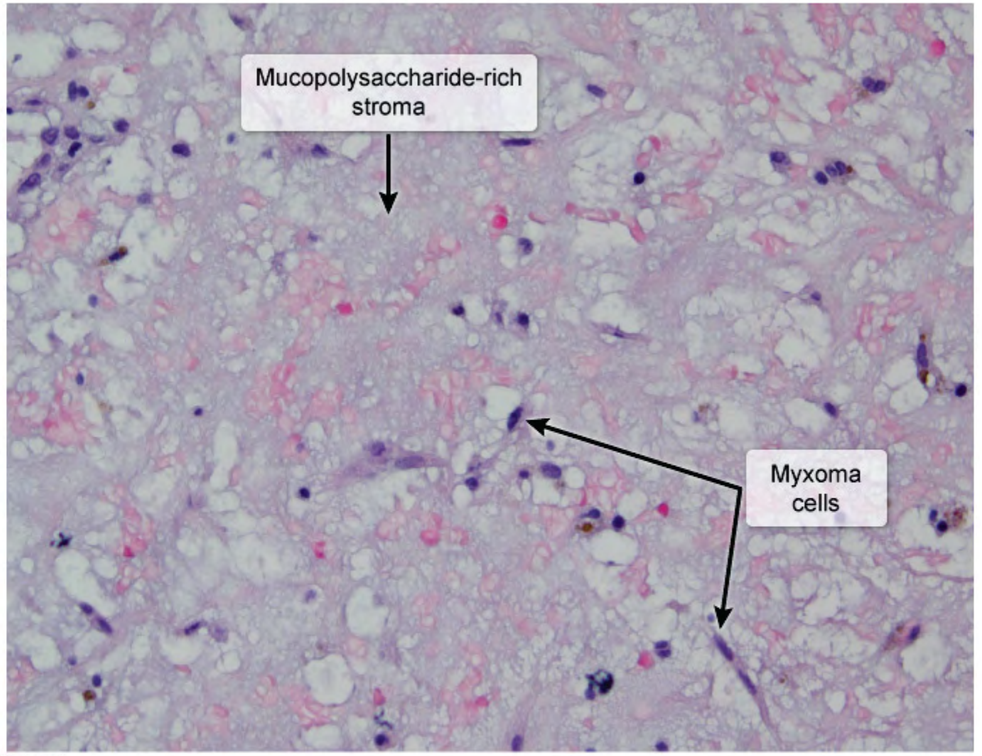
Cardiac myxoma



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This patient presented with **acute limb ischemia**, as evidenced by the **6 Ps**: pain, pallor, poikilothermia (coolness to touch), paresthesia, paralysis, and reduced or absent pulses. The ischemia resulted from **systemic embolization** of a gelatinous mass, with histopathology demonstrating an **amorphous extracellular matrix** with scattered stellate myxoma cells in mucopolysaccharide ground substance; this is consistent with tumor fragments from an **atrial myxoma**.

Myxomas are the most common primary cardiac neoplasm. Approximately 80% of these benign tumors originate in the **left atrium**. Patients can initially present with nonspecific constitutional symptoms (eg, fatigue, low-grade fever, weight loss) or systemic embolization (eg, stroke, mesenteric ischemia, acute limb ischemia), as in this patient. In addition, the tumor can cause position-dependent obstruction of the mitral valve, leading to signs and symptoms that **mimic mitral valve stenosis** (eg, dyspnea, cough, mid-diastolic murmur).

(Choices A and E) The aortic and mitral valves can be the source of systemic embolism due to vegetations from bacterial endocarditis or nonbacterial thrombotic endocarditis (Libman-Sacks endocarditis). However, only 5% of myxomas originate on the mitral valve and even fewer originate on the aortic valve.

(Choice B) A complex atherosclerotic plaque or mural thrombus attached to the walls of the descending aorta can lead to systemic embolization to the kidneys, bowel, or lower extremities. However, myxomas very rarely originate from the aorta.

(Choice D) A mural thrombus can originate in the left ventricular cavity in patients with severe left ventricular systolic dysfunction, especially with recent anterior wall myocardial infarction. Other than the left atrium, the right atrium is the most common site for myxoma development. It is quite rare for myxomas to originate in either ventricle.

Educational objective:
Myxomas are the most common primary cardiac neoplasm and approximately 80% originate in the left atrium. Patients may present with symptomatic mitral valve obstruction that may worsen with certain body positions.

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to touch), paresthesia, paralysis, and reduced or absent pulses. The ischemia resulted from **systemic embolization** of a gelatinous mass, with histopathology demonstrating an **amorphous extracellular matrix** with scattered stellate myxoma cells in mucopolysaccharide ground substance; this is consistent with tumor fragments from an **atrial myxoma**.

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Educational objective:
Myxomas are the most common primary cardiac neoplasm and approximately 80% originate in the left atrium. Patients may present with symptomatic mitral valve obstruction that may worsen with certain body positions, constitutional findings (eg, fever, weight loss), or systemic embolization (eg, stroke, mesenteric ischemia, acute limb ischemia).

References

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A 20-year-old, previously healthy woman is brought to the emergency department after a motor vehicle collision. The patient was a restrained, rear-seat passenger when another car struck the side door. There was extensive damage to the vehicle, resulting in a prolonged extrication time. On arrival at the emergency department, blood pressure is 90/60 mm Hg and pulse is 120/min. On physical examination, the patient is alert but appears anxious, pale, and diaphoretic. There is no obvious head or neck injury. The lungs are clear bilaterally and cardiac auscultation reveals tachycardia. The abdomen is soft. Several actively bleeding lacerations are present on the lower extremities. X-rays show pelvic and left femoral shaft fractures. Which of the following findings are most likely present in this patient?

	Cardiac contractility	Baroreceptor firing	Atrial natriuretic peptide release
A.	↑	↑	↑
B.	↓	↑	↑
C.	↑	↓	↑
D.	↓	↑	↓
E.	↑	↓	↓
F.	↓	↓	↓
G.	↓	↓	↑
H.	↑	↑	↓

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A 20-year-old, previously healthy woman is brought to the emergency department after a motor vehicle collision. The patient was a restrained, rear-seat passenger when another car struck the side door. There was extensive damage to the vehicle, resulting in a prolonged extrication time. On arrival at the emergency department, blood pressure is 90/60 mm Hg and pulse is 120/min. On physical examination, the patient is alert but appears anxious, pale, and diaphoretic. There is no obvious head or neck injury. The lungs are clear bilaterally and cardiac auscultation reveals tachycardia. The abdomen is soft. Several actively bleeding lacerations are present on the lower extremities. X-rays show pelvic and left femoral shaft fractures. Which of the following findings are most likely present in this patient?

	Cardiac contractility	Baroreceptor firing	Atrial natriuretic peptide release	
✗ A.	↑	↑	↑	(5%)
B.	↓	↑	↑	(1%)
C.	↑	↓	↑	(4%)
D.	↓	↑	↓	(3%)
✓ E.	↑	↓	↓	(52%)
F.	↓	↓	↓	(5%)
G.	↓	↓	↑	(0%)
H.	↑	↑	↓	(24%)

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Correct answer

52%
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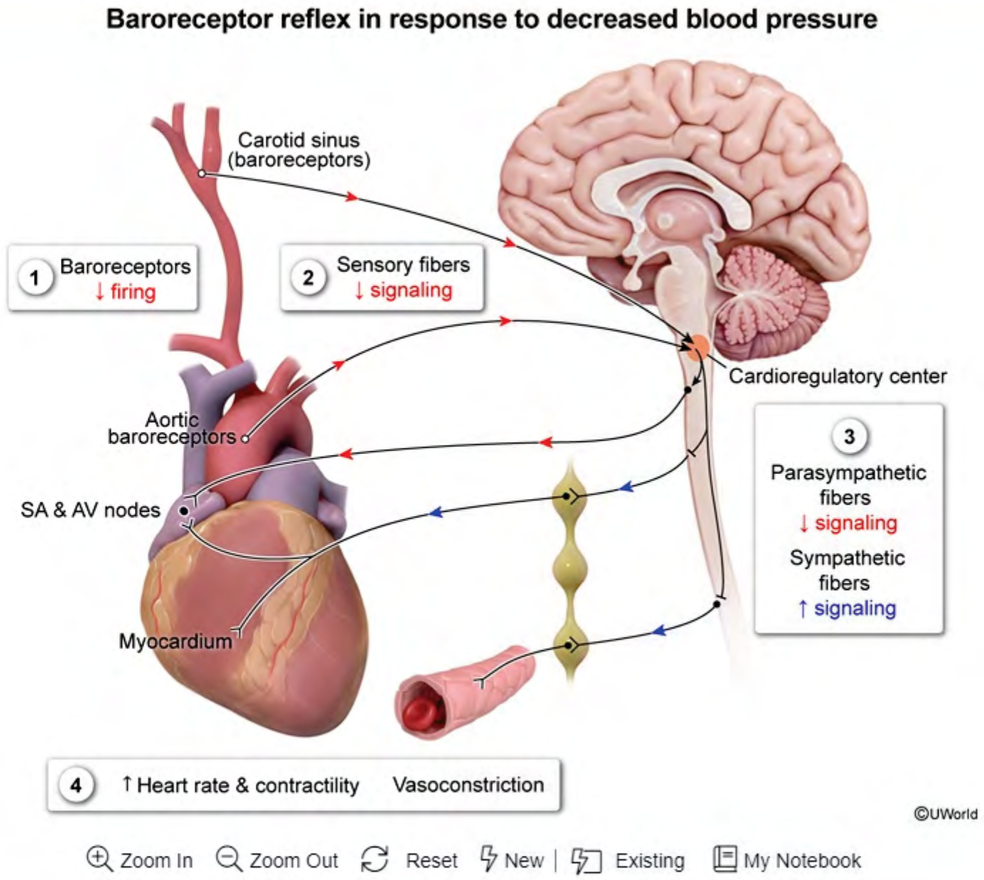

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cause of severe internal bleeding) are consistent with hypovolemic shock due to **massive hemorrhage**. Following

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This patient's **hypotension** and tachycardia in the setting of multiple lacerations and pelvic fracture (a common cause of severe internal bleeding) are consistent with hypovolemic shock due to **massive hemorrhage**. Following significant intravascular volume depletion, baroreceptors in the aorta and carotid arteries rapidly adjust autonomic output to help maintain blood pressure.

Baroreceptors are stretch-sensitive mechanoreceptors that increase their firing rate in response to arterial distension. These impulses are transmitted to the solitary nucleus, which then modulates sympathetic and parasympathetic output in response to the firing rate.

With an **abrupt decrease** in cardiac output and blood pressure (eg, acute blood loss, standing from a seated position), reduced stretch of the arterial walls leads to **reduced baroreceptor firing** and decreased activation of the solitary nucleus. This causes an increase in sympathetic outflow with a corresponding reduction in parasympathetic tone, leading to **vasoconstriction** (increases peripheral resistance) and **increased heart rate and contractility** (improves cardiac output), minimizing the drop in blood pressure.

Via a similar mechanism, stretch receptors in the atrial walls of the heart control secretion of atrial natriuretic peptide (ANP). With hypovolemia there is **reduced atrial stretch** that leads to **decreased ANP secretion**.

(Choices A, F, G, and H) Because baroreceptor firing reduces sympathetic output, the degree of baroreceptor firing is usually inversely related to cardiac contractility.

(Choice B) In hypervolemic states, blood pressure is increased, leading to increased baroreceptor firing with a decrease in sympathetic outflow and corresponding increase in parasympathetic outflow. A resulting decrease in vascular tone, heart rate, and cardiac contractility reduces blood pressure. Increased stretching of the atrial walls leads to increased ANP release, which promotes vasodilation and diuresis.

(Choices C and D) Because conditions that increase (eg, hypervolemia) or decrease (eg, hypovolemia) baroreceptor stretch typically have the same effect on atrial stretch, the degree of baroreceptor firing is usually directly related to the level of ANP release.

Educational objective:

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distension. These impulses are transmitted to the solitary nucleus, which then modulates sympathetic and parasympathetic output in response to the firing rate.

With an **abrupt decrease** in cardiac output and blood pressure (eg, acute blood loss, standing from a seated position), reduced stretch of the arterial walls leads to **reduced baroreceptor firing** and decreased activation of the solitary nucleus. This causes an increase in sympathetic outflow with a corresponding reduction in parasympathetic tone, leading to **vasoconstriction** (increases peripheral resistance) and **increased heart rate and contractility** (improves cardiac output), minimizing the drop in blood pressure.

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Educational objective:
Low blood pressure results in decreased arterial distension and reduced firing of aortic and carotid baroreceptors. This leads to an increase in sympathetic outflow and decrease in parasympathetic outflow, stimulating vasoconstriction and increased heart rate and contractility in order to help maintain blood pressure. Hypovolemia reduces atrial stretch, causing decreased atrial natriuretic peptide secretion.

References

- Physiology, baroreceptors.

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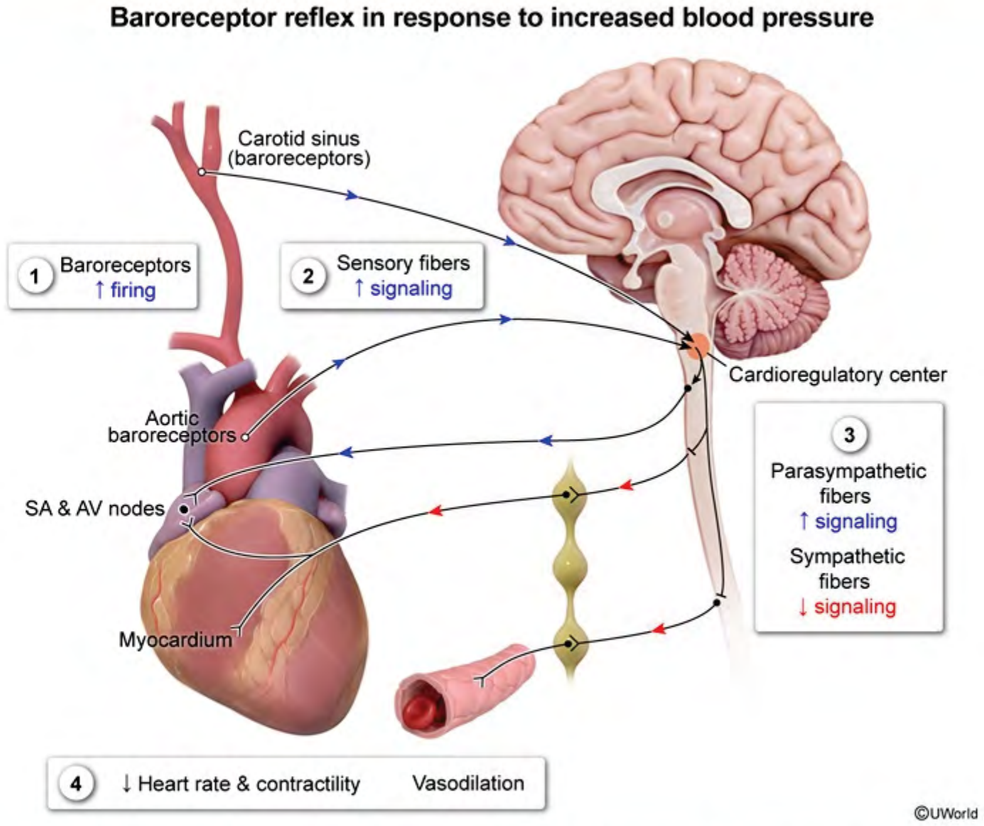

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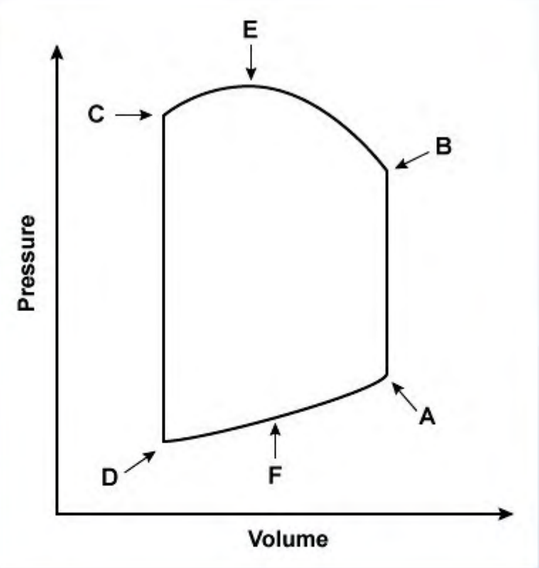
A 34-year-old woman who recently emigrated from Russia comes to the physician due to weakness, exertional dyspnea, and orthopnea. On cardiac auscultation, a snap followed by a rumbling diastolic murmur is heard over the cardiac apex. The snap most likely occurs nearest to which of the following points on the cardiac pressure-volume loop?



- A. A
- B. B
- C. C
- D. D
- E. E
- F. F

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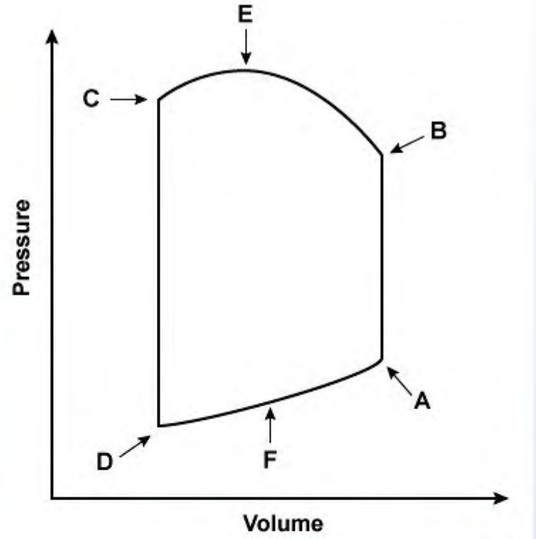
the cardiac apex. The snap most likely occurs nearest to which of the following points on the cardiac pressure-volume loop?



- A. A (11%)
- B. B (5%)
- ✗ C. C (9%)
- ✓ D. D (50%)
- E. E (3%)
- F. F (19%)

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Correct answer
D

50%
Answered correctly

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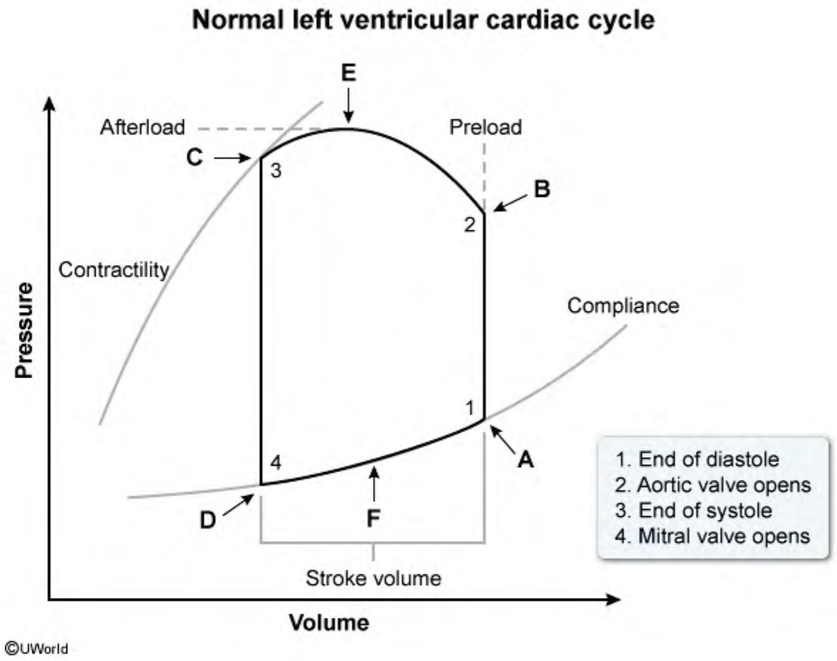

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1. **Isovolumetric contraction:** The cardiac cycle begins with initiation of left ventricular contraction and closure

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The **left ventricular (LV) pressure-volume** loop follows the cardiac cycle through left ventricular contraction, ejection, relaxation, and refilling, as follows:

1. **Isovolumetric contraction:** The cardiac cycle begins with initiation of left ventricular contraction and closure of the mitral valve at point 1; an S1 is heard at this time, marking the end of diastole **(Choice A)**. Although intraventricular pressure increases after point 1, initially, no blood leaves the ventricle because both the aortic and mitral valves are closed. At point 2, LV pressure overcomes systemic diastolic pressure and the aortic valve opens **(Choice B)**.
2. **Ventricular ejection:** LV volume decreases as blood is ejected into the aorta. The peak of the ejection curve denotes systolic blood pressure and represents the high point of LV afterload **(Choice E)**. LV ejection ends at point 3 with aortic valve closure and an S2 is heard **(Choice C)**. The horizontal distance between point 2 (or point 1) and point 3 represents the stroke volume.
3. **Isovolumetric relaxation:** LV pressure rapidly falls from point 3 to point 4. No blood leaves or enters the left ventricle during this time because both the aortic and mitral valves are closed.
4. **Ventricular filling:** The mitral valve opens at point 4 and diastolic filling of the left ventricle begins. LV volume and pressure gradually increase until mitral valve closure occurs again at point 1 and the cycle begins anew. Extra heart sounds (ie, an S3 or S4) may be heard during diastolic filling between point 4 and point 1 **(Choice F)**.

This patient with an audible snap followed by a **rumbling diastolic murmur** over the cardiac apex likely has **mitral stenosis**, which is most commonly seen due to underlying rheumatic heart disease in patients emigrating from Latin America, Africa, or Asia. The audible snap observed with mitral stenosis is created by the stenotic and calcified valve snapping open (ie, **opening snap**) and is heard during mitral valve opening at point 4.

Educational objective:
Mitral stenosis is characterized by an opening snap followed by a rumbling diastolic murmur that is best heard over the cardiac apex. On the left ventricular pressure-volume loop, the opening snap occurs during mitral valve opening at the end of isovolumetric relaxation, and it is followed by a diastolic rumble during diastolic filling.

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of the mitral valve at point 1; an S1 is heard at this time, marking the end of diastole **(Choice A)**. Although intraventricular pressure increases after point 1, initially, no blood leaves the ventricle because both the aortic and mitral valves are closed. At point 2, LV pressure overcomes systemic diastolic pressure and the aortic valve opens **(Choice B)**.

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Educational objective:
Mitral stenosis is characterized by an opening snap followed by a rumbling diastolic murmur that is best heard over the cardiac apex. On the left ventricular pressure-volume loop, the opening snap occurs during mitral valve opening at the end of isovolumetric relaxation, and it is followed by a diastolic rumble during diastolic filling.

Physiology	Cardiovascular System	Mitral stenosis
Subject	System	Topic

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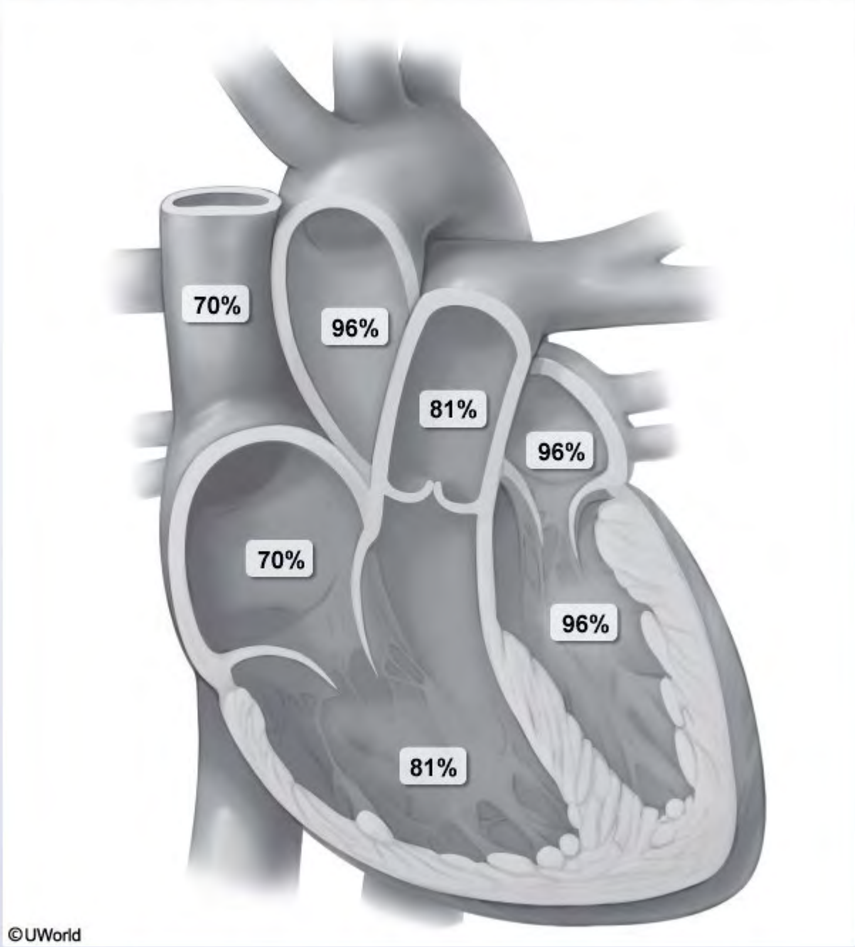
Cardiac catheterization is performed on a 7-year-old boy. The image below shows the pattern of oxygen saturation in the patient's cardiac chambers and outflow tracts (structural abnormalities are not shown).



This patient most likely has which of the following findings on physical examination?

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Cardiac catheterization is performed on a 7-year-old boy. The image below shows the pattern of oxygen saturation in the

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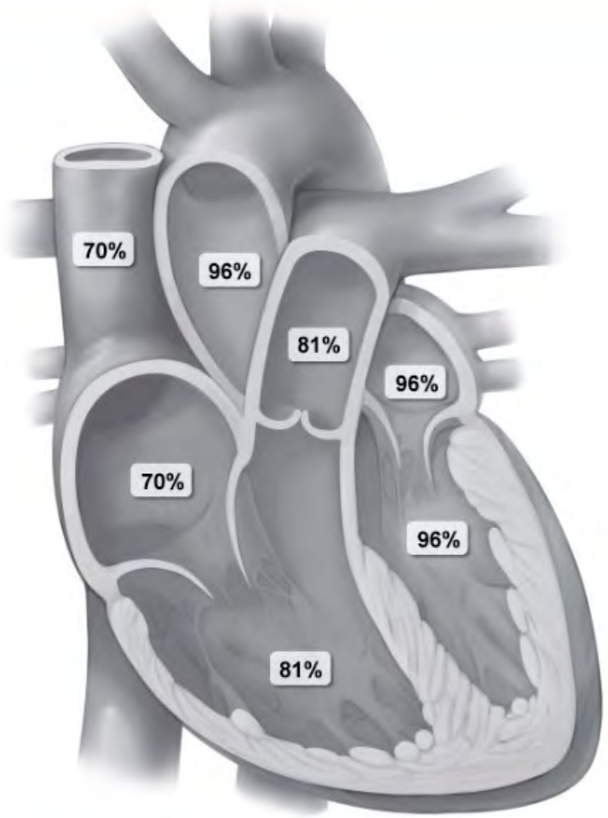

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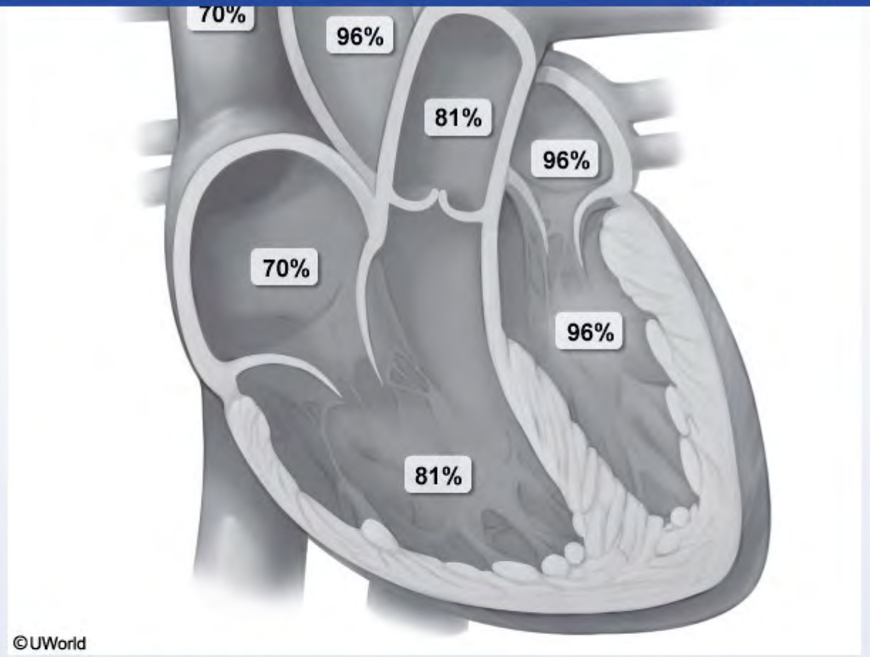

This patient most likely has which of the following findings on physical examination?

- A. Bifid carotid pulse with brisk upstroke
- B. Continuous murmur over the upper precordium
- C. Decreased femoral-to-brachial blood pressure ratio
- D. Fixed splitting of S2 on cardiac auscultation
- E. Holosystolic murmur over the left sternal border
- F. Mucosal cyanosis and fingernail clubbing

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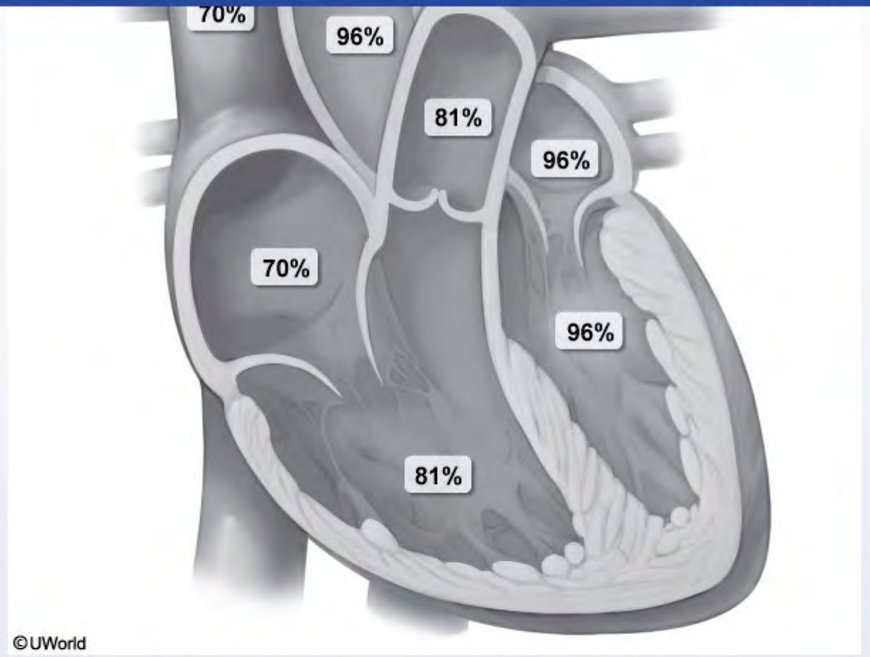

This patient most likely has which of the following findings on physical examination?

- ✗ A. Bifid carotid pulse with brisk upstroke (1%)
- B. Continuous murmur over the upper precordium (10%)
- C. Decreased femoral-to-brachial blood pressure ratio (3%)
- D. Fixed splitting of S2 on cardiac auscultation (17%)
- ✓ E. Holosystolic murmur over the left sternal border (61%)
- F. Mucosal cyanosis and fingernail clubbing (5%)

Incorrect

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Correct answer
E

61%
Answered correctly

10 secs
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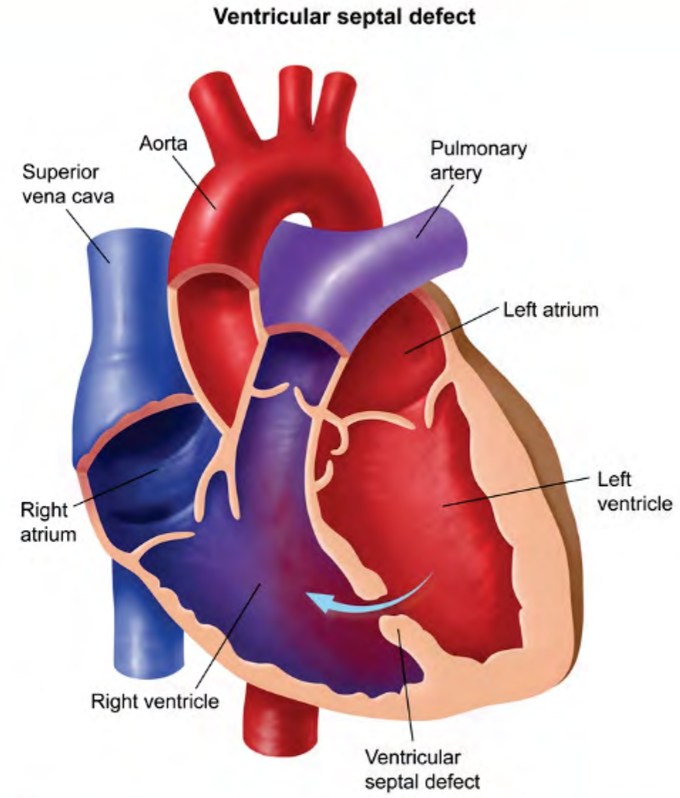

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This patient has an **abnormal increase in oxygen saturation** (SpO_2) between the right atrium and the right ventricle (see normal oxygenation pattern), indicating the presence of a **left-to-right shunt** that is most likely the result of a **ventricular septal defect** (VSD). With smaller VSDs, blood is shunted mainly from left to right due to the high pressure differential between the ventricles. As a result, **right ventricular SpO_2 increases** but left ventricular SpO_2 remains relatively normal. Larger VSDs allow equalization of chamber pressures, which results in more bidirectional mixing and decreased left ventricular SpO_2. Right atrial SpO_2 remains normal with VSDs of any size, unless tricuspid regurgitation is also present.

Turbulent blood flow across a small VSD produces a loud **holosystolic murmur** best heard over the **lower left sternal border**. This murmur may be absent in larger (nonrestrictive) VSDs due to decreased resistance across the VSD and a lower trans-septal pressure gradient.

(Choice A) A bifid carotid pulse with brisk upstroke ("spike-and-dome") is characteristic of hypertrophic cardiomyopathy, a condition with dynamic left ventricular outflow tract obstruction during systole. SpO_2 in the cardiac chambers remains normal.

(Choice B) A precordial continuous, machine-like murmur indicates patent ductus arteriosus, which produces isolated pulmonary artery SpO_2 elevation.

(Choice C) A decreased femoral-to-brachial blood pressure ratio is found in coarctation of the aorta, which does not alter right ventricular SpO_2.

(Choice D) Fixed splitting of S2 occurs with an atrial septal defect (ASD), which causes increased SpO_2 in the right atrium compared with the vena cava but identical right atrial and ventricular SpO_2. VSD may result in *wide* splitting of S2 due to increased right ventricular volume causing delayed closure of the pulmonary valve; however, *fixed* splitting of S2 is more indicative of an ASD, as equalization of left and right atrial pressures minimizes the respiratory variation in ventricular blood flow.

(Choice F) Mucosal cyanosis and fingernail clubbing may be observed in cyanotic congenital heart disease or as late features of ASDs or VSDs (eg, Eisenmenger syndrome). Blood flow across the VSD in Eisenmenger

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the VSD and a lower trans-septal pressure gradient.

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(Choice F) Mucosal cyanosis and fingernail clubbing may be observed in cyanotic congenital heart disease or as late features of ASDs or VSDs (eg, Eisenmenger syndrome). Blood flow across the VSD in Eisenmenger syndrome is right to left due to elevated pulmonary vascular resistance, resulting in decreased SpO_2 in the left ventricle.

Educational objective:
Increased blood oxygen saturation between 2 right-sided vessels or chambers indicates the presence of a left-to-right shunt. If the abnormal oxygen increase occurs between the right atrium and the right ventricle, a ventricular septal defect (VSD) is likely present. Small VSDs produce a holosystolic murmur that is loudest over the lower left sternal border.

References

- Shunt lesions.

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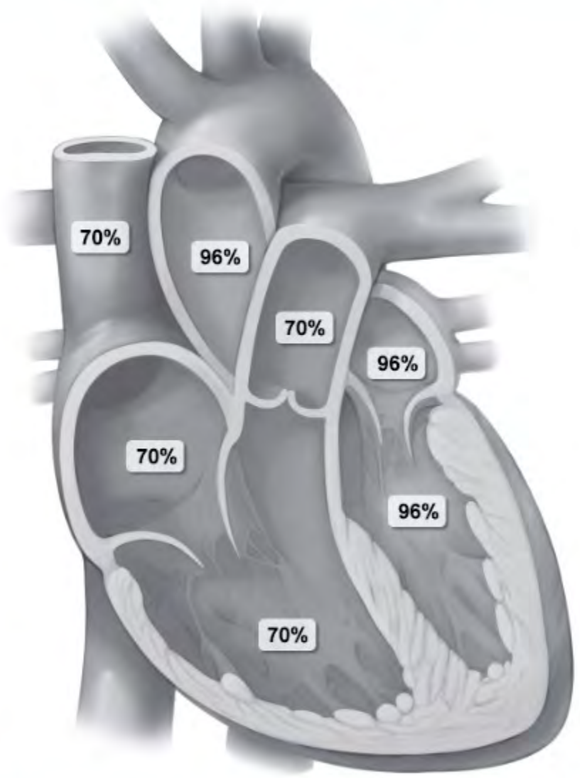
Oxygen saturation: normal heart



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fixed splitting of S2 is more indicative of an ASD, as equalization of left and right atrial pressures minimizes the

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the high pressure differential between the ventricles. As a result, **right ventricular SpO_2 increases** but left ventricular SpO_2 remains relatively normal. Larger VSDs allow equalization of chamber pressures, which results in more b
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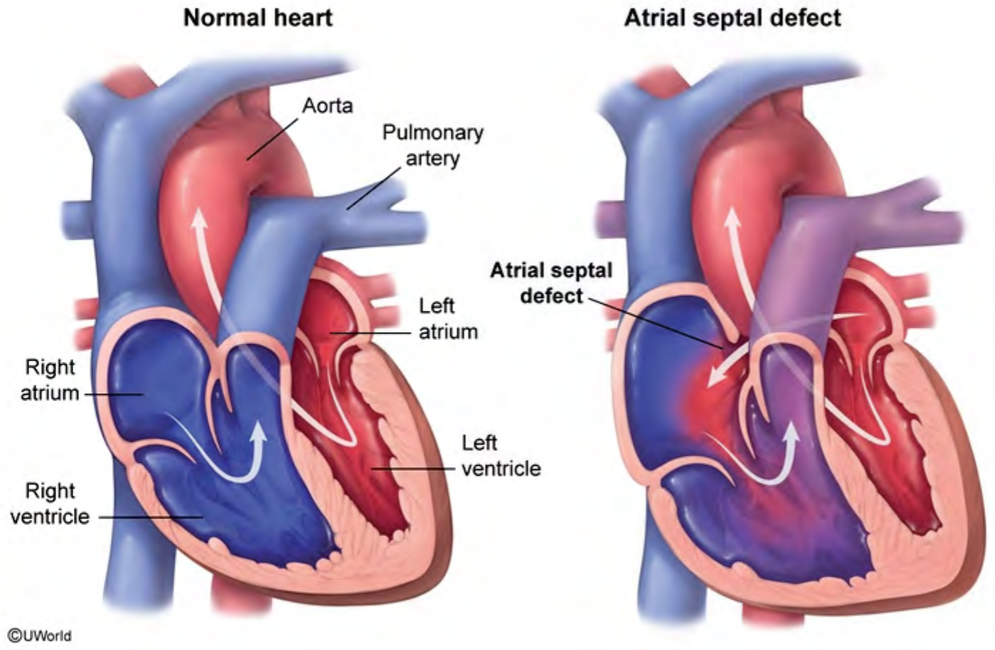

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Educational objective:

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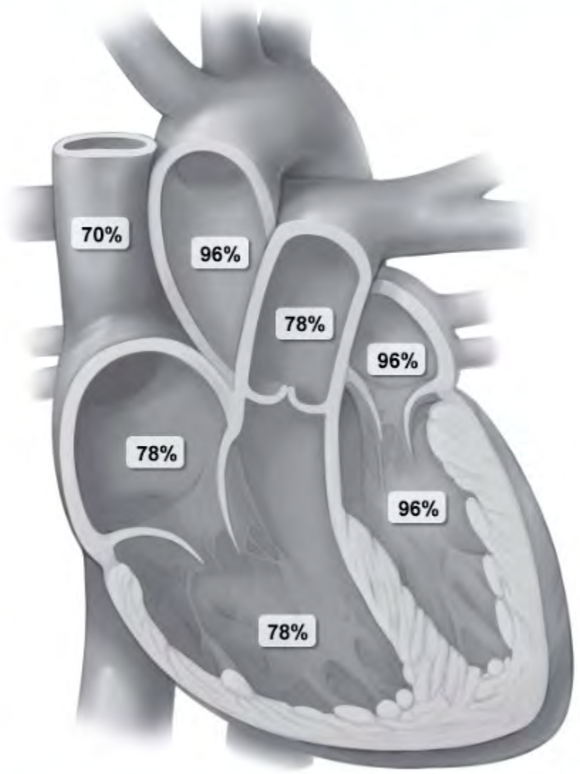
Oxygen saturation: atrial septal defect



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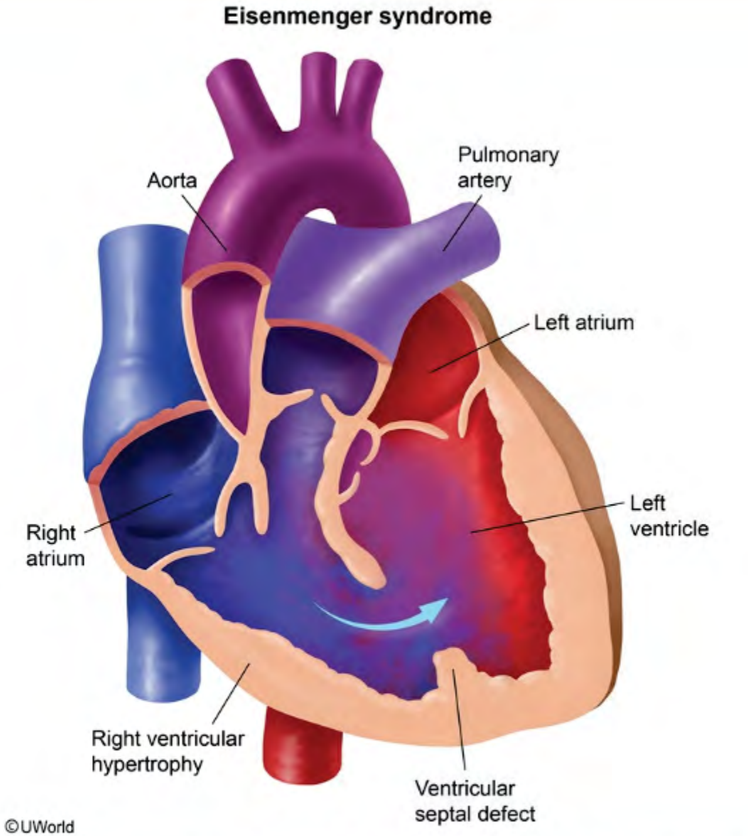

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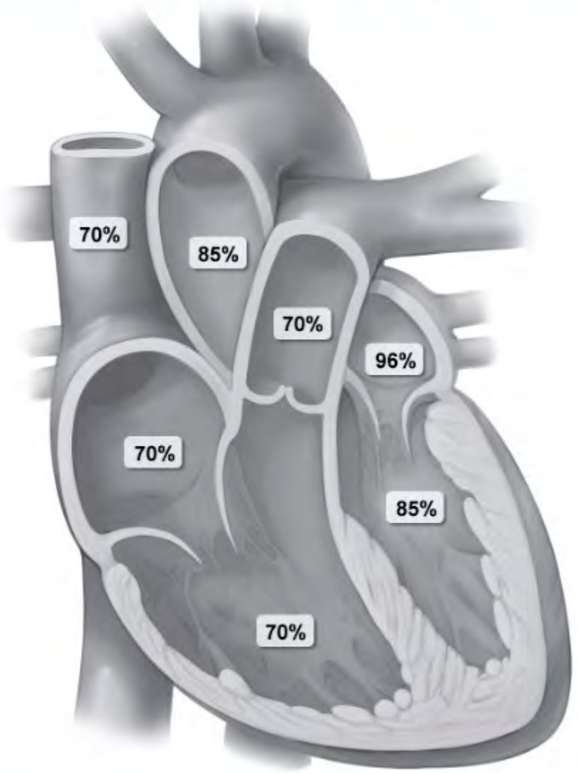
Oxygen saturation: Eisenmenger syndrome



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A newborn boy delivered 2 hours ago is evaluated for cyanosis. Physical examination is significant for cyanosis of the upper and lower extremities, the trunk, and the mucous membranes. S1 is widely split, and there is a 2/6 holosystolic murmur best heard at the left lower sternal border that increases in intensity with inspiration. Pulses are 1+ and equal in all extremities. The lungs are clear to auscultation. Chest x-ray reveals an enlarged heart. Which of the following is most likely present in this patient?

- A. Apical displacement of the tricuspid valve
- B. Failed fusion of the ventricular septum
- C. Narrowing of the preductal aorta
- D. Prolapse of the mitral valve

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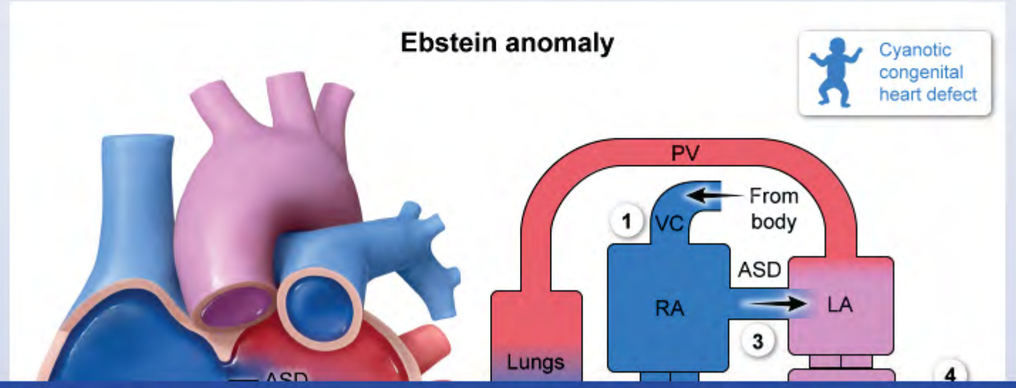

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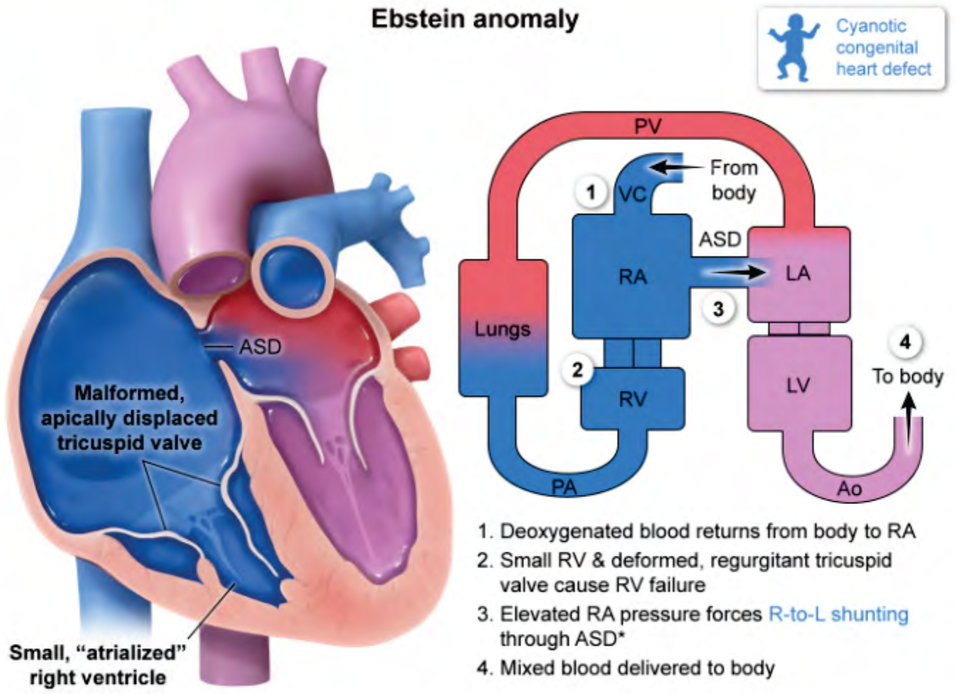

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R-to-L shunting and are acyanotic.

Ao = aorta; **ASD** = atrial septal defect; **LA** = left atrium; **LV** = left ventricle; **PA** = pulmonary arteries; **PV** = pulmonary veins; **RA** = right atrium; **R-to-L** = right-to-left; **RV** = right ventricle; **VC** = vena cava.

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This patient with cyanosis at birth, a widely split S1, and a murmur at the left lower sternal border consistent with tricuspid regurgitation likely has **Ebstein anomaly**. This congenital cardiac defect involves malformation and **apical displacement of the tricuspid valve**, resulting in "atrialization" of the superior portion of the right ventricle (RV) and a small functional portion of the RV. Affected patients develop tricuspid regurgitation and **right-sided heart failure** at different points in life (eg, birth, childhood, early adulthood) depending on the severity of the defect.

When severe, Ebstein anomaly commonly presents with **cyanosis at birth**, resulting from abrupt RV failure and markedly elevated right-sided pressures forcing **right-to-left shunting** through an atrial septal defect (present in two-thirds of patients) or patent foramen ovale. Cyanosis is rare beyond infancy, but the characteristic cardiac auscultation findings of Ebstein anomaly are usually present: **wide splitting of S1** (due to delayed closure of the malformed tricuspid valve), an early systolic click (ie, **sail sound** produced by the malformed tricuspid valve), and a murmur of tricuspid regurgitation.

(Choice B) Failed fusion of the ventricular septum results in a ventricular septal defect (VSD). A VSD causes a holosystolic murmur at the left lower sternal border, but splitting of S1 is not expected. In addition, a VSD does not cause cyanosis at birth (cyanosis may develop years later in the form of Eisenmenger syndrome).

(Choice C) Narrowing of the preductal aorta occurs with coarctation of the aorta. Severe coarctation may cause right-to-left shunting through the ductus arteriosus at birth, resulting in differential cyanosis affecting only the lower extremities (this patient's cyanosis affects the entire body). Weak lower extremity pulses in comparison to strong upper extremity pulses are also expected.

(Choice D) Mitral valve prolapse commonly results from a congenital deformity of the mitral valve that leads to impaired valve closure. Typical cardiac auscultation findings are a midsystolic click followed by a mitral regurgitation murmur. It can present with dyspnea during adulthood, but it rarely causes symptoms in neonates and does not cause cyanosis.

Educational objective:

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Educational objective:
Ebstein anomaly is a congenital heart defect that involves malformation and apical displacement of the tricuspid valve, creating tricuspid regurgitation and a small functional right ventricle. Patients with severe Ebstein anomaly commonly are cyanotic at birth, which results from abrupt right ventricular failure and right-to-left shunting through an atrial septal defect or patent foramen ovale.

Pathophysiology	Cardiovascular System	Congenital cardiac defects
Subject	System	Topic

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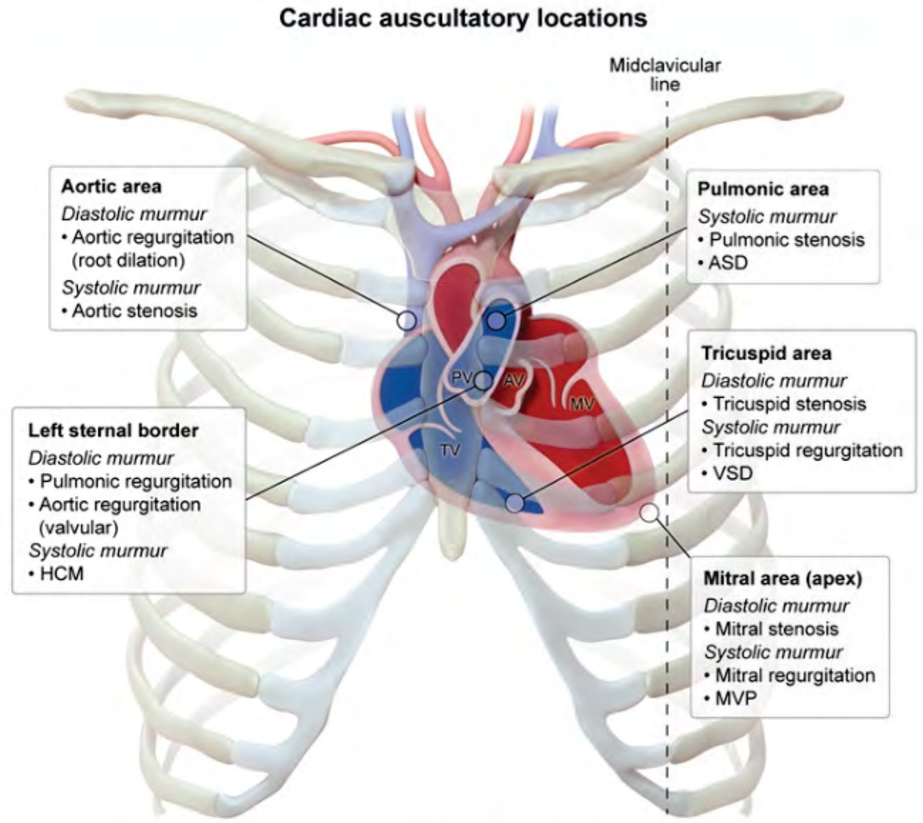

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Cardiac murmurs

Systolic	Aortic stenosis*	S1 A2 P2 S1	Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*	S1 S2 S1	Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy	S1 S2 S1	Crescendo-decrescendo at mid–left sternal border
	Mitral valve prolapse	S1 Click S2 S1	Midsystolic click followed by late systolic murmur
	Atrial septal defect	S1 A2 P2 S1	Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect	S1 S2 S1	Holosystolic at left sternal border
Diastolic	Aortic regurgitation*	S1 S2 S1	Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*	S1 S2 OS S1	Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus	S1 S2 S1	Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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R-to-L shunting and are acyanotic.

Ao = aorta; ASD = atrial septal defect; LA = left atrium; LV = left ventricle; PA = pulmonary arteries; PV = pulmonary veins;

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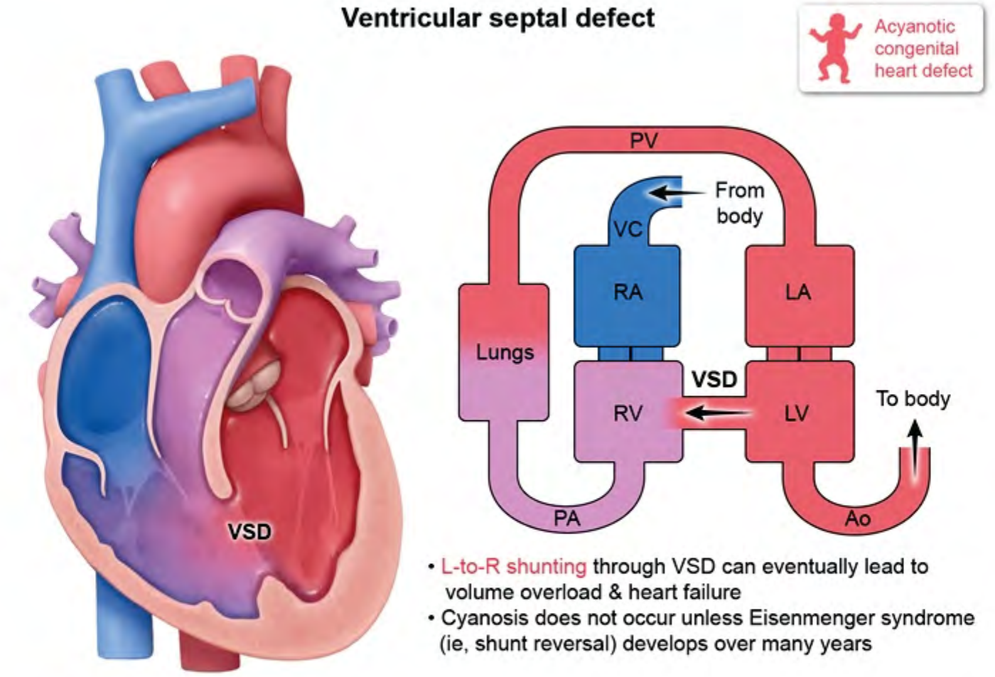

Ao = aorta; ASD = atrial septal defect; LA = left atrium; L-to-R = left-to-right; LV = left ventricle; PA = pulmonary artery; PV = pulmonary vein; RA = right atrium; RV = right ventricle; VC = vena cava; VSD = ventricular septal defect. ©UWorld

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Educational objective:

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A 38-year-old woman is being evaluated for shortness of breath. The patient has had poor exercise tolerance for the last several months that she initially attributed to deconditioning. Over the last several weeks, her symptoms have gotten progressively worse and she currently feels short of breath even during mild exertion. The patient has no family history of heart disease. As part of her evaluation, she undergoes right and left heart catheterization. The following data are obtained:

Left ventricular end-diastolic pressure	10 mm Hg (normal: 3-12 mm Hg)
Left ventricular peak systolic pressure	110 mm Hg (normal: 100-140 mm Hg)
Pulmonary capillary wedge pressure	36 mm Hg (normal: <12 mm Hg)

Which of the following is the most likely cause of this patient's symptoms?

- A. Aortic stenosis
- B. Cardiac tamponade
- C. Dilated cardiomyopathy
- D. Mitral stenosis
- E. Restrictive cardiomyopathy

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Pulmonary capillary wedge pressure	36 mm Hg (normal: <12 mm Hg)

Which of the following is the most likely cause of this patient's symptoms?

- ✗ A. Aortic stenosis (2%)
- B. Cardiac tamponade (2%)
- C. Dilated cardiomyopathy (4%)
- ✓ D. Mitral stenosis (83%)
- E. Restrictive cardiomyopathy (6%)

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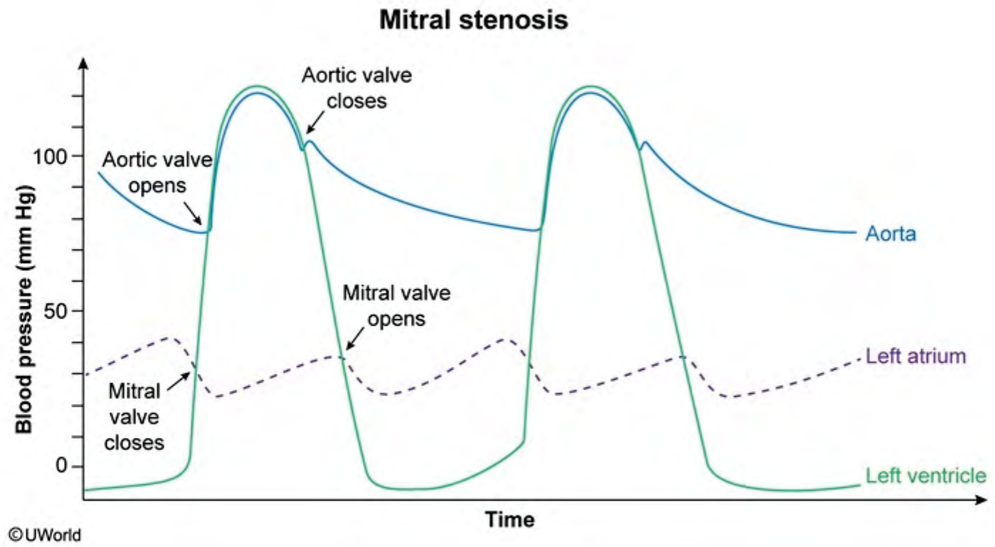

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A balloon-tipped pulmonary artery (Swan-Ganz) catheter is often used to obtain hemodynamic measurements in patients with shock and certain cardiopulmonary conditions. During pulmonary artery (right heart) catheterization, the catheter is advanced to a small branch of the pulmonary artery and the balloon is inflated to obstruct blood flow in that vessel and its distal branches (which eventually form the pulmonary veins). Because blood flow toward the left atrium (LA) is obstructed beyond this point of occlusion, the pressure at the tip of the "wedged" catheter becomes nearly equal to the LA pressure and left ventricular end-diastolic pressure (LVEDP). This is referred to as the pulmonary capillary wedge pressure (PCWP).

During normal diastole, the LA pressure is nearly equal to the LVEDP as the open mitral valve offers minimal resistance to flow between the 2 chambers. In patients with **mitral stenosis**, there is resistance to blood flow from the LA to LV that leads to an **increase** in the **LA pressure**. This increase in LA pressure is transmitted back to the pulmonary veins and is recorded as **elevated PCWP** during pulmonary artery catheterization. If the LA pressure is sufficiently high to overcome the resistance between the LA and LV, LV filling may not be significantly affected, resulting in **normal LVEDP**.

(Choice A) Aortic stenosis causes a pressure gradient between LV and aortic peak systolic pressures. LVEDP and PCWP can be elevated in patients with aortic stenosis; however, both these values would remain in approximately the same range.

(Choice B) Cardiac tamponade leads to equalization of average intracardiac diastolic pressures (left ventricular, right ventricular, and pulmonary artery diastolic pressures). LVEDP and PCWP are both elevated to a similar degree.

(Choices C and E) Both dilated and restrictive cardiomyopathy are associated with pulmonary and systemic venous congestion, resulting in a similar increase in both LVEDP and PCWP. In dilated cardiomyopathy, this occurs due to systolic dysfunction (impaired ventricular contractility), whereas in restrictive cardiomyopathy this occurs due to diastolic dysfunction (impaired ventricular relaxation/filling).

Educational objective:

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Educational objective:
Under normal circumstances, pulmonary capillary wedge pressure (PCWP) closely reflects left atrial (LA) and left ventricular end-diastolic pressure (LVEDP). Mitral stenosis leads to an increase in the LA pressure that is reflected as elevated PCWP during pulmonary artery catheterization. Left ventricular filling may be normal, resulting in an increased pressure gradient between the LA and LV during diastole.

References

- Hemodynamics in the cardiac catheterization laboratory of the 21st century.

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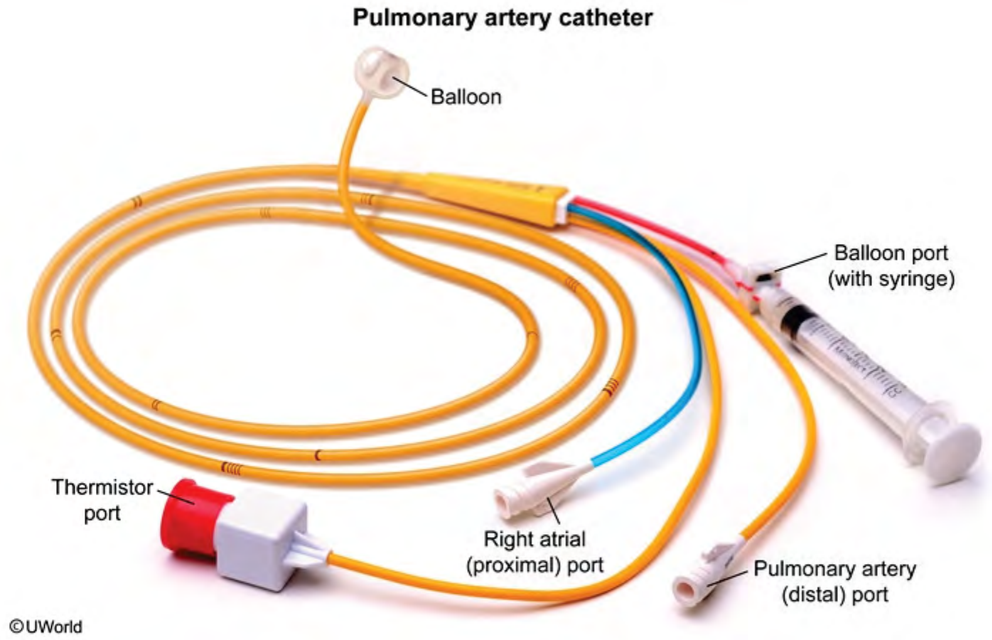

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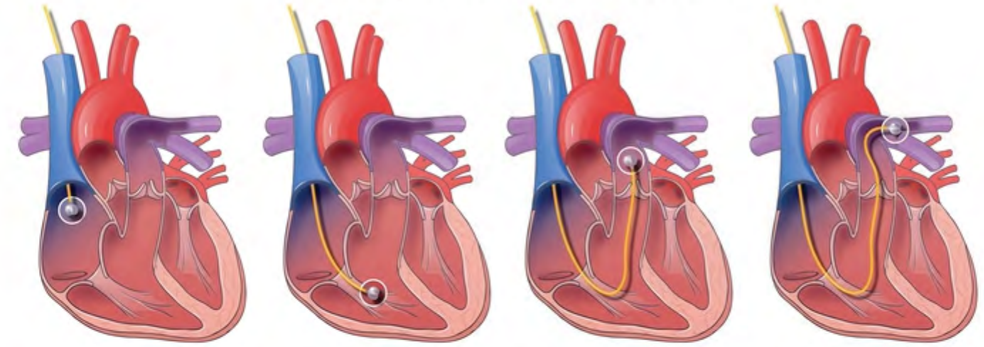
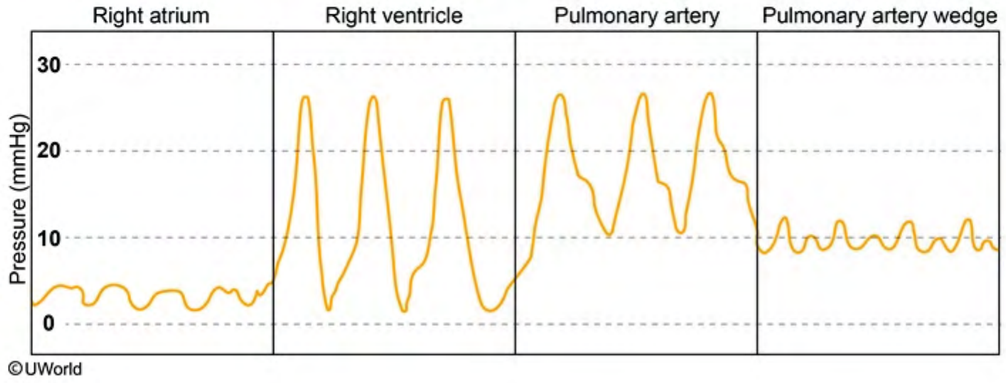
Pulmonary artery catheter waveforms





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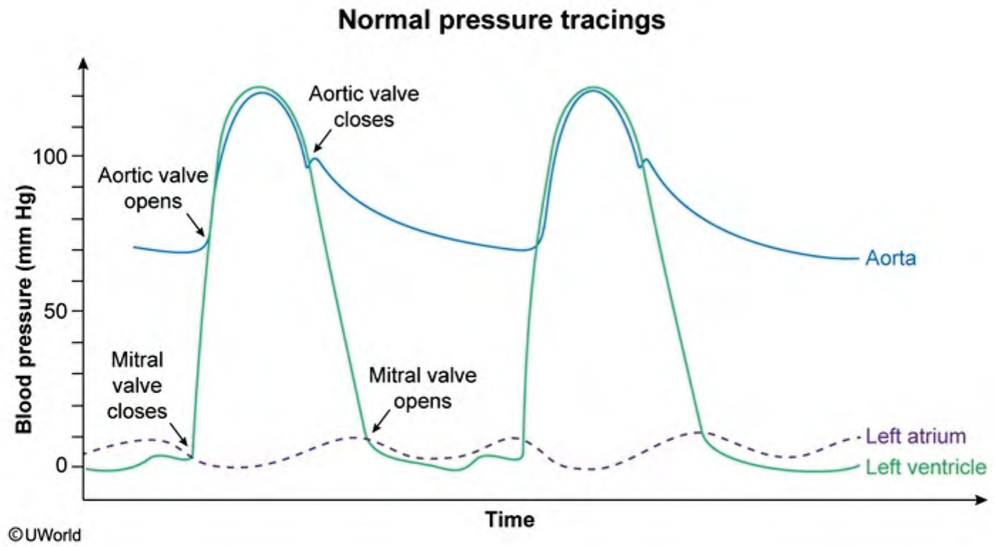

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A 54-year-old man is being evaluated for exertional dyspnea that started 6 months ago. The patient has no associated cough or wheezing. There is no known history of heart disease in his family. ECG, chest x-ray, exercise stress test, and echocardiogram are obtained to evaluate his symptoms. He subsequently undergoes right and left cardiac catheterization, and the results are as follows:

Arterial blood oxygen content	20 mL O_2/100 mL blood
Pulmonary artery diastolic pressure	12 mm Hg
Left ventricular diastolic pressure	10 mm Hg
Mean right atrial pressure	4 mm Hg
Venous blood oxygen content	15 mL O_2/100 mL blood

What additional information is necessary to calculate cardiac output in this patient?

- A. Arteriovenous difference in oxygen content
- B. Oxygen-binding capacity of hemoglobin
- C. Percentage of hemoglobin saturation
- D. Rate of oxygen consumption
- E. Respiratory quotient

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A 54-year-old man is being evaluated for exertional dyspnea that started 6 months ago. The patient has no associated cough or wheezing. There is no known history of heart disease in his family. ECG, chest x-ray, exercise stress test, and echocardiogram are obtained to evaluate his symptoms. He subsequently undergoes right and left cardiac catheterization, and the results are as follows:

Arterial blood oxygen content	20 mL O_2/100 mL blood
Pulmonary artery diastolic pressure	12 mm Hg
Left ventricular diastolic pressure	10 mm Hg
Mean right atrial pressure	4 mm Hg
Venous blood oxygen content	15 mL O_2/100 mL blood

What additional information is necessary to calculate cardiac output in this patient?

- ✗ A. Arteriovenous difference in oxygen content (10%)
- B. Oxygen-binding capacity of hemoglobin (2%)
- C. Percentage of hemoglobin saturation (6%)
- ✓ D. Rate of oxygen consumption (73%)
- E. Respiratory quotient (6%)

Incorrect
Correct answer
D

73%
Answered correctly

05 secs
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Explanation

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Cardiac output, expressed in liters per minute, is commonly calculated using the following formula:

Cardiac output = stroke volume x heart rate

Cardiac output can also be determined with a pulmonary artery (Swan-Ganz) catheter by applying the **Fick principle**, which uses the rate of oxygen consumption and the arteriovenous oxygen difference:

Cardiac output = rate of O_2 consumption / arteriovenous O_2 content difference

Arterial oxygen content is measured directly from arterial blood, and mixed venous oxygen content is measured from pulmonary arterial blood. The rate of oxygen consumption can be determined with an oxygen meter by measuring the rate of disappearance of oxygen in exhaled air.

(Choice A) The arteriovenous difference in oxygen content can be calculated using the information provided in the question stem (eg, arterial blood O_2 content - venous blood O_2 content).

(Choices B and C) The oxygen-binding capacity of hemoglobin and percentage of hemoglobin saturation can be used to calculate blood oxygen content using the following equation:

Blood O_2 content = (O_2-binding capacity x % saturation) + dissolved O_2

Dissolved oxygen is the amount of oxygen unbound to hemoglobin and typically makes up a small fraction of the total blood oxygen content.

(Choice E) The respiratory quotient is the ratio of CO_2 to O_2 across the alveolar membrane, and is used to estimate the metabolic rate. The normal value is typically 0.8.

Educational objective:
The Fick principle can be applied to calculate cardiac output using the rate of oxygen consumption and the arteriovenous oxygen content difference:

Cardiac output = rate of O_2 consumption / arteriovenous O_2 content difference

References

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A 76-year-old woman is brought to the emergency department by her son due to lethargy and confusion over the past 2 days. The patient has persistent atrial fibrillation and congestive heart failure and is on multiple medications. Recently, she has had nausea and decreased appetite. Today, she had 3 episodes of vomiting. The patient also reports vision difficulties. Temperature is 36.7 C (98 F) and blood pressure is 133/80 mm Hg. Laboratory results are as follows:

Sodium	140 mEq/L
Potassium	5.7 mEq/L
Calcium	9.3 mg/dL
Creatinine	1.2 mg/dL
Glucose	85 mg/dL

ECG shows junctional escape rhythm at a rate of 48/min with occasional ventricular premature beats. Increased blood level of which of the following medications is most likely responsible for her symptoms?

- A. Amiodarone
- B. Aspirin
- C. Digoxin
- D. Diltiazem
- E. Furosemide
- F. Metoprolol
- G. Spironolactone
- H. Valsartan

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Sodium	140 mEq/L
Potassium	5.7 mEq/L
Calcium	9.3 mg/dL
Creatinine	1.2 mg/dL
Glucose	85 mg/dL

ECG shows junctional escape rhythm at a rate of 48/min with occasional ventricular premature beats. Increased blood level of which of the following medications is most likely responsible for her symptoms?

- ✗ A. Amiodarone (10%)
- B. Aspirin (0%)
- ✓ C. Digoxin (59%)
- D. Diltiazem (2%)
- E. Furosemide (2%)
- F. Metoprolol (2%)
- G. Spironolactone (21%)
- H. Valsartan (1%)

Incorrect
Correct answer
C

59%
Answered correctly

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Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
Neurologic	• Fatigue • Confusion • Weakness • **Color vision alterations**

This patient with atrial fibrillation and congestive heart failure has signs and symptoms of **digoxin toxicity**. Although calcium channel blockers and beta blockers are the preferred treatments for patients with atrial fibrillation with rapid ventricular response, digoxin is a common second-line treatment that is particularly useful in patients with underlying systolic cardiac dysfunction. Its major effects are:

1. **Increased vagal tone**, causing slowing of conduction through the atrioventricular (AV) node (rate-control effect)
2. Na-K-ATPase inhibition, causing increased intracellular sodium and calcium (increases cardiac contractility)

Digoxin has a narrow therapeutic index, making toxicity relatively common. Symptoms of digoxin intoxication are fairly **nonspecific** and include nausea, abdominal pain, fatigue, dizziness, confusion, blurred vision, and abnormal color perception. Digoxin toxicity can cause a wide range of **cardiac arrhythmias**, including bradycardia and junctional escape beats due to increased AV nodal block. Hypokalemia can precipitate toxicity by increasing digoxin binding to Na-K-ATPase. However, **elevated potassium** is a sign of digoxin toxicity as inhibition of Na-K-ATPase pumps increases extracellular potassium.

(Choice A) Amiodarone can cause pulmonary toxicity, thyroid dysfunction, cardiac arrhythmias, elevated liver

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- **Color vision alterations**

This patient with atrial fibrillation and congestive heart failure has signs and symptoms of **digoxin toxicity**. Although calcium channel blockers and beta blockers are the preferred treatments for patients with atrial fibrillation with rapid ventricular response, digoxin is a common second-line treatment that is particularly useful in patients with underlying systolic cardiac dysfunction. Its major effects are:

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(Choice A) Amiodarone can cause pulmonary toxicity, thyroid dysfunction, cardiac arrhythmias, elevated liver enzymes, and visual disturbances. However, it does not cause hyperkalemia.

(Choice B) Acute aspirin overdose presents with vertigo, tinnitus, vomiting, and diarrhea, whereas severe intoxication can lead to coma, hyperpyrexia, pulmonary edema, and death.

(Choices D and F) Beta blocker and/or calcium channel blocker overdose usually cause profound bradycardia and hypotension (absent in this patient). In addition, nausea and vomiting are more common in digoxin overdose than in beta blocker overdose, which typically causes bronchospasm and hypoglycemia.

(Choice E) Furosemide overdose causes volume depletion, along with electrolyte abnormalities such as hypokalemia and hypomagnesemia.

(Choice G) Excess spironolactone can cause hyperkalemia, gynecomastia, impotence, and decreased libido.

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effect)

2. Na-K-ATPase inhibition, causing increased intracellular sodium and calcium (increases cardiac contractility)

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(Choice G) Excess spironolactone can cause hyperkalemia, gynecomastia, impotence, and decreased libido.

(Choice H) Valsartan is an angiotensin II receptor blocker. Overdose usually causes hypotension, renal failure, and hyperkalemia.

Educational objective:
Digoxin toxicity typically presents with cardiac arrhythmias and nonspecific gastrointestinal (nausea, vomiting), neurological (confusion, weakness), and visual symptoms. Elevated potassium is another sign of digoxin toxicity and is caused by inhibition of Na-K-ATPase pumps.

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- Anorexia

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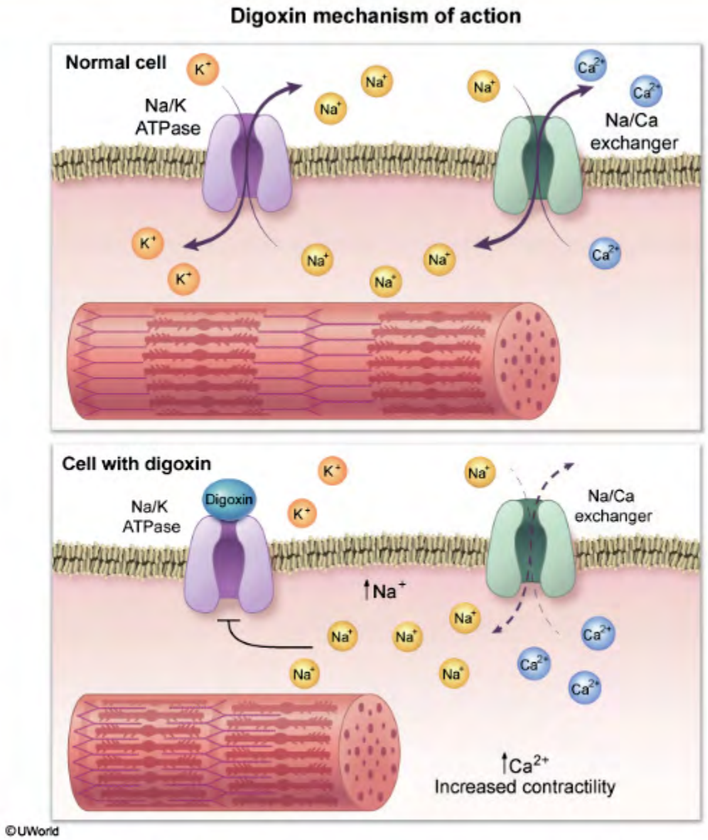

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 53-year-old man comes to the emergency department due to fever and progressive weakness over the last 2 weeks. Yesterday, he also developed shortness of breath. The patient emigrated from Eastern Europe 2 years ago and says he was diagnosed with "heart disease" in the distant past, but he does not recall any details. He does not use tobacco, alcohol, or illicit drugs. Despite receiving appropriate medical care, the patient expires during hospitalization. On autopsy, gross examination of his heart shows large, friable masses on the mitral valve with extensive destruction of cuspal tissue.

Item 1 of 2

Which of the following is the most likely predisposing factor for this patient's presenting condition?

- A. Myocardial hypertrophy
- B. Myocardial thinning and fibrosis
- C. Pericardial effusion
- D. Rupture of chordae tendineae
- E. Valvular inflammation and scarring

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Item 2 of 2

The blood cultures obtained from this patient on admission grow *Streptococcus* species. Which of the following processes was the most likely initiating step in the pathogenesis of this patient's condition?

- A. Endocardial fibrosis
- B. Fibrin deposition
- C. Liquefactive necrosis
- D. Myxomatous degeneration
- E. White blood cell infiltration

Submit

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Item 1 of 2

Which of the following is the most likely predisposing factor for this patient's presenting condition?

- ✗ A. Myocardial hypertrophy (2%)
- B. Myocardial thinning and fibrosis (1%)
- C. Pericardial effusion (1%)
- D. Rupture of chordae tendineae (12%)
- ✓ E. Valvular inflammation and scarring (82%)

Incorrect
Correct answer
E

82%
Answered correctly

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Explanation

This patient's presentation - fever, progressive weakness, and dyspnea (due to valvular regurgitation) - along with

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Explanation

This patient's presentation - fever, progressive weakness, and dyspnea (due to valvular regurgitation) - along with the presence of vegetations on the mitral valve is consistent with **infective endocarditis** (IE). Predisposing factors for IE include **valvular abnormalities** (eg, rheumatic heart disease, mitral valve prolapse, prosthetic valves, congenital heart disease) and conditions that promote **bacteremia/fungemia** (eg, intravenous drug use, dental procedures).

This patient likely had a remote history of **rheumatic heart disease** (immigrant from developing region, history of heart disease) with underlying degeneration of the mitral valve due to chronic **valvular inflammation and scarring**.

(Choice A) Myocardial hypertrophy is seen in patients with uncontrolled hypertension, aortic stenosis, and hypertrophic cardiomyopathy and, in general, does not predispose to IE.

(Choice B) Myocardial fibrosis is associated with infiltrative cardiomyopathy (sarcoidosis, amyloidosis), Chagas disease, myocarditis, and prior myocardial infarction. It does not lead to increased risk of IE.

(Choice C) Pericardial effusion occurs as a result of inflammation of the pericardium (acute or chronic pericarditis) and has no association with the risk of IE.

(Choice D) Rupture of chordae tendineae is a complication of endocarditis or myocardial infarction; it is not a risk factor for development of IE.

Educational objective:
Chronic valvular inflammation and scarring associated with rheumatic heart disease predispose to an increased risk of infective endocarditis, which is characterized by valvular vegetations with destruction of the underlying cardiac tissue.

References

- Underlying cardiac lesions in adults with infective endocarditis. The changing spectrum.

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Item 2 of 2

The blood cultures obtained from this patient on admission grow *Streptococcus* species. Which of the following processes was the most likely initiating step in the pathogenesis of this patient's condition?

- A. Endocardial fibrosis (8%)
- ✓ B. Fibrin deposition (52%)
- C. Liquefactive necrosis (1%)
- D. Myxomatous degeneration (13%)
- E. White blood cell infiltration (24%)

Correct | 52% Answered correctly | 05 secs Time Spent | 2023 Version

Explanation

The initial process involved in the pathogenesis of infective endocarditis (IE) is a **disruption of normal endocardial surface**. This occurs most commonly at the areas of maximal turbulence to blood flow in preexisting valvular lesions, typically the atrial surface of incompetent atrioventricular valves or the ventricular surface of incompetent semilunar valves. This is followed by focal adherence of fibrin and platelets, forming a **sterile fibrin-platelet nidus**.

During bacteremia from any cause, microorganisms colonize the sterile nidus on the endothelial surface with subsequent microbial growth leading to further activation of the coagulation system. Streptococci infect the cardiac valves with preexisting endothelial lesions. In contrast, *Staphylococcus aureus* can adhere to damaged or normal endothelial cells. Macroscopic vegetations consist of fibrin and platelets on the surface, with red blood cell debris,

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(Choice A) Endomyocardial fibrosis is a restrictive cardiomyopathy characterized by thickening and fibrosis of the apical endocardial surface. It occurs most commonly in tropical regions (eg, Uganda).

(Choices C and E) Neutrophil-rich inflammatory infiltrates with subsequent abscess formation and/or liquefactive necrosis of underlying cardiac tissue are common in advanced lesions.

(Choice D) Myxomatous degeneration of the mitral valve is characterized by thickened and redundant mitral leaflets with elongated chordae leading to prolapse of one or both mitral leaflets (mitral valve prolapse). Myxomatous degeneration can predispose patients to the development of endocarditis; however, endocarditis vegetations themselves are not formed through myxomatous degeneration.

Educational objective:
Vegetations are caused by bacterial colonization and growth on a sterile fibrin-platelet nidus that forms on the damaged/disrupted endothelial surface of the valvular apparatus.

References

- Pathogenesis of streptococcal and staphylococcal endocarditis.
- Mechanisms of infective endocarditis: pathogen-host interaction and risk states.

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A 56-year-old man comes to the emergency department due to palpitations and lightheadedness over the last 2 hours. He has a history of hypertension but no other cardiac conditions and never before had similar symptoms. The patient drinks 1 to 2 alcoholic beverages daily but does not smoke cigarettes or use illicit drugs. Blood pressure is 132/76 mm Hg and pulse is 116/min and irregular. Cardiopulmonary examination is normal, except for tachycardia. Resting ECG shows atrial fibrillation. The patient undergoes transesophageal echocardiography, which reveals normal cardiac anatomy and no intracardiac thrombi. Electrical cardioversion is performed. After the intervention, sinus rhythm is restored, and amiodarone therapy is begun to maintain normal sinus rhythm. Which of the following cardiac electrophysiologic changes are most likely to occur in this patient due to the medication?

	Sinus rate	PR interval	QRS duration	QT interval
○ A.	Decreased	Prolonged	No change	No change
○ B.	Decreased	Prolonged	Prolonged	Prolonged
○ C.	No change	No change	No change	No change
○ D.	No change	No change	Prolonged	No change
○ E.	No change	No change	Prolonged	Prolonged

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	Sinus rate	PR interval	QRS duration	QT interval	
✗ A.	Decreased	Prolonged	No change	No change	(8%)
✓ B.	Decreased	Prolonged	Prolonged	Prolonged	(35%)
C.	No change	No change	No change	No change	(4%)
D.	No change	No change	Prolonged	No change	(9%)
E.	No change	No change	Prolonged	Prolonged	(40%)

Incorrect
Correct answer
B

35%
Answered correctly

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Explanation

ECG effects of antiarrhythmic drugs

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ECG effects of antiarrhythmic drugs			
	PR interval (prolonged via Ca^{2+} channel inhibition or sympathetic blockade)	**QRS duration** (prolonged via Na^{+} channel inhibition)	**QT interval** (prolonged via K^{+} channel inhibition)
Class IA (eg, procainamide)	–	↑	↑
Class IB (eg, lidocaine)	–	–	–
Class IC (eg, flecainide)	–	↑	
Class II (eg, metoprolol)	↑	–	–
Amiodarone	↑	↑	↑
Sotalol	↑	–	↑
Other class III (eg, dofetilide)	–	–	↑
Class IV (eg, verapamil)	↑	–	–

Amiodarone is a commonly used antiarrhythmic drug with wide-ranging antiarrhythmic effects and corresponding ECG manifestations. It primarily functions as a **class III antiarrhythmic** by **inhibiting** the rapid component of the

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(eg, verapamil)			

Amiodarone is a commonly used antiarrhythmic drug with wide-ranging antiarrhythmic effects and corresponding ECG manifestations. It primarily functions as a **class III antiarrhythmic** by **inhibiting** the rapid component of the delayed rectifier **potassium current**, which is responsible for ventricular repolarization. This prolongs action potential duration and the effective refractory period, suppressing electrical foci that stimulate atrial and ventricular arrhythmias. It also **prolongs the QT interval** on ECG, which increases the risk of torsade de pointes; however, compared to other class III antiarrhythmics (eg, dofetilide, ibutilide), the risk of torsade de pointes with amiodarone is low.

Amiodarone also has the following effects:

- Class I effect: Amiodarone somewhat **inhibits** the **fast sodium channels** responsible for cardiomyocyte depolarization (phase 0). This slows ventricular depolarization to suppress arrhythmogenic foci, and it **prolongs QRS complex duration** on ECG.
- Class II and IV effects: Amiodarone has beta-blocking properties that inhibit sympathetic stimulation of the conduction system. The drug also **inhibits** the **slow L-type calcium channels** responsible for depolarization of the sinus node and atrioventricular node. Both effects prolong the refractory period of the conduction system to suppress arrhythmogenic foci and in doing so contribute to a **decreased sinus rate** (ie, slowed heart rate) and a **prolonged PR interval** on ECG.

(Choice A) Both class II (ie, beta blockers) and class IV (ie, nondihydropyridine calcium channel blockers) antiarrhythmics slow the sinus rate and prolong the PR interval, but have no effect on QRS duration or the QT interval.

(Choices C, D, and E) Class I antiarrhythmics inhibit fast sodium channels to slow depolarization and prolong QRS duration. However, the effects of class IB antiarrhythmics (eg, lidocaine) occur only at rapid heart rates, and no change in QRS duration is seen on resting ECG. Class IA antiarrhythmics (eg, procainamide) also inhibit the delayed rectifier potassium current to prolong the QT interval.

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Educational objective:
Amiodarone primarily functions as a class III antiarrhythmic, inhibiting the delayed rectifier potassium current to slow ventricular repolarization and prolong the QT interval. It also inhibits fast sodium channels (class I effect) to slow ventricular depolarization and prolong QRS complex duration. Beta blockade (class II effect) and inhibition of slow L-type calcium channels (class IV effect) slow conduction in the sinus node and atrioventricular node causing decreased sinus rate and a prolonged PR interval.

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Other class III	–	–	↑

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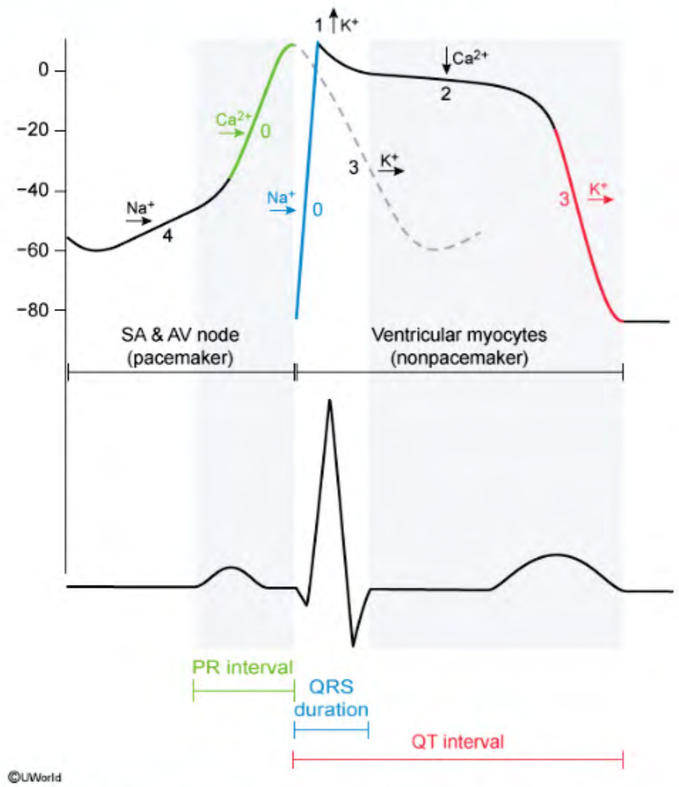
ECG manifestations of cardiac action potentials



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(Choices C, D, and E) Class I antiarrhythmics inhibit fast sodium channels to slow depolarization and prolong

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A 65-year-old man comes to the physician after missing a follow-up appointment for congestive heart failure. He missed his appointment due to an upset stomach that will not go away. A detailed history reveals disturbed color perception, anorexia, nausea, vomiting, and diarrhea that have worsened over the past 2 weeks. His congestive heart failure has thus far been controlled effectively with multiple medications. If the cause of this patient's current symptoms is not corrected, which of the following complications is most likely to develop?

- A. Angioedema
- B. Arrhythmia
- C. Bronchoconstriction
- D. Hypotension
- E. Pulmonary edema

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- ✗ A. Angioedema (2%)
- ✓ B. Arrhythmia (72%)
- C. Bronchoconstriction (1%)
- D. Hypotension (15%)
- E. Pulmonary edema (7%)

Incorrect
Correct answer
B

72%
Answered correctly

03 secs
Time Spent

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Explanation

Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
	• Fatigue

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Explanation

Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
Neurologic	• Fatigue • Confusion • Weakness • **Color vision alterations**

This patient most likely has chronic **digoxin toxicity**, which can cause nonspecific gastrointestinal and neurologic symptoms. Although rare, **changes in color vision** are particularly associated with digoxin overdose. The most serious complication of digoxin toxicity is the development of potentially fatal **cardiac arrhythmias** of virtually any type.

Digoxin has a narrow therapeutic index, making toxicity relatively common. Precipitating causes include hypokalemia (increased digoxin binding), hypovolemia, and renal failure. Patients with severe toxicity (eg, life-threatening arrhythmias, end-organ dysfunction) are treated with anti-digoxin antibody fragments.

(Choice A) Angioedema is a severe side effect that manifests as rapidly developing edema that often affects the lips and larynx. Of the drugs used to treat heart failure, angiotensin-converting enzyme inhibitors are most commonly associated with angioedema.

(Choice C) Bronchoconstriction can be caused by nonspecific beta-adrenergic blockers (eg, propranolol) in patients with chronic obstructive pulmonary disease or asthma. Selective beta blockers (eg, metoprolol) are

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Digoxin has a narrow therapeutic index, making toxicity relatively common. Precipitating causes include hypokalemia (increased digoxin binding), hypovolemia, and renal failure. Patients with severe toxicity (eg, life-threatening arrhythmias, end-organ dysfunction) are treated with anti-digoxin antibody fragments.

(Choice A) Angioedema is a severe side effect that manifests as rapidly developing edema that often affects the lips and larynx. Of the drugs used to treat heart failure, angiotensin-converting enzyme inhibitors are most commonly associated with angioedema.

(Choice C) Bronchoconstriction can be caused by nonspecific beta-adrenergic blockers (eg, propranolol) in patients with chronic obstructive pulmonary disease or asthma. Selective beta blockers (eg, metoprolol) are commonly used to treat patients with congestive heart failure and are less likely to cause bronchoconstriction.

(Choice D) Hypotension can occur with digoxin toxicity due to bradyarrhythmias and severe vomiting and diarrhea that lead to hypovolemia. However, digoxin is also an inotropic agent that causes increased cardiac output. Therefore, hypotension is a variable manifestation of digoxin toxicity.

(Choice E) Pulmonary edema may occur with uncontrolled heart failure due to poor forward flow that causes blood to pool in the lungs. It can be caused by both systolic and diastolic left ventricular dysfunction due to decreased forward ejection of blood and impaired ventricular filling, respectively.

Educational objective:
Digoxin toxicity presents with nonspecific gastrointestinal (eg, anorexia, nausea, vomiting) and neurologic (eg, fatigue, confusion, weakness) symptoms. Changes in color vision are a more specific, but rarer, finding. Life-threatening ventricular arrhythmias are the most serious complication.

References

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A 55-year-old man comes to the office for follow-up. The patient was diagnosed with hypertension 3 weeks ago, and an ECG performed at that time showed features of left ventricular hypertrophy. He was subsequently referred for echocardiography. The patient feels well and has had no shortness of breath, fatigue, or leg swelling. He has no other medical conditions, takes no medications, and does not use tobacco, alcohol, or illicit drugs. Blood pressure is 155/90 mm Hg and pulse is 80/min. BMI is 35 kg/m^2. Physical examination shows normal jugular venous pressure. There are no lung crackles, heart gallops, or lower extremity edema. Echocardiography reveals a left ventricular ejection fraction of 30%. Which of the following patterns of chemical mediators is most likely present in this patient?

	Angiotensin II	Norepinephrine	Atrial natriuretic peptide
A.	Decreased	Decreased	Decreased
B.	Decreased	Decreased	Increased
C.	Decreased	Increased	Increased
D.	Increased	Decreased	Decreased
E.	Increased	Decreased	Increased
F.	Increased	Increased	Increased

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		Angiotensin II	Norepinephrine	Atrial natriuretic peptide	
✗	A.	Decreased	Decreased	Decreased	(2%)
	B.	Decreased	Decreased	Increased	(11%)
	C.	Decreased	Increased	Increased	(6%)
	D.	Increased	Decreased	Decreased	(14%)
	E.	Increased	Decreased	Increased	(13%)
✓	F.	Increased	Increased	Increased	(51%)

Incorrect
Correct answer
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51%
Answered correctly

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Explanation

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D. Increased Decreased Decreased (14%)

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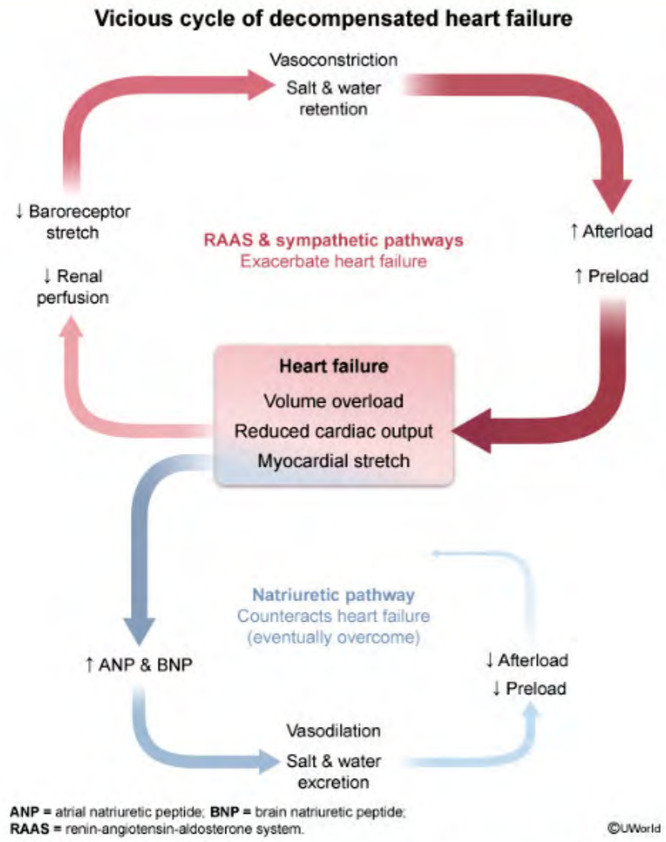

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ANP = atrial natriuretic peptide; **BNP** = brain natriuretic peptide;
RAAS = renin-angiotensin-aldosterone system.

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This patient's echocardiography reveals a reduced left ventricular (LV) ejection fraction (<50%) in the absence of heart failure symptoms, a condition known as **asymptomatic left ventricular systolic dysfunction**. An early asymptomatic period is common in the progression of heart failure; it occurs in part due to compensatory eccentric hypertrophy in response to LV volume overload. In addition, **neurohormonal mechanisms** help maintain cardiac output and organ perfusion in the setting of declining cardiac function.

Early in the course of heart failure, small reductions in cardiac output lead to decreased stretch of aortic and carotid baroreceptors with consequent **sympathetic nervous system activation** (increased norepinephrine) to stimulate vasoconstriction, increased heart rate, and increased cardiac contractility. In addition, decreased renal perfusion stimulates the juxtaglomerular apparatus to increase renin secretion and activate the **renin-angiotensin-aldosterone system** (RAAS), leading to vasoconstriction via increased angiotensin II levels and salt and water retention via increased aldosterone levels. The volume retention (increased preload) and vasoconstriction (increased afterload) help maintain cardiac output and organ perfusion in the short term; however, the **long-term effects** are **deleterious** because increased hemodynamic stress and neurohormonal-induced cardiac remodeling eventually lead to **decompensated heart failure**.

Atrial and ventricular myocardial stretch triggers **increased atrial and brain natriuretic peptide** secretion through separate neurohormonal pathways. These peptides stimulate vasodilation and salt and water excretion, counteracting the effects of the sympathetic nervous system and RAAS; however, the natriuretic pathways are eventually overwhelmed by the downward compensatory spiral.

Educational objective:
Asymptomatic left ventricular systolic dysfunction is a common stage in the progression of heart failure. Neurohormonal mechanisms, including the sympathetic nervous system and renin-angiotensin-aldosterone system, help maintain the asymptomatic period by increasing volume retention and peripheral resistance to maintain organ perfusion. Although these mechanisms are beneficial in the short term, they are ultimately deleterious, increasing hemodynamic stress and cardiac remodeling that eventually lead to decompensated heart

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heart failure symptoms, a condition known as **asymptomatic left ventricular systolic dysfunction**. An early asymptomatic period is common in the progression of heart failure; it occurs in part due to compensatory eccentric hypertrophy in response to LV volume overload. In addition, **neurohormonal mechanisms** help maintain cardiac output and organ perfusion in the setting of declining cardiac function.

Early in the course of heart failure, small reductions in cardiac output lead to decreased stretch of aortic and carotid baroreceptors with consequent **sympathetic nervous system activation** (increased norepinephrine) to stimulate vasoconstriction, increased heart rate, and increased cardiac contractility. In addition, decreased renal perfusion stimulates the juxtaglomerular apparatus to increase renin secretion and activate the **renin-angiotensin-aldosterone system** (RAAS), leading to vasoconstriction via increased angiotensin II levels and salt and water retention via increased aldosterone levels. The volume retention (increased preload) and vasoconstriction (increased afterload) help maintain cardiac output and organ perfusion in the short term; however, the **long-term effects** are **deleterious** because increased hemodynamic stress and neurohormonal-induced cardiac remodeling eventually lead to **decompensated heart failure**.

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Asymptomatic left ventricular systolic dysfunction is a common stage in the progression of heart failure. Neurohormonal mechanisms, including the sympathetic nervous system and renin-angiotensin-aldosterone system, help maintain the asymptomatic period by increasing volume retention and peripheral resistance to maintain organ perfusion. Although these mechanisms are beneficial in the short term, they are ultimately deleterious, increasing hemodynamic stress and cardiac remodeling that eventually lead to decompensated heart failure.

Pathophysiology	Cardiovascular System	Acute heart failure
Subject	System	Topic

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ANP = atrial natriuretic peptide; **BNP** = brain natriuretic peptide; **RAAS** = renin-angiotensin-aldosterone system.
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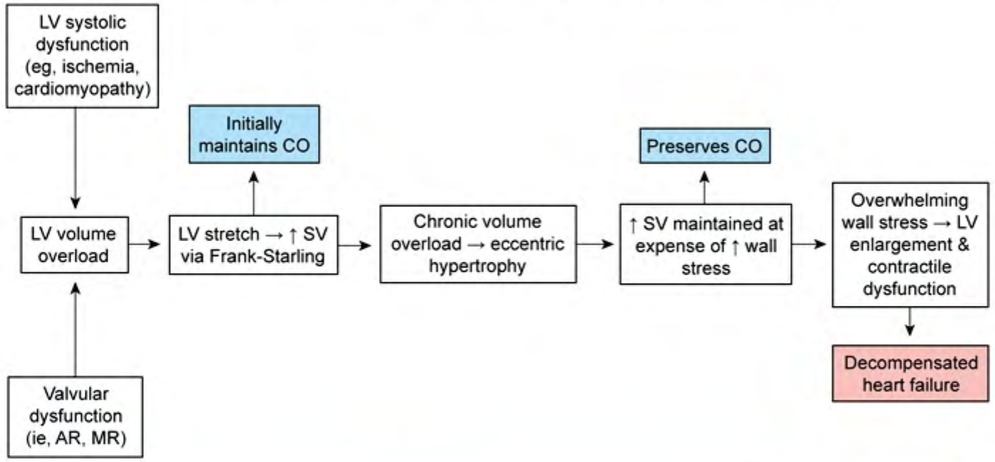
Eccentric left ventricular enlargement & heart failure



AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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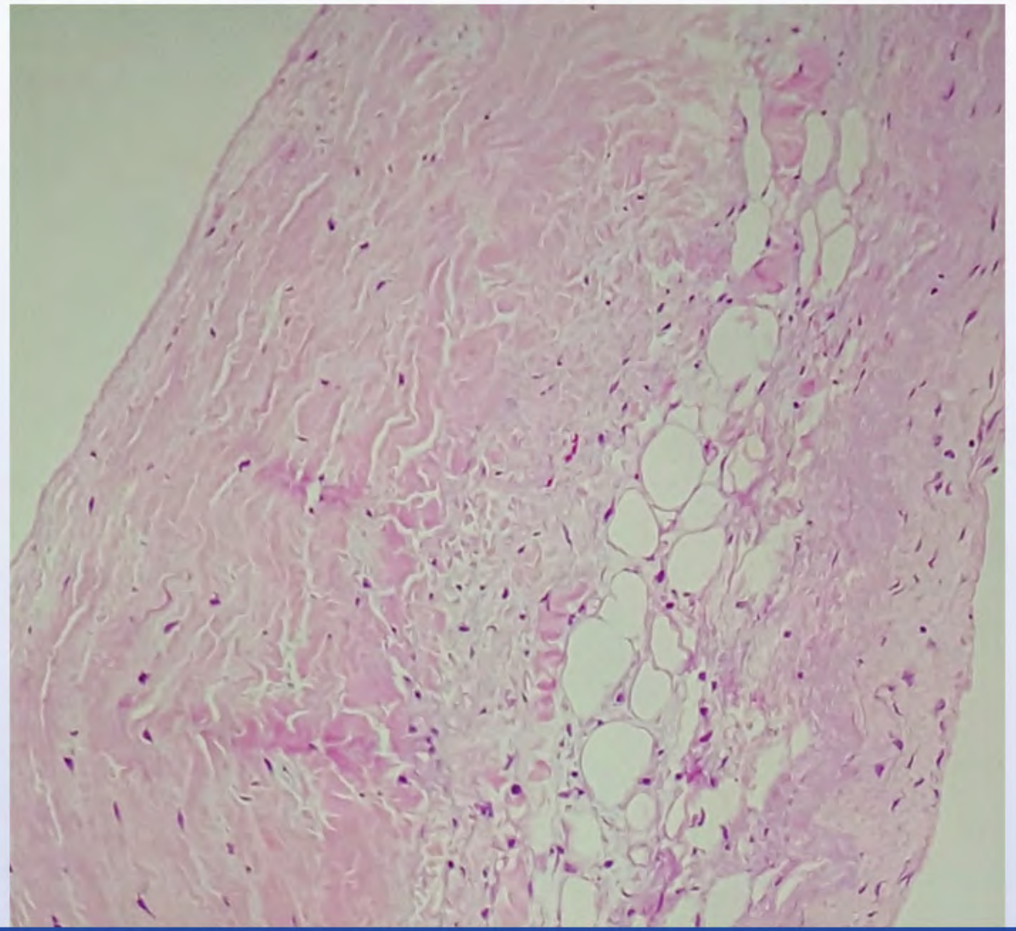
A 50-year-old man is brought to the emergency department due to chest pain. He passed out briefly after the pain started. Medical history includes hypertension, hyperlipidemia, and early-stage chronic kidney disease. The patient reports that his father had a heart attack at a young age. Despite treatment, the patient dies during hospitalization. An autopsy is performed. Light microscopy of the aortic wall is shown on the slide below.



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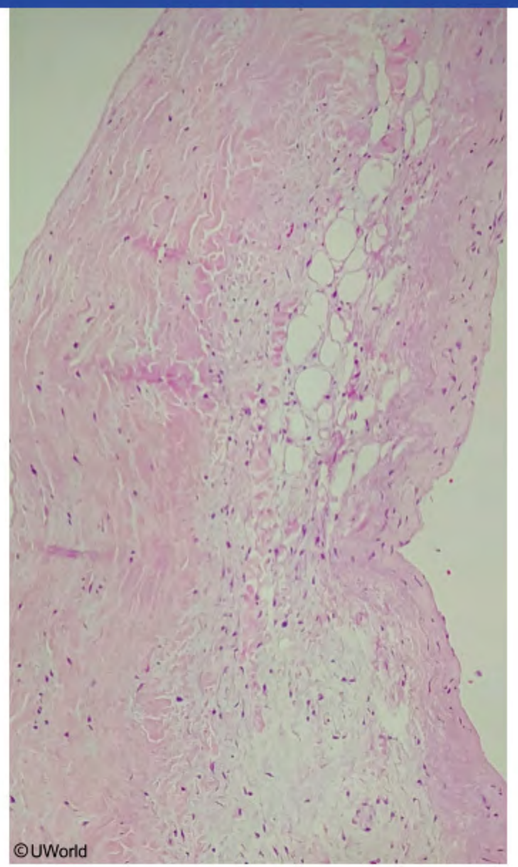

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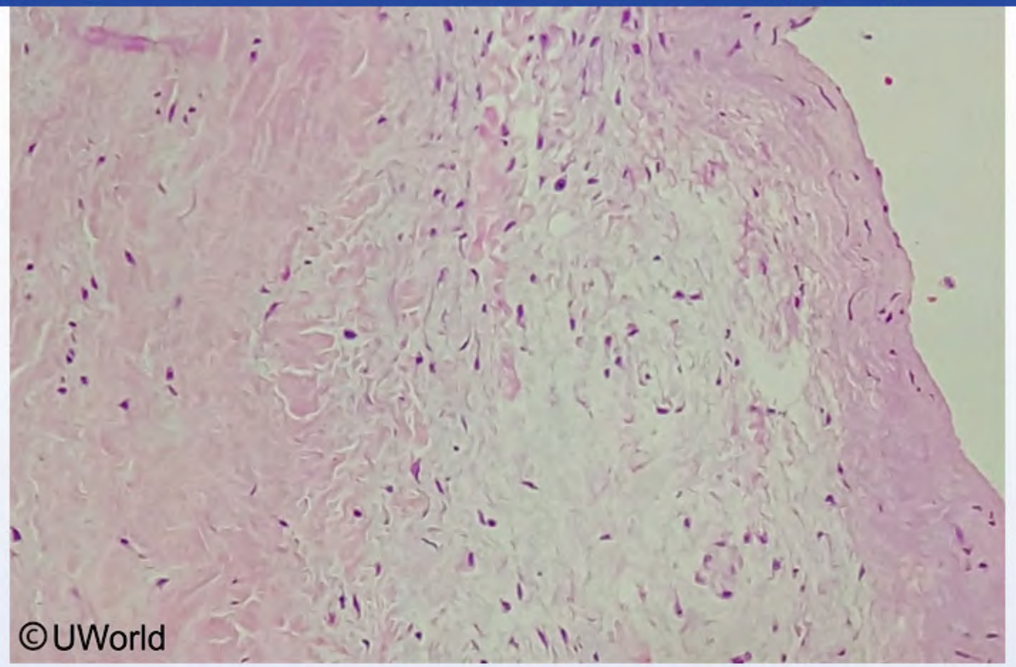

Which of the following underlying mechanisms is most likely responsible for this patient's disease process?

- A. Atherosclerosis
- B. Calciphylaxis
- C. Cystic degeneration of the media
- D. Granulomatous inflammation
- E. Monckeberg sclerosis

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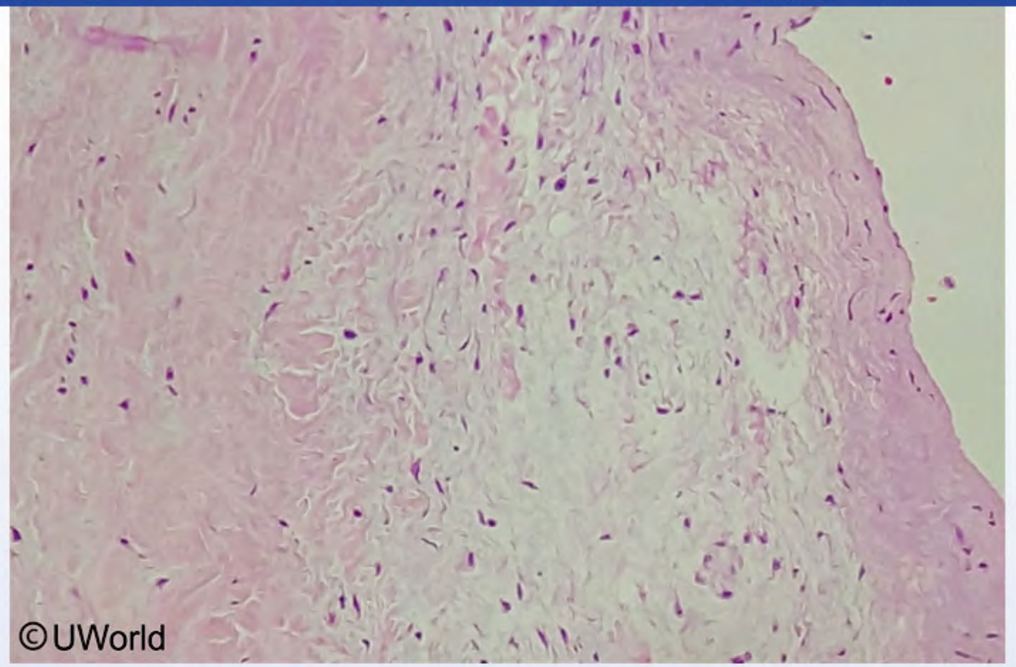

Which of the following underlying mechanisms is most likely responsible for this patient's disease process?

- A. Atherosclerosis (30%)
- B. Calciphylaxis (1%)
- ✓ C. Cystic degeneration of the media (63%)
- D. Granulomatous inflammation (1%)
- E. Monckeberg sclerosis (4%)

Correct

63% Answered correctly

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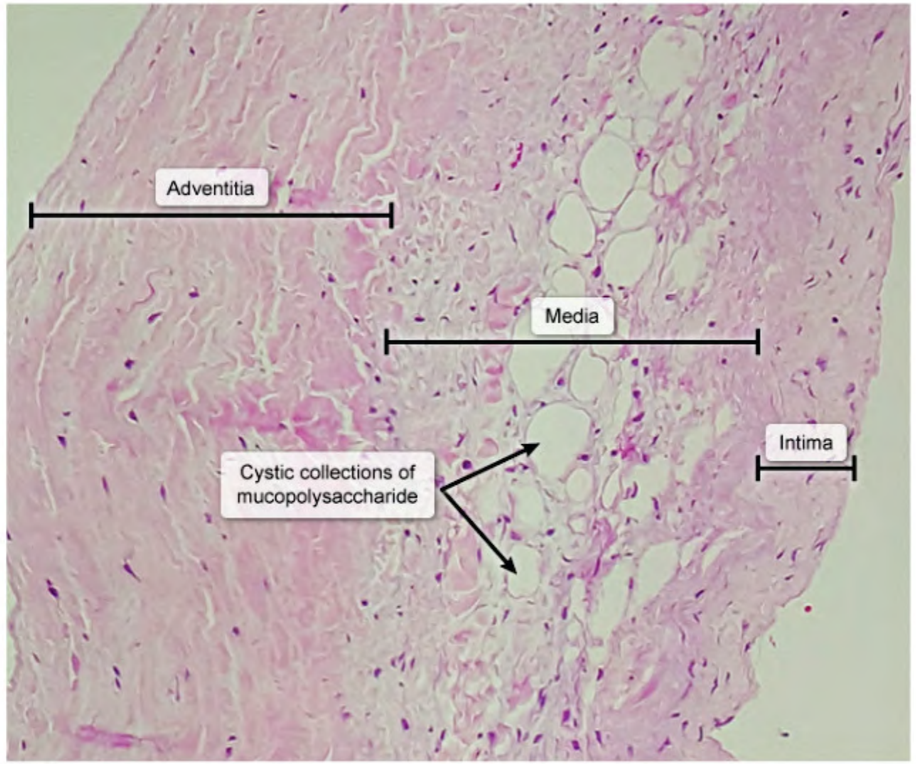
Marfan syndrome (cystic medial degeneration)



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The above autopsy image shows **cystic medial degeneration** (necrosis), the classic histologic finding in **aortic**

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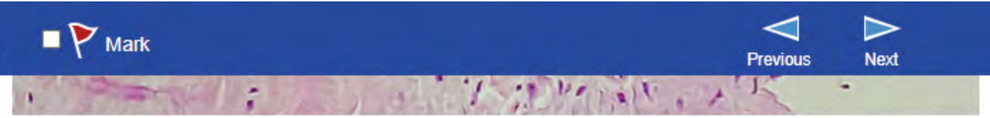

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The above autopsy image shows **cystic medial degeneration** (necrosis), the classic histologic finding in **aortic dissection**. The tunica media is the largest and strongest of the 3 portions of the aortic wall. With aging, collagen, elastin, and smooth muscle in the aortic media are broken down and replaced by a mucoid extracellular matrix. There is elastic tissue fragmentation in a "basket weave" pattern with development of many cystic collections of mucopolysaccharide. These changes result in progressive **loss of integrity**, reducing the vessel's ability to handle wall stress from conditions such as systemic hypertension.

Once cystic medial degeneration develops, a **small intimal tear** can readily **extend** into and throughout the media, creating a lengthy **dissection plane** that propagates proximally or distally. As blood enters the dissection plane a false aortic lumen is created and aortic branches (eg, left subclavian artery, renal arteries) may become obstructed. The enlarging hematoma in the false lumen can eventually penetrate the adventitia, resulting in full-thickness rupture.

(Choice A) Atherosclerosis primarily affects the intima and is an important contributor to acute coronary syndrome. Histologically it appears as an atheroma with an endothelial covering, fibrous cap, and necrotic core. Atherosclerosis can contribute to cystic medial degeneration but is not the primary pathophysiologic mechanism.

(Choice B) Calcific uremic arteriolopathy (calciphylaxis) can occur with advanced chronic kidney disease and leads to severe skin ischemia and necrosis. Histologically, it is characterized by superficial arteriolar calcification, subintimal fibrosis, and thrombosis. It does not typically affect large vessels such as the aorta.

(Choice D) Granulomatous inflammation of large vessels can occur in Takayasu arteritis and tuberculous aortitis. Histopathology demonstrates granuloma formation in the media with multinucleated giant cells.

(Choice E) Monckeberg sclerosis is a rare disease that results in diffuse medial calcification of small and medium-sized arteries. Calcification typically appears as blue-to-violet irregular deposits on hematoxylin–eosin stains, as seen in this calcified atherosclerotic plaque.

Educational objective:

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wall stress from conditions such as systemic hypertension.

Once cystic medial degeneration develops, a **small intimal tear** can readily **extend** into and throughout the media, creating a lengthy **dissection plane** that propagates proximally or distally. As blood enters the dissection plane a false aortic lumen is created and aortic branches (eg, left subclavian artery, renal arteries) may become obstructed. The enlarging hematoma in the false lumen can eventually penetrate the adventitia, resulting in full-thickness rupture.

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Educational objective:
Cystic medial degeneration (necrosis) is the classic histologic finding in aortic dissection, as it weakens the aortic wall and allows a small intimal tear to readily propagate. Collagen, elastin, and smooth muscle are replaced by a basophilic mucoid extracellular matrix with elastic tissue fragmentation and cystic collections of mucopolysaccharide.

References

- Impact of connective tissue disease on the surgical outcomes of aortic dissection in patients with cystic medial

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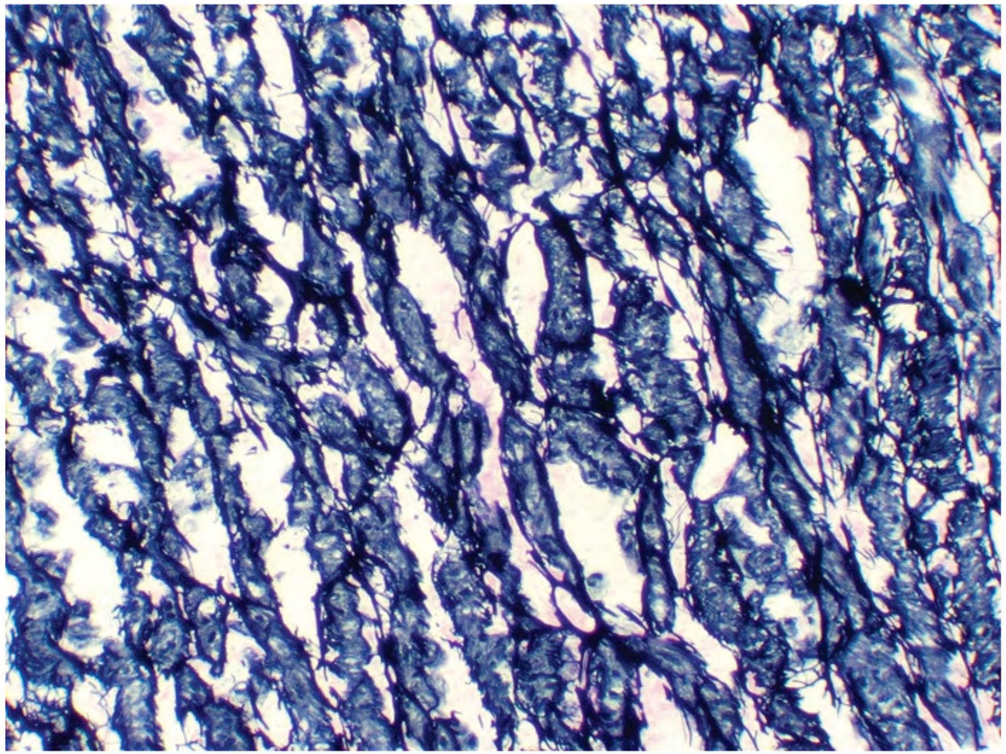

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(Choice E) Monckeberg sclerosis is a rare disease that results in diffuse medial calcification of small and medium-

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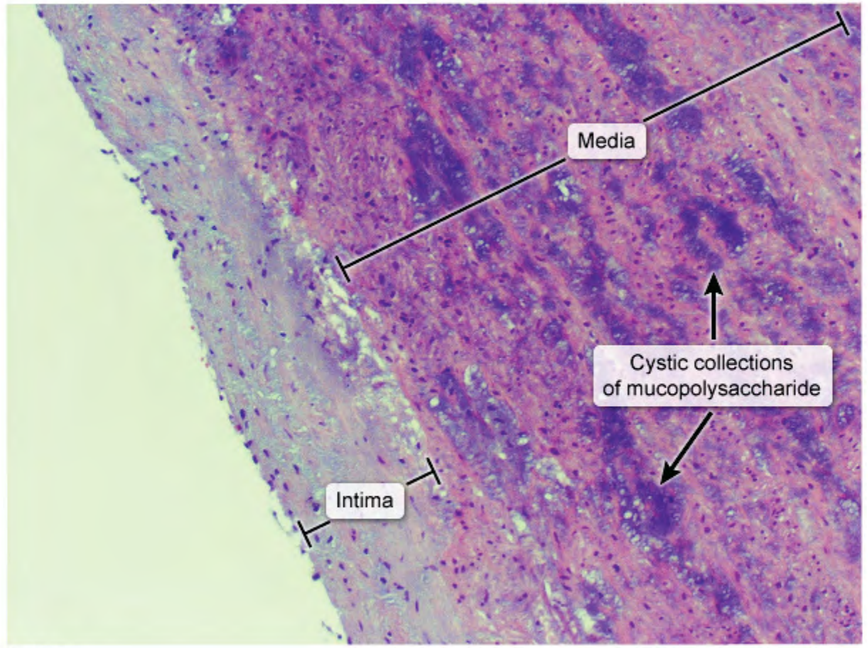
Cystic medial degeneration of aorta | Aortic dissection and cystic medial degeneration.

Cystic medial degeneration of aorta



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(Choice E) Monckeberg sclerosis is a rare disease that results in diffuse medial calcification of small and medium-

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Atherosclerotic plaque

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Educational objective:

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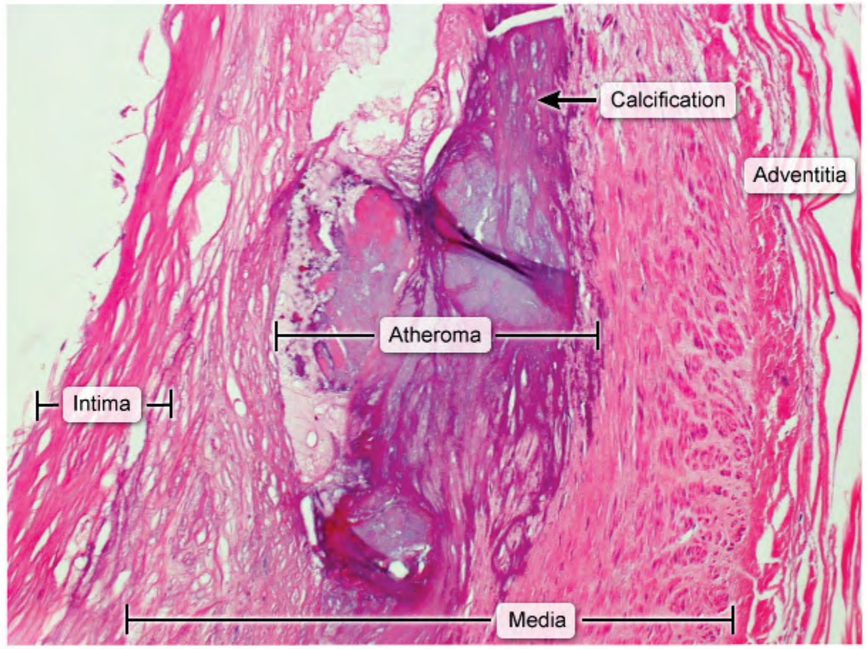
Calcific atherosclerosis



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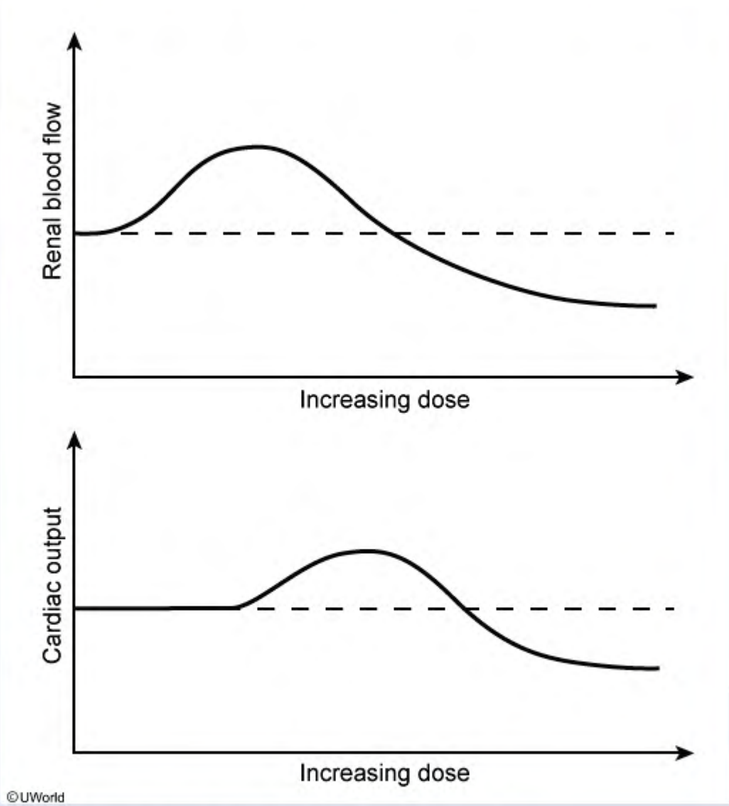
A medical student is conducting a pharmacology experiment to determine the effects of a certain drug. She infuses this drug intravenously over different dose ranges and measures several important hemodynamic parameters. Graphs plotting the observed changes in renal blood flow and cardiac output are shown below.



Which of the following drugs was most likely used in this experiment?

A. Dopamine

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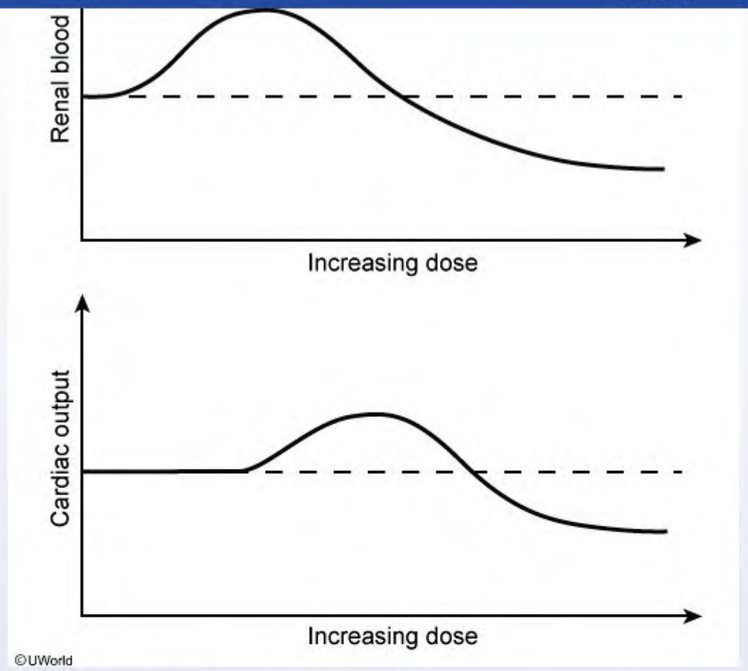

Which of the following drugs was most likely used in this experiment?

- A. Dopamine
- B. Edrophonium
- C. Epinephrine
- D. Esmolol
- E. Phenylephrine

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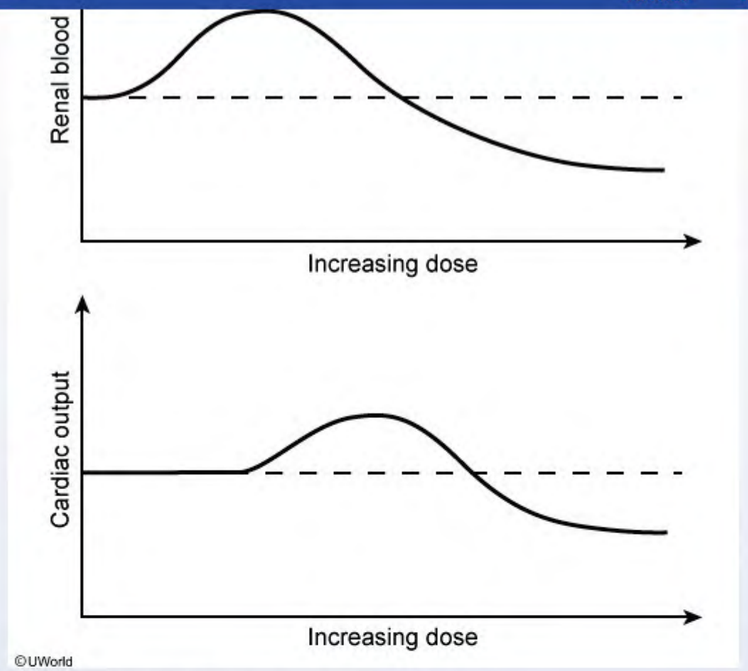

Which of the following drugs was most likely used in this experiment?

- ✓ A. Dopamine (67%)
- ✗ B. Edrophonium (1%)
- C. Epinephrine (19%)
- D. Esmolol (5%)
- E. Phenylephrine (5%)

Incorrect
Correct answer
A

67%
Answered correctly

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Hemodynamic effects of adrenergic agonists

- α_1: peripheral vasoconstriction, ↑ SVR
- β_1: ↑ heart rate & contractility, ↑ cardiac output
- β_2: peripheral vasodilation, ↓ SVR
- D_1: renal arteriolar vasodilation

Agonist	Receptor binding	SVR	Cardiac output	Renal blood flow
Dobutamine	$\beta_1 > \beta_2$	↓	↑	–
Dopamine	$D_1 > \beta_1 > \alpha_1$ (low-dose)	–/↓	↑	↑
	$\alpha_1 > \beta_1 > D_1$ (high-dose)	↑	–/↓	↓
Epinephrine	$\beta_1 > \beta_2 > \alpha_1$ (low-dose)	–/↓	↑	–
	$\alpha_1 > \beta_1 > \beta_2$ (high-dose)	↑	–/↓	↓
Isoproterenol	$\beta_1 = \beta_2$	↓	↑	–
Norepinephrine	$\alpha_1 = \beta_1 > \beta_2$	↑	–	↓
Phenylephrine	α_1	↑	↓	↓

SVR = systemic vascular resistance.

Dopamine is an adrenergic agonist that has varying effects based on the dose. At **low doses**, dopamine stimulates **D1 receptors** in the renal vasculature and tubules, inducing an **increase in renal blood flow**

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SVR = systemic vascular resistance.

Dopamine is an adrenergic agonist that has varying effects based on the dose. At **low doses**, dopamine stimulates **D1 receptors** in the renal vasculature and tubules, inducing an **increase in renal blood flow**, glomerular filtration rate, and sodium excretion. Vasodilation of the mesenteric, cerebral, and coronary vascular beds is also prominent at low doses.

As the dosage increases, dopamine begins to stimulate **$\beta_1$adrenergic receptors** in the heart, increasing cardiac contractility, heart rate, and systolic blood pressure. At the high end of the dose range, stimulation of **$\alpha_1$receptors** in the systemic vasculature leads to generalized vasoconstriction, which can result in decreased cardiac output due to the increased afterload.

(Choice B) Edrophonium is a short-acting acetylcholinesterase inhibitor used in the diagnosis of myasthenia gravis (ie, Tensilon test). Through its cholinergic effects, edrophonium reduces heart rate, cardiac conduction, and cardiac contractility.

(Choice C) Epinephrine increases heart rate, myocardial contractility, and cardiac output by stimulating β_1 receptors. At low doses, stimulation of β_2 receptors in the skeletal muscle vasculature produces vasodilation that can cause decreased blood pressure. At high doses, α_1-induced vasoconstriction in the skin and viscera becomes more pronounced. Epinephrine does not significantly increase renal blood flow (even at lower doses) due to the lack of D_1 receptor stimulation.

(Choice D) Esmolol is a cardioselective β_1 blocker with a short duration of action. Esmolol decreases heart rate, myocardial contractility, and cardiac conduction without affecting renal blood flow.

(Choice E) Phenylephrine is a selective α_1-adrenergic receptor agonist that causes a generalized increase in peripheral vascular resistance and blood pressure. Its use results in significant renal, splanchnic, and mesenteric vasoconstriction.

Educational objective:

Low-dose dopamine infusion stimulates D1 receptors in the renal and mesenteric vasculature, resulting in

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As the dosage increases, dopamine begins to stimulate **β1adrenergic receptors** in the heart, increasing cardiac contractility, heart rate, and systolic blood pressure. At the high end of the dose range, stimulation of **α1receptors** in the systemic vasculature leads to generalized vasoconstriction, which can result in decreased cardiac output due to the increased afterload.

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Educational objective:
Low-dose dopamine infusion stimulates D_1 receptors in the renal and mesenteric vasculature, resulting in vasodilation and increased blood flow to these sites. Increasing doses of dopamine stimulate β_1 and α_1 receptors, resulting in increased cardiac output and elevated systemic vascular resistance. At the high end of the dose range, the increase in afterload can result in decreased cardiac output.

Pharmacology	Cardiovascular System	Sympathomimetic agents
Subject	System	Topic

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A 61-year-old woman is hospitalized due to chest pressure and shortness of breath. Two days ago her husband died in a car accident. The patient's medical history is significant for hypertension, hyperlipidemia, and gout. Five years ago, she was diagnosed with breast cancer and treated with surgery and chemotherapy. Blood pressure is 165/90 mm Hg and pulse is 95/min and regular. Physical examination is unremarkable. ECG shows normal sinus rhythm with T-wave inversions in the anterior leads. Echocardiogram shows hypokinesis of the apical wall with decreased left ventricular ejection fraction. Diagnostic coronary angiography shows no evidence of obstructive coronary artery disease. The patient is treated medically and discharged home. Four weeks later, she is seen in the office and has no symptoms. Repeat echocardiography demonstrates normal left ventricular wall motion and ejection fraction. Which of the following is the likely cause of this patient's initial presentation?

- A. Catecholamine-induced myocardial stunning
- B. Ischemia-induced transmural myocardial necrosis
- C. Myocardial hypertrophy and fibrosis due to uncontrolled hypertension
- D. Myocardial infiltration by mature lymphocytes
- E. Toxin-induced myocardial injury

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- ✓ A. Catecholamine-induced myocardial stunning (83%)
- B. Ischemia-induced transmural myocardial necrosis (4%)
- C. Myocardial hypertrophy and fibrosis due to uncontrolled hypertension (4%)
- D. Myocardial infiltration by mature lymphocytes (1%)
- E. Toxin-induced myocardial injury (5%)

Correct

83% Answered correctly

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Explanation

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A 61-year-old woman is hospitalized due to chest pressure and shortness of breath. Two days ago her husband died in
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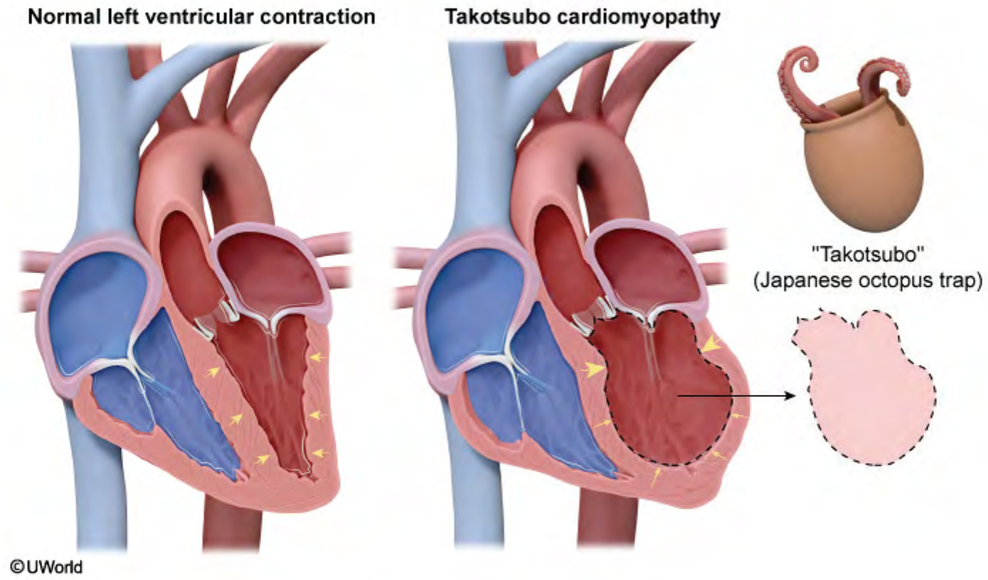

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This patient likely has **stress-induced (takotsubo) cardiomyopathy**, which is characterized by hypokinesis of the mid and apical segments and hyperkinesis of the basal segments of the left ventricle (LV), leading to **systolic dysfunction** and reduced ejection fraction. The condition is likely caused by a **catecholamine surge** in the setting of **physical or emotional stress** (eg, death of a loved one). Catecholamines may cause microvascular spasm leading to ischemia and myocardial stunning or they may cause direct myocardial dysfunction. The resulting segmental LV dysfunction creates a characteristic **balloon shape** on echocardiogram that mimics that of an octopus trap (takotsubo means "octopus trap" in Japanese).

Stress-induced cardiomyopathy most commonly affects **postmenopausal women**. Patients typically have chest pain that can mimic a myocardial infarction and may also have symptoms of heart failure (eg, dyspnea, lower extremity swelling). ECG often shows evidence of ischemia (eg, ST elevation, T-wave inversion) in the anterior precordial leads; however, coronary angiography typically reveals an **absence of obstructive coronary artery disease**. The condition usually resolves within several weeks with supportive treatment only.

(Choice B) Ischemia-induced transmural myocardial necrosis is seen in patients with acute myocardial infarction, which is unlikely in this patient with no evidence of obstructive coronary artery disease on coronary angiography.

(Choice C) LV hypertrophy due to uncontrolled hypertension typically leads to diastolic dysfunction without systolic wall motion abnormalities.

(Choices D and E) Myocardial infiltration with lymphocytes is seen in patients with acute viral myocarditis. Toxin-induced myocardial injury can occur with exposure to a variety of agents, including alcohol, cocaine, and certain chemotherapy drugs (eg, anthracyclines). These myocardial insults typically lead to dilated cardiomyopathy with global (rather than segmental) LV systolic dysfunction.

Educational objective:
Stress-induced (takotsubo) cardiomyopathy is characterized by hypokinesis of the mid and apical segments and hyperkinesis of the basal segments of the left ventricle, resulting in systolic dysfunction. The condition is likely caused by a surge of catecholamines in the setting of physical or emotional stress. It usually affects

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Stress-induced (takotsubo) cardiomyopathy is characterized by hypokinesis of the mid and apical segments and hyperkinesis of the basal segments of the left ventricle, resulting in systolic dysfunction. The condition is likely caused by a surge of catecholamines in the setting of physical or emotional stress. It usually affects postmenopausal women and resolves on its own within several weeks.

References

- Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review.

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A 66-year-old man comes to the hospital due to sudden-onset chest pain and dyspnea. The patient has a history of asthma and gastroesophageal reflux disease but says his current symptoms feel different than what he usually experiences. An ECG is consistent with ST-elevation myocardial infarction, and an emergent cardiac catheterization is performed. Evaluation of the left and right coronary arteries reveals left-dominant circulation. A stenotic region is identified in one of the coronary vessels just before the origin of the artery supplying the atrioventricular node. Which of the following arteries is most likely affected?

- A. Anterior interventricular artery
- B. Left circumflex artery
- C. Left diagonal artery
- D. Right coronary artery
- E. Right marginal artery

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- A. Anterior interventricular artery (6%)
- ✓ B. Left circumflex artery (55%)
- C. Left diagonal artery (2%)
- D. Right coronary artery (30%)
- E. Right marginal artery (4%)

Correct

55% Answered correctly

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Explanation

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A. Anterior interventricular artery (6%)

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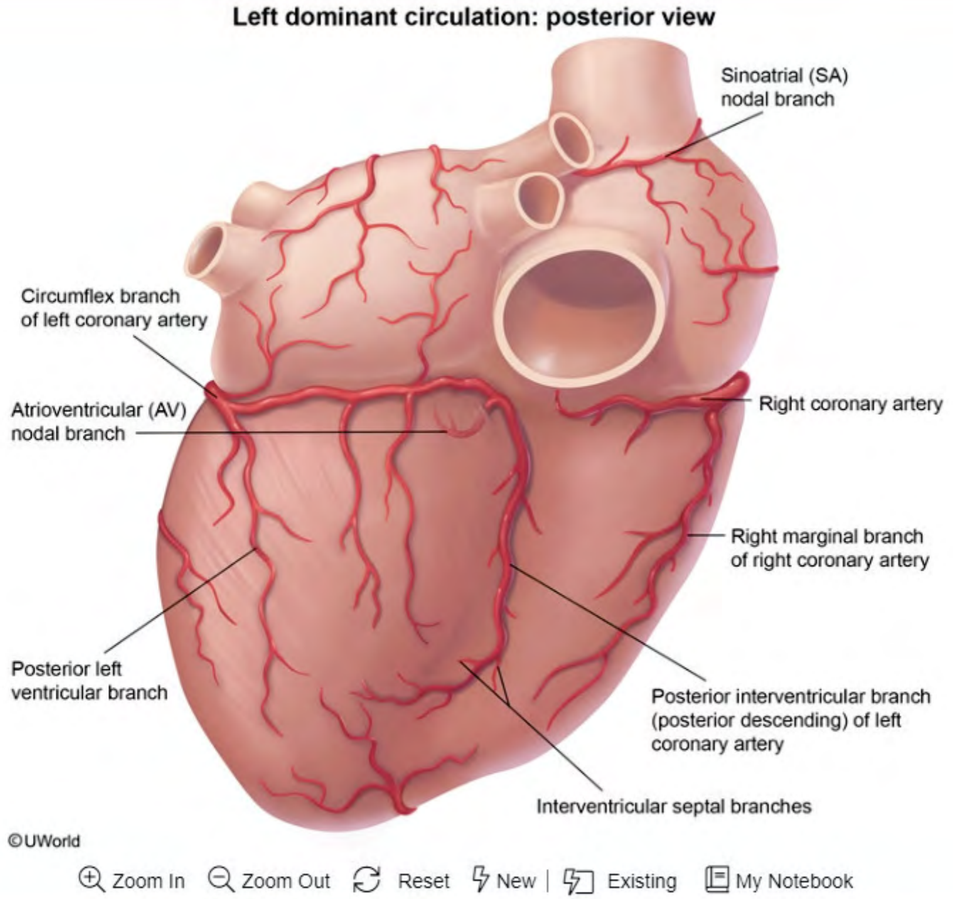

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Interventricular septal branches

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Coronary dominance is determined by the coronary artery that supplies blood to the **posterior descending artery** (PDA [or posterior interventricular artery]). The PDA originates from one of the following:

- Right coronary artery in approximately 70%-80% of the population (right dominant)
- Left circumflex artery in approximately 5%-10% of the population (**left dominant**)
- Both right coronary and left circumflex artery in 10%-20% of the population (codominant)

The atrioventricular **(AV) nodal artery** most often **arises** from the **dominant coronary artery**. This patient has left-dominant coronary circulation; therefore, his atherosclerotic lesion is most likely in the **left circumflex artery**. Involvement of the AV nodal artery during myocardial infarction can cause varying degrees of AV block.

(Choice A) The left anterior descending artery travels in the anterior interventricular groove and supplies the anterior part of the septum and the anterior wall of the left ventricle. It does not supply the AV node.

(Choice C) The diagonal arteries arise from the left anterior descending artery and supply the anterolateral wall of the left ventricle.

(Choice D) The right coronary artery gives rise to the AV nodal artery in patients with right-dominant or codominant circulation. This patient has left-dominant circulation, so the AV node is most likely supplied by the left circumflex artery.

(Choice E) The right marginal arteries arise from the right coronary artery and supply blood to the free wall of the right ventricle.

Educational objective:
Coronary dominance is determined by the coronary artery supplying the posterior descending artery. The posterior descending artery originates from the right coronary artery in approximately 70%-80% of patients (right dominant), both the right coronary and left circumflex artery in 10%-20% (codominant), and the left circumflex artery in 5%-10% (left dominant). The dominant coronary artery supplies blood to the atrioventricular (AV) node via the AV nodal artery.

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- Right coronary artery in approximately 70%-80% of the population (right dominant)
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References

- Anatomical aspects of the arterial blood supply to the sinoatrial and atrioventricular nodes of the human heart.

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Posterior left ventricular branch

Posterior interventricular branch

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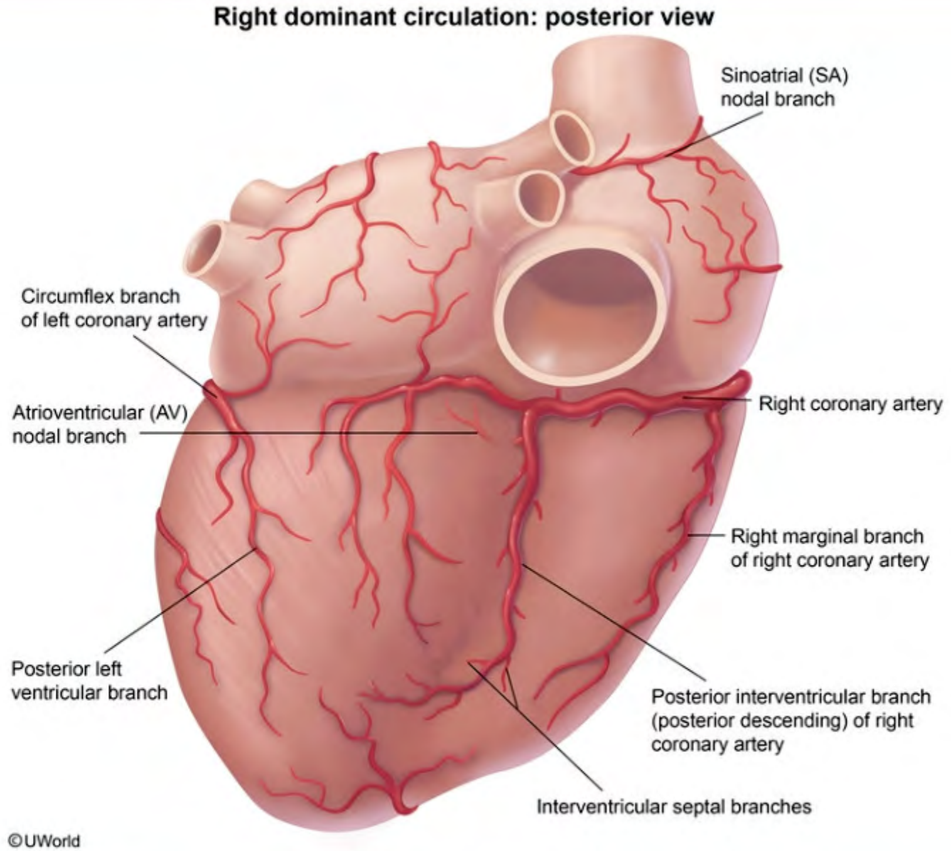

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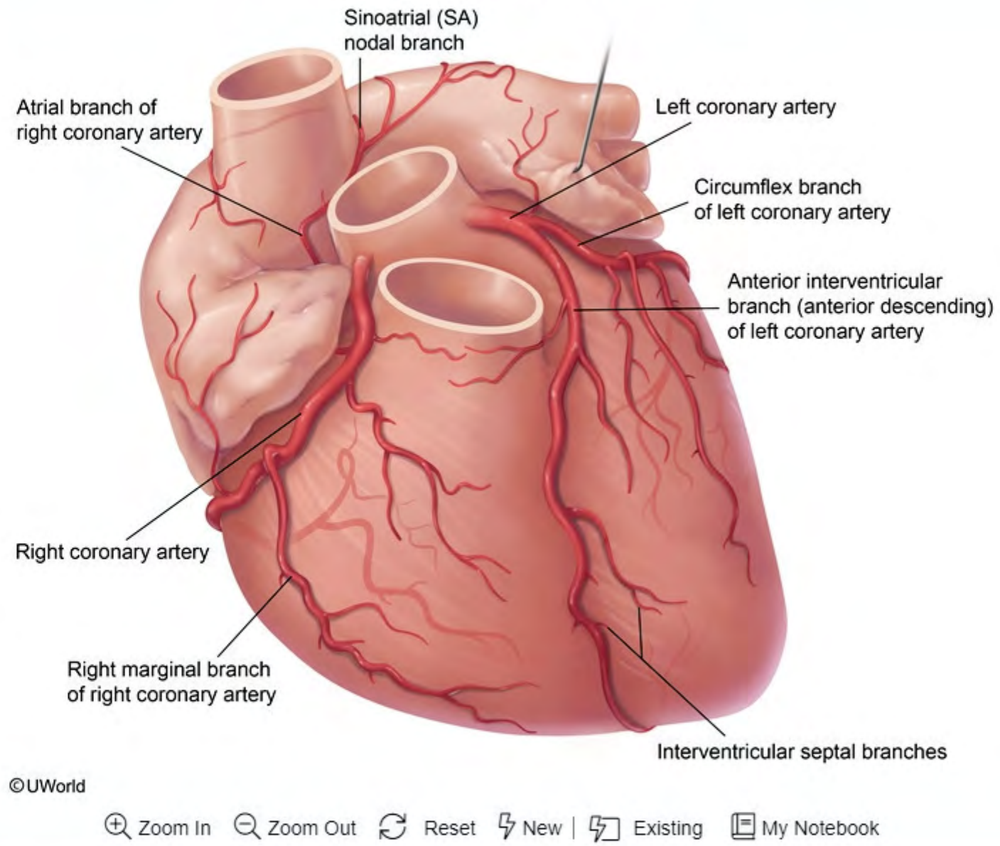

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A 27-year-old man comes to the office due to dyspnea on exertion and intermittent episodes of palpitations. Blood pressure is 130/80 mm Hg and pulse is 78/min. On examination, there is no peripheral edema or jugular venous distension. The lungs are clear to auscultation. There is a 3/6 ejection-type systolic murmur along the left sternal border that decreases in intensity when the patient moves from the standing to the squatting position. Which of the following is the most likely form of inheritance of this patient's condition?

- A. Autosomal dominant
- B. Autosomal recessive
- C. Multifactorial
- D. X-linked dominant
- E. X-linked recessive

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- ✓ A. Autosomal dominant (58%)
- ✗ B. Autosomal recessive (12%)
- C. Multifactorial (22%)
- D. X-linked dominant (2%)
- E. X-linked recessive (3%)

Incorrect
Correct answer
A

58%
Answered correctly

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Explanation

Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	Left ventricular blood volume	Murmur intensity
Valsalva (strain phase)			
Abrupt	↓ Preload	↓	↑

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Explanation

Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	**Left ventricular blood volume**	**Murmur intensity**
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

This patient's clinical presentation is consistent with **hypertrophic cardiomyopathy** (HCM). Many patients with HCM are asymptomatic and are recognized when an abnormal murmur or ECG is noted during evaluation for other purposes. HCM typically involves left ventricular (LV) enlargement that is most prominent in the basal anterior septum and can lead to **LV outflow tract obstruction**. The clinical manifestations, which depend on the extent of hypertrophy and outflow tract obstruction, include:

- exertional dyspnea, chest pain, fatigue, palpitations, presyncope, or syncope. Sudden cardiac arrest can also

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Squatting	↑ Afterload & preload	↑	↓
Passive leg raise	↑ Preload		

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- exertional dyspnea, chest pain, fatigue, palpitations, presyncope, or syncope. Sudden cardiac arrest can also occur.
- a harsh crescendo-decrescendo systolic murmur best heard at the apex and left sternal border, with characteristic changes in intensity during physiologic maneuvers.

The condition is inherited in **autosomal dominant** fashion, resulting from mutations in genes encoding for myocardial contractile proteins of the sarcomere. The proteins **cardiac myosin binding protein C** and **cardiac beta-myosin heavy chain** are most commonly affected. The phenotypic manifestations of the disease (eg, septal enlargement) typically begin to develop around the time of adolescence.

Despite the autosomal dominant inheritance, affected patients may not always have an identifiable first-degree relative who is similarly affected because not all mutations responsible for HCM have been identified and many known mutations have variable penetrance.

Educational objective:
Hypertrophic cardiomyopathy (HCM) is an autosomal dominant genetic disorder caused by mutation in one of several genes encoding the myocardial contractile proteins of the cardiac sarcomere. Mutations that affect cardiac myosin binding protein C or cardiac beta-myosin heavy chain are responsible for the majority of identifiable mutations in patients with HCM.

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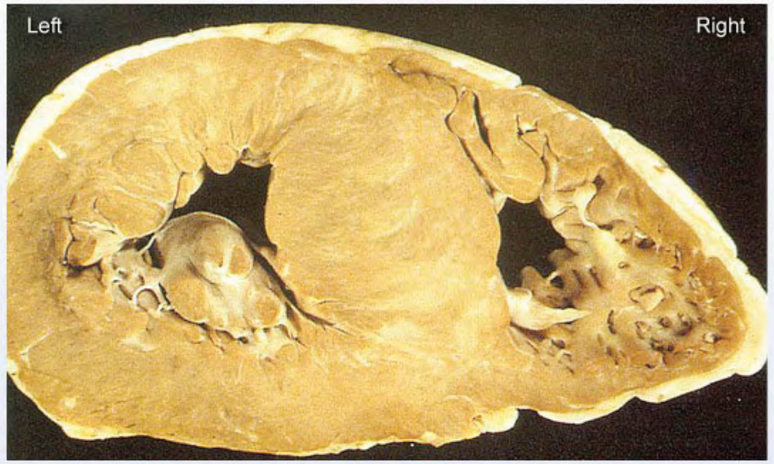
A 25-year-old man with an insignificant medical history was playing soccer when he suddenly collapsed. Despite all attempts to save his life, he died. Autopsy is performed. Gross examination of the heart reveals a weight of 580 g (normal: <350). The cut surface of the heart is shown in the image.



If this patient had a preparticipation sports screening, cardiac auscultation would have likely revealed a murmur that increases in intensity after which of the following?

- A. Passive leg raising
- B. Phenylephrine infusion
- C. Squatting
- D. Sudden standing
- E. Sustained hand grip

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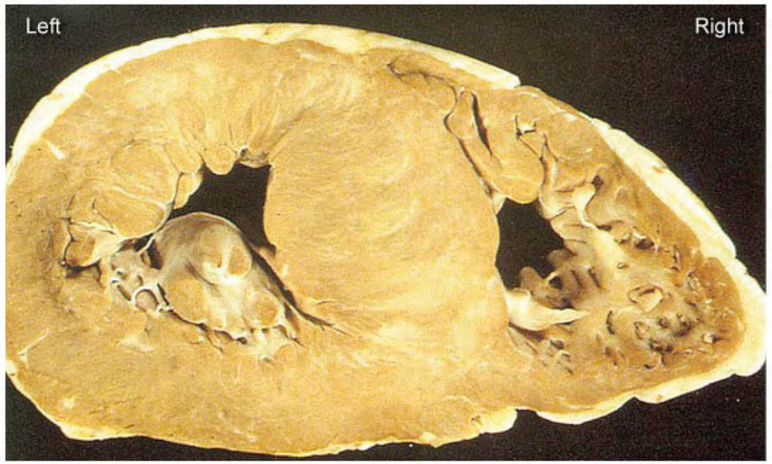

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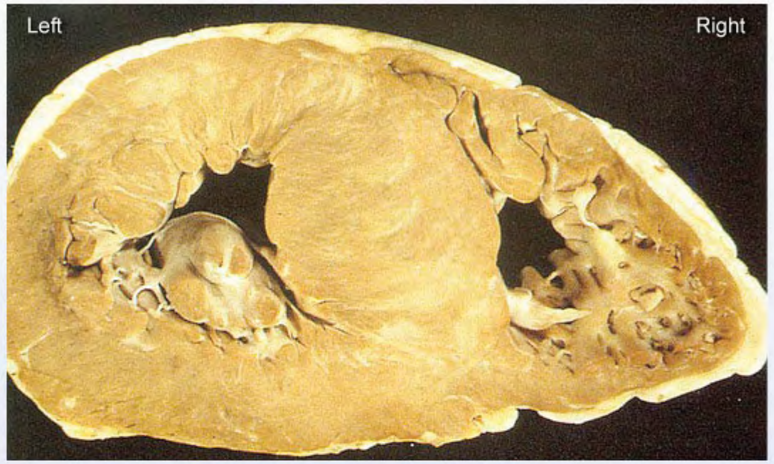

If this patient had a preparticipation sports screening, cardiac auscultation would have likely revealed a murmur that increases in intensity after which of the following?

- A. Passive leg raising (6%)
- ✗ B. Phenylephrine infusion (2%)
- C. Squatting (14%)
- ✓ D. Sudden standing (65%)
- E. Sustained hand grip (11%)

Incorrect
Correct answer

65%
Answered correctly

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Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	**Left ventricular blood volume**	**Murmur intensity**
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

This patient's heart shows asymmetric ventricular septal hypertrophy, a finding characteristic of **hypertrophic cardiomyopathy** (HCM). Dynamic left ventricular outflow tract (LVOT) obstruction is common in HCM and creates a harsh crescendo-decrescendo, **systolic ejection murmur** that is best heard along the lower left sternal border.

Maneuvers or conditions that **decrease LV preload** (eg, abrupt standing, Valsalva strain phase, dehydration) or **decrease LV afterload** (eg, administration of an arterial vasodilator) decrease LV blood volume to worsen LVOT obstruction and **increase the intensity** of the HCM murmur. In contrast, maneuvers or conditions that increase LV preload (eg, passive leg raising, squatting) or increase LV afterload (eg, infusion of an arterial vasoconstrictor like the alpha-1 agonist phenylephrine, sustained handgrip) increase LV blood volume to alleviate LVOT obstruction

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leg raise			

This patient's heart shows asymmetric ventricular septal hypertrophy, a finding characteristic of **hypertrophic cardiomyopathy** (HCM). Dynamic left ventricular outflow tract (LVOT) obstruction is common in HCM and creates a harsh crescendo-decrescendo, **systolic ejection murmur** that is best heard along the lower left sternal border.

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The murmur of HCM is best differentiated from that of aortic stenosis by maneuvers that affect LV preload. Unlike HCM, the intensity of the aortic stenosis murmur correlates directly with LV preload. Increases in LV preload increase the pressure gradient across the aortic valve and increase the intensity of the murmur, whereas decreases in LV preload decrease the intensity of the murmur. Changes in afterload have the same effect on the murmurs of HCM and aortic stenosis, but for different reasons. In HCM, increased afterload increases LV blood volume to relieve LVOT obstruction and decrease the intensity of the murmur. In aortic stenosis, increased afterload decreases the pressure gradient across the aortic valve to decrease the intensity of the murmur.

Educational objective:
Hypertrophic cardiomyopathy (HCM) is characterized by asymmetric ventricular septal hypertrophy and dynamic left ventricular outflow tract (LVOT) obstruction. Decreases in LV blood volume, via maneuvers or conditions that decrease preload (eg, abrupt standing, Valsalva strain phase) or afterload, worsen LVOT obstruction and increase the intensity of the HCM murmur.

Pathophysiology	Cardiovascular System	Hypertrophic cardiomyopathy
Subject	System	Topic

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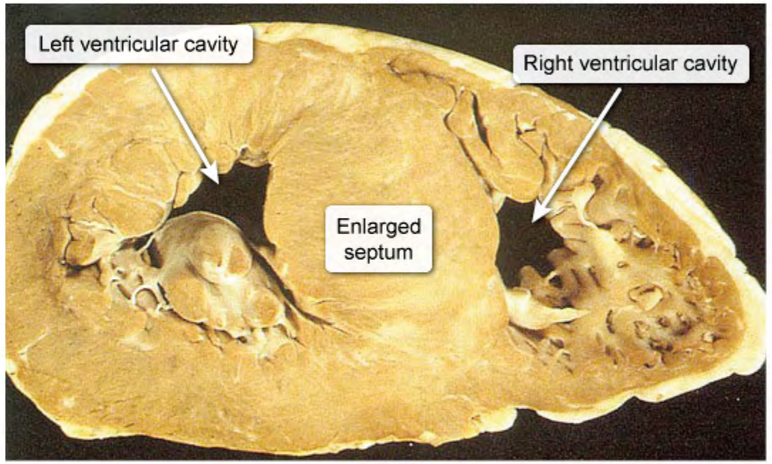
Hypertrophic cardiomyopathy



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Educational objective:
Hypertrophic cardiomyopathy (HCM) is characterized by asymmetric ventricular septal hypertrophy and dynamic

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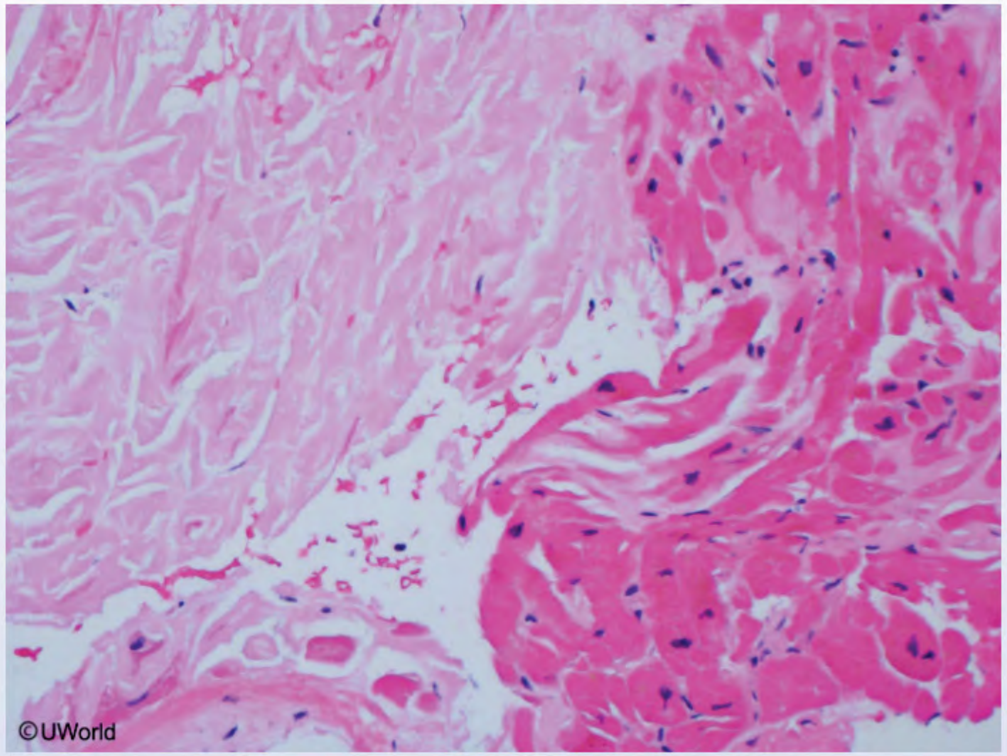
A 65-year-old man is evaluated for progressive shortness of breath. He has had exertional dyspnea for the past year and has been experiencing weight gain and peripheral edema for the last several weeks. He has no previous history of cardiac or pulmonary disease. Cardiac biopsy of the left ventricle shows the histopathologic findings on the slide below.



Which of the following are the most likely echocardiographic findings in this patient?

	Left atrial cavity size	Left ventricular cavity size	Left ventricular wall thickness	Left ventricular relaxation

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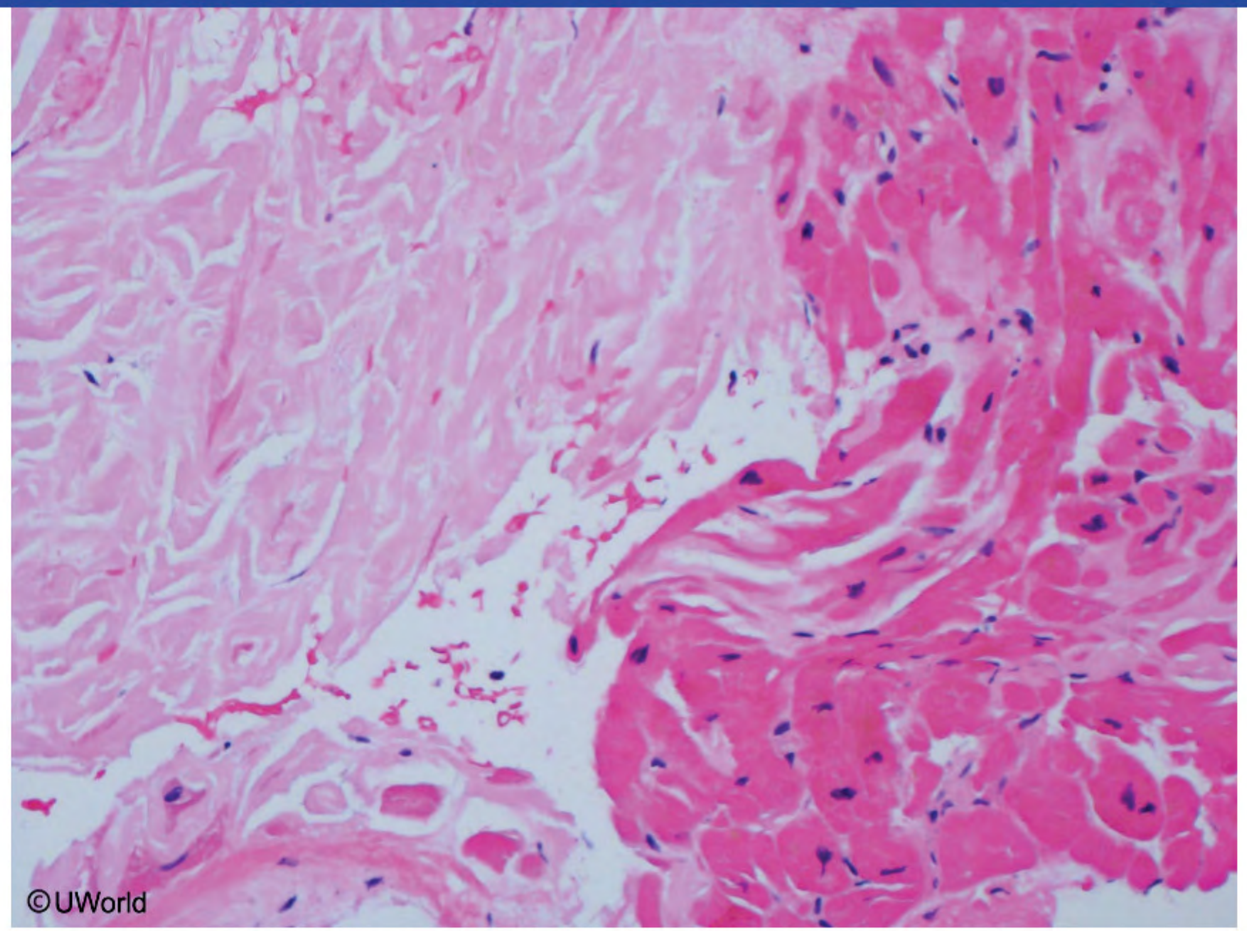

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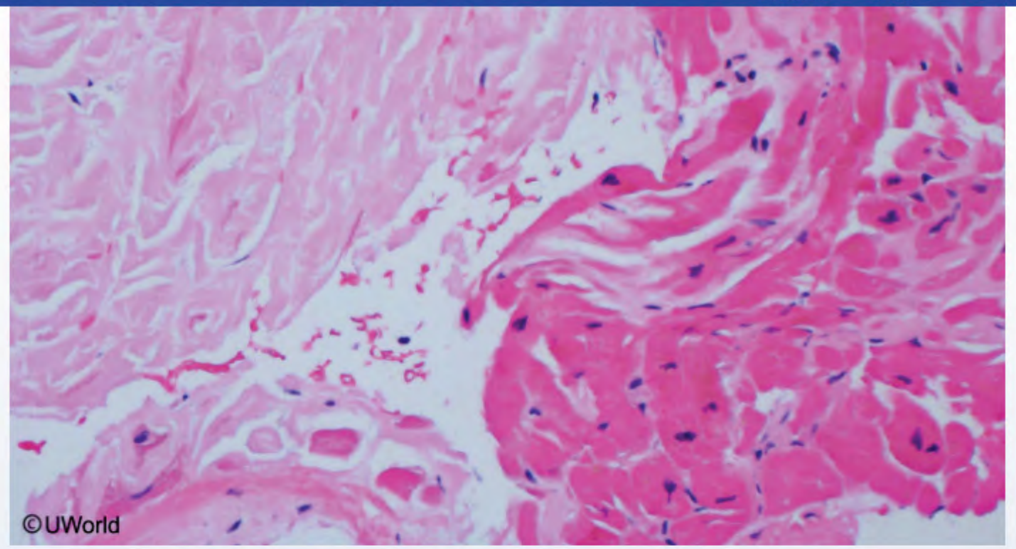

Which of the following are the most likely echocardiographic findings in this patient?

	Left atrial cavity size	Left ventricular cavity size	Left ventricular wall thickness	Left ventricular relaxation
A.	Dilated	Not dilated	Normal	Normal
B.	Dilated	Dilated	Normal	Impaired
C.	Dilated	Not dilated	Increased	Impaired
D.	Not dilated	Dilated	Increased	Impaired
E.	Not dilated	Dilated	Increased	Normal

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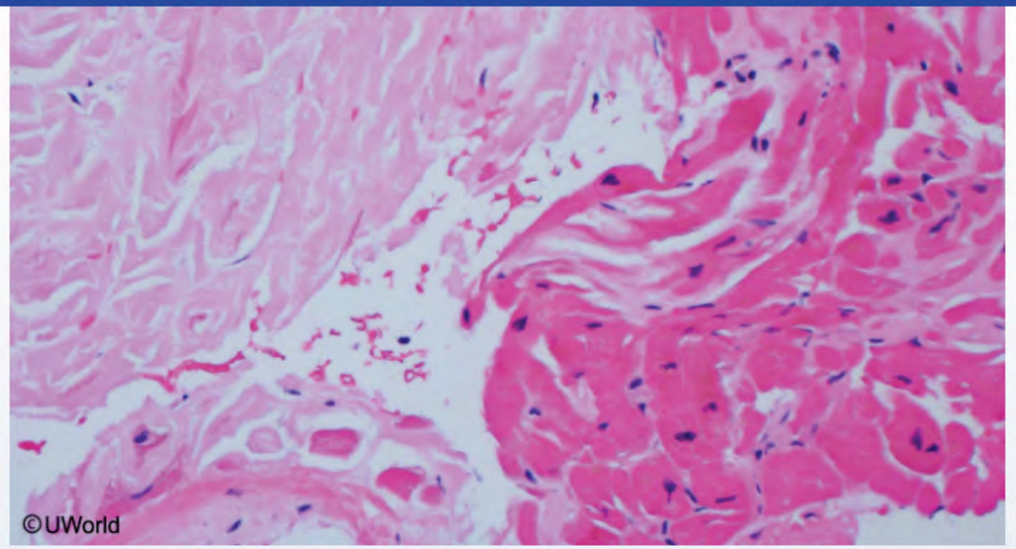

Which of the following are the most likely echocardiographic findings in this patient?

	Left atrial cavity size	Left ventricular cavity size	Left ventricular wall thickness	Left ventricular relaxation	
✗ A.	Dilated	Not dilated	Normal	Normal	(3%)
B.	Dilated	Dilated	Normal	Impaired	(19%)
✓ C.	Dilated	Not dilated	Increased	Impaired	(55%)
D.	Not dilated	Dilated	Increased	Impaired	(17%)
E.	Not dilated	Dilated	Increased	Normal	(5%)

Incorrect
Correct answer
C

55%
Answered correctly

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D. Not dilated Dilated Increased Impaired (17%)

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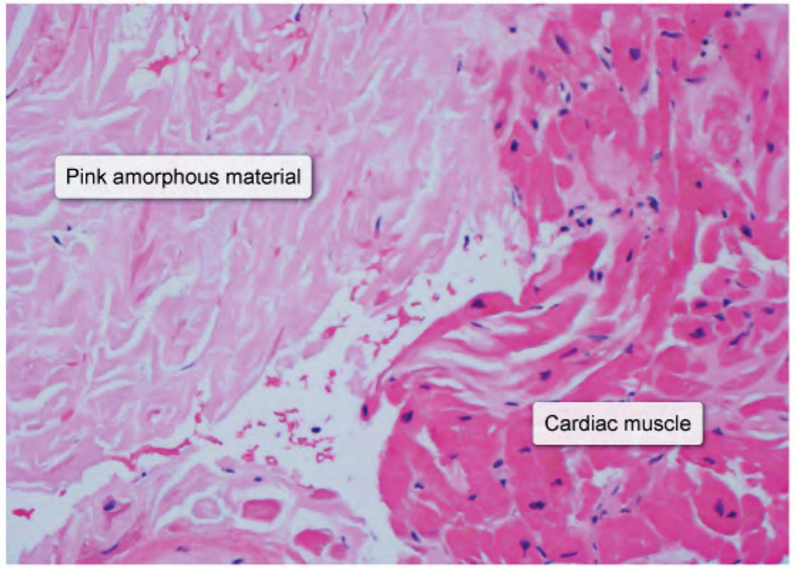
Amyloid



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systemic disorder caused by the **extracellular deposition** of fibrils of misfolded amyloid protein in various tissues (eg, heart, liver, kidney, blood vessels). The 2 types of amyloidosis that most commonly affect the heart are

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This patient with **heart failure** (eg, progressive dyspnea, weight gain, peripheral edema) has copious pink, amorphous material (hyaline) on cardiac biopsy, which is characteristic of **cardiac amyloidosis**. Amyloidosis is a systemic disorder caused by the **extracellular deposition** of fibrils of misfolded amyloid protein in various tissues (eg, heart, liver, kidney, blood vessels). The 2 types of amyloidosis that most commonly affect the heart are amyloid transthyretin (ATTR) and amyloid light chain (AL).

Cardiac amyloid deposition typically causes a **restrictive cardiomyopathy** with uniformly **thickened ventricular walls** that are stiff and have **impaired diastolic relaxation** (normal wall thickness can be seen with other, noninfiltrative etiologies of restrictive cardiomyopathy). Left ventricular (LV) cavity size is normal or decreased, and the impaired diastolic relaxation both increases LV filling pressure and reduces cardiac output (leading to dyspnea). The elevated LV pressure is transmitted backward, leading to **left atrial dilation**, increased pulmonary arterial pressure, and progressive right-sided heart failure (eg, jugular venous distension, peripheral edema).

(Choice A) A dilated left atrial cavity with normal LV parameters is indicative of mitral stenosis. Patients typically have pulmonary edema in addition to symptoms of right-sided heart failure. Accumulation of amorphous extracellular material is not expected on autopsy.

(Choice B) Dilated left atrial and ventricular cavities with normal LV wall thickness is consistent with dilated cardiomyopathy, which results from contractile dysfunction after direct myocardial insult (eg, infection, chronic alcohol toxicity). LV relaxation may be normal or impaired depending on the disease progression and underlying etiology. Interstitial fibrosis would be prominent on microscopy in addition to findings specific to the underlying cause (eg, inflammatory cells in viral myocarditis).

(Choice D) These changes are not characteristic of any specific cardiac condition.

(Choice E) Normal left atrial cavity size with a dilated LV cavity and thickened LV walls but normal LV relaxation is consistent with the changes that occur in response to intense endurance training in an athlete's heart. As these changes are physiologic, heart failure does not occur.

Educational objective:

Cardiac amyloidosis, caused by the accumulation of misfolded amyloid fibrils, appears as pink, amorphous

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systemic disorder caused by the **extracellular deposition** of fibrils of misfolded amyloid protein in various tissues (eg, heart, liver, kidney, blood vessels). The 2 types of amyloidosis that most commonly affect the heart are amyloid transthyretin (ATTR) and amyloid light chain (AL).

Cardiac amyloid deposition typically causes a **restrictive cardiomyopathy** with uniformly **thickened ventricular walls** that are stiff and have **impaired diastolic relaxation** (normal wall thickness can be seen with other, noninfiltrative etiologies of restrictive cardiomyopathy). Left ventricular (LV) cavity size is normal or decreased, and the impaired diastolic relaxation both increases LV filling pressure and reduces cardiac output (leading to dyspnea). The elevated LV pressure is transmitted backward, leading to **left atrial dilation**, increased pulmonary arterial pressure, and progressive right-sided heart failure (eg, jugular venous distension, peripheral edema).

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Educational objective:
Cardiac amyloidosis, caused by the accumulation of misfolded amyloid fibrils, appears as pink, amorphous extracellular material on light microscopy. The ventricular walls become stiff and uniformly thickened, leading to impaired diastolic relaxation, a dilated left atrial cavity, and progressive left- and right-sided heart failure. Left ventricular cavity size is typically normal or decreased.

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Major types of systemic amyloidosis

	Primary	Secondary	Age related	Dialysis related
Disease association	Plasma cell dyscrasia	Chronic infection/ inflammation	Elderly men	ESRD on dialysis
Precursor protein ↓ **Misfolding mechanism** ↓ **Amyloid fibril type**	Immunoglobulin light chains ↓ Increased production ↓ AL	Serum amyloid A ↓ Increased production ↓ AA	Transthyretin (prealbumin) ↓ Accumulation over time ↓ ATTR*	β_2-Microglobulin ↓ Decreased clearance ↓ $A\beta_2M$
Clinical manifestations	Nephropathy, hepatosplenomegaly, cardiomyopathy (AL > AA), peripheral neuropathy, macroglossia, skin bruising		Cardiomyopathy, CTS, other peripheral neuropathy	Scapulohumeral arthritis, CTS

Amyloid names begin with "A" followed by the precursor protein abbreviation.
*ATTR also occurs in a hereditary form that affects younger patients.
CTS = carpal tunnel syndrome; **ESRD** = end-stage renal disease.

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Cardiac amyloidosis, caused by the accumulation of misfolded amyloid fibrils, appears as pink, amorphous

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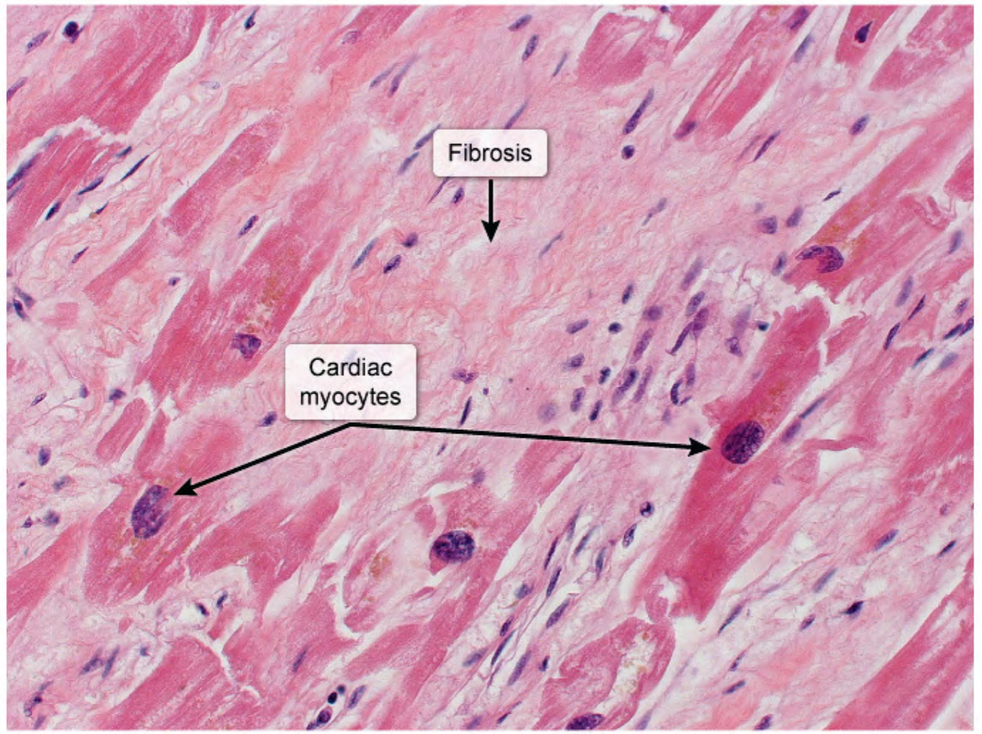
Cardiac interstitial fibrosis



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A 34-year-old man diagnosed with advanced non-Hodgkin lymphoma undergoes doxorubicin-containing chemotherapy with good clinical response. Several weeks after the last chemotherapy cycle, the patient comes to the office with progressive exertional dyspnea. He has difficulty sleeping flat at night and needs to use multiple pillows to fall asleep. The patient is a lifetime nonsmoker and has no family history of heart disease. Blood pressure is 124/72 mm Hg, pulse is 86/min, and respirations are 16/min. Oxygen saturation on room air is 96%. Which of the following is most likely responsible for this patient's current symptoms?

- A. Dilated cardiomyopathy
- B. Focal myocardial scarring
- C. Hypertrophic cardiomyopathy
- D. Pericardial thickening and fibrosis
- E. Restrictive cardiomyopathy
- F. Right ventricular pressure overload

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- ✓ A. Dilated cardiomyopathy (75%)
- B. Focal myocardial scarring (1%)
- C. Hypertrophic cardiomyopathy (2%)
- D. Pericardial thickening and fibrosis (6%)
- E. Restrictive cardiomyopathy (10%)
- F. Right ventricular pressure overload (3%)

Correct

75% Answered correctly

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Explanation

Specific toxicities of common chemotherapeutic agents		
Chemotherapy agent	**Mechanism of Action**	**Toxicity**
Anthracyclines (eg, doxorubicin)	• Binds with topoisomerase II to cleave DNA • Binds with iron to generate free radicals	• Dilated cardiomyopathy
Bleomycin	• Induces free radical formation	• Pulmonary fibrosis

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Bleomycin	• Induces free radical formation	• Pulmonary fibrosis
Cisplatin	• Cross-links DNA to inhibit DNA synthesis	• Nephrotoxicity • Ototoxicity • Peripheral neuropathy
Cyclophosphamide	• Cross-links DNA to inhibit DNA synthesis	• Hemorrhagic cystitis • Bladder cancer
Paclitaxel	• Inhibits microtubule disassembly	• Neuropathy
Vincristine/ vinblastine	• Binds beta-tubulin to inhibit microtubule formation	• Neuropathy

Anthracyclines (eg, daunorubicin, doxorubicin) are chemotherapeutic agents associated with severe cardiotoxicity that manifests as **dilated cardiomyopathy**. The toxicity is dependent on the cumulative dose of anthracycline; affected patients develop symptoms of heart failure (eg, dyspnea on exertion, orthopnea, peripheral edema) anywhere from several weeks to years following treatment.

Anthracyclines exert their antitumor effect by binding to the **topoisomerase II** enzyme in cancer cells to create DNA cleavage complexes that **break DNA** and cause cell death. Anthracycline cardiotoxicity results from a similar anthracycline–topoisomerase II complex that forms in healthy cardiomyocytes. In addition, anthracycline can form iron-containing complexes that produce DNA-damaging free radicals, which likely also plays a role in cardiotoxicity.

The most effective method of preventing anthracycline-induced cardiomyopathy is the administration of **dexrazoxane**, a chelating agent thought to both block the formation of iron-associated free radicals and inhibit the

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The most effective method of preventing anthracycline-induced cardiomyopathy is the administration of **dexrazoxane**, a chelating agent thought to both block the formation of iron-associated free radicals and inhibit the formation of anthracycline–topoisomerase II complexes in healthy cardiomyocytes.

(Choice B) Focal myocardial scarring commonly results following a myocardial infarction.

(Choice C) Hypertrophic cardiomyopathy is an autosomal dominant disorder often caused by mutation of the beta-myosin heavy chain.

(Choice D) Pericardial thickening and fibrosis usually follow cardiac surgery, radiation therapy, or viral infections of the pericardium.

(Choice E) Restrictive cardiomyopathy often occurs as the initial cardiac manifestation of infiltrative disease (eg, hemochromatosis, amyloidosis, sarcoidosis) or may occur with radiation therapy.

(Choice F) Right ventricular pressure overload can occur with acute pulmonary embolism. Although this patient with malignancy is at increased risk for venous thromboembolic disease, orthopnea is not explained by acute pulmonary embolism and is more consistent with heart failure.

Educational objective:
Anthracycline chemotherapeutic agents (eg, doxorubicin, daunorubicin) cause cardiotoxicity mainly through the formation of anthracycline–topoisomerase II DNA cleavage complexes that affect healthy cardiomyocytes. The cardiotoxicity is dependent on the cumulative dose of anthracycline received, and it manifests as dilated cardiomyopathy.

References

- Anthracycline chemotherapy and cardiotoxicity

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A 45-year-old woman who recently immigrated to the United States is hospitalized with exertional dyspnea and fatigue. She has no significant past medical history and takes no medications. The patient's blood pressure is 110/80 mm Hg and heart rate is 90/min and regular. After cardiopulmonary examination, the physician suspects mitral stenosis. Which of the following is the most useful measure for assessing the degree of mitral stenosis?

- A. A2-to-opening snap time interval
- B. Audible S3
- C. Audible S4
- D. Diastolic murmur intensity
- E. Presystolic accentuation of the murmur

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A 45-year-old woman who recently immigrated to the United States is hospitalized with exertional dyspnea and fatigue. She has no significant past medical history and takes no medications. The patient's blood pressure is 110/80 mm Hg and heart rate is 90/min and regular. After cardiopulmonary examination, the physician suspects mitral stenosis. Which of the following is the most useful measure for assessing the degree of mitral stenosis?

- ✓ A. A2-to-opening snap time interval (63%)
- ✗ B. Audible S3 (6%)
- C. Audible S4 (3%)
- D. Diastolic murmur intensity (22%)
- E. Presystolic accentuation of the murmur (4%)

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Correct answer
A

63%
Answered correctly

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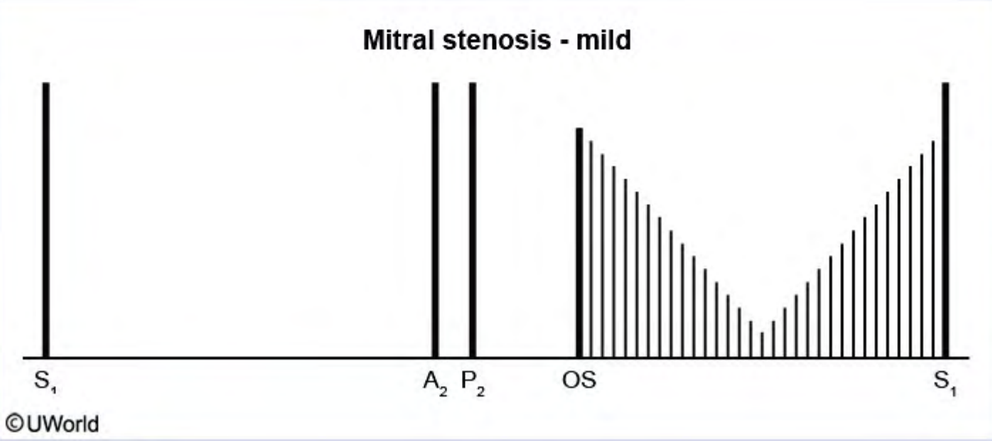
Explanation



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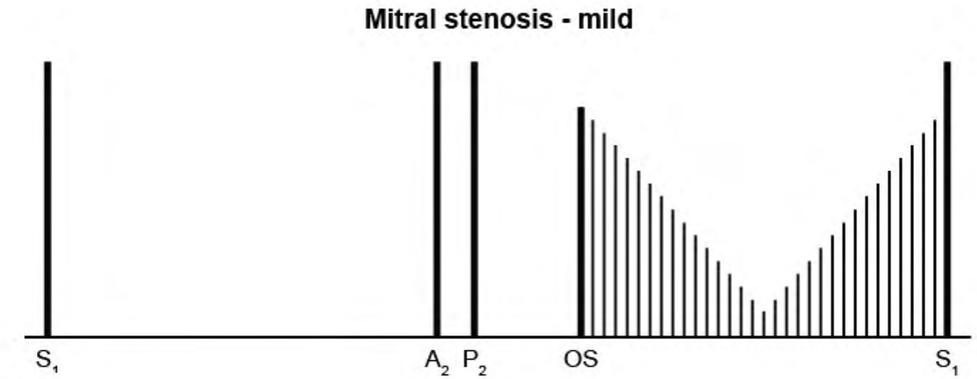

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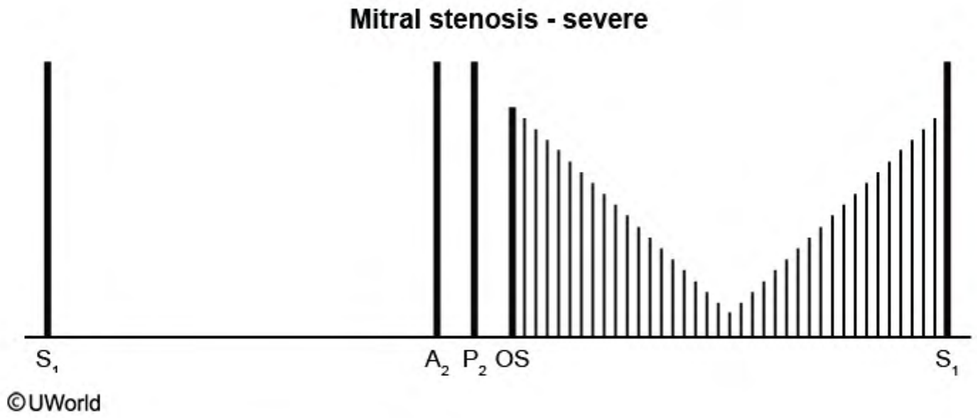

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(Choices B and C) Left-sided S3 and/or S4 gallops are generally absent in MS, since left ventricular filling is normal or decreased. When MS is severe enough to produce pulmonary hypertension, patients may develop right-

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On auscultation, the best indicator of mitral stenosis (MS) severity is the length of time between S2 (specifically the A2 component, caused by aortic valve closure) and the opening snap (OS). The OS occurs due to abrupt tensing of the valve leaflets as the mitral valve reaches its maximum diameter during forceful opening. As MS worsens, left atrial pressures increase due to impaired movement of blood into the left ventricle. Higher pressure causes the valve to open more forcefully; as a result, the A2-OS interval becomes shorter as left atrial pressure increases. The current standard for diagnosis and determination of MS severity is measurement of mean transvalvular pressure gradients via 2-D Doppler echocardiography.

(Choices B and C) Left-sided S3 and/or S4 gallops are generally absent in MS, since left ventricular filling is normal or decreased. When MS is severe enough to produce pulmonary hypertension, patients may develop right-sided heart failure. As a result, a right-sided S3 and/or S4 can occur. However, these right-sided gallops arise only in severe, end-stage MS and are not useful for differentiating between mild and moderate MS.

(Choice D) Diastolic rumble intensity in MS does not correlate well with stenosis severity. This is due to murmur intensity being dependent on both the transvalvular pressure gradient and the amount of blood flowing through the valve. In very severe stenosis, the diastolic murmur may be completely absent due to greatly reduced flow across the valve.

(Choice E) Presystolic accentuation of the MS murmur occurs due to increased transvalvular flow associated with left atrial contraction. This accentuation is heard across a wide range of MS severity. More significant than the degree of presystolic accentuation is its presence or absence. When MS becomes severe enough to precipitate atrial fibrillation, presystolic accentuation of the MS murmur disappears.

Educational objective:
The best and most reliable auscultatory indicator of the degree of mitral stenosis is the A2-OS interval. A shorter interval indicates more severe stenosis. Other auscultatory findings can include a diastolic rumbling murmur with presystolic accentuation due to left-atrial contraction.

Pathophysiology	Cardiovascular System	Mitral stenosis
Subject	System	Topic

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A 10-year-old boy is brought to the emergency department due to shortness of breath and palpitations for the past day. He also has associated fever and fatigue. Vital signs indicate tachypnea, tachycardia, and hypotension. On cardiac auscultation, the patient has a new holosystolic murmur. He is admitted to the hospital for further workup and management. A cardiac biopsy is performed due to decompensation and an unclear diagnosis. Light microscopy of the tissue specimen is shown in the image below.

Which of the following most likely preceded development of this patient's current condition?

- A. Antibiotic exposure
- B. Bacterial infection
- C. Chemotherapy
- D. Genetic mutation
- E. Travel to South America
- F. Viral infection

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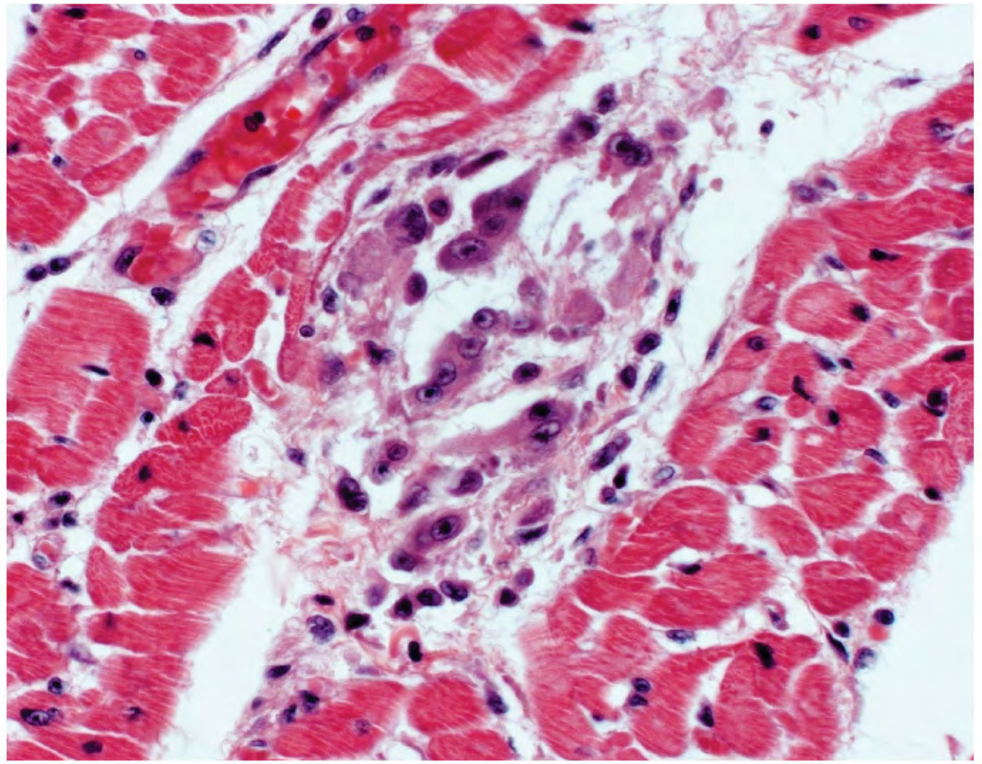

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Which

B. Bacterial infection

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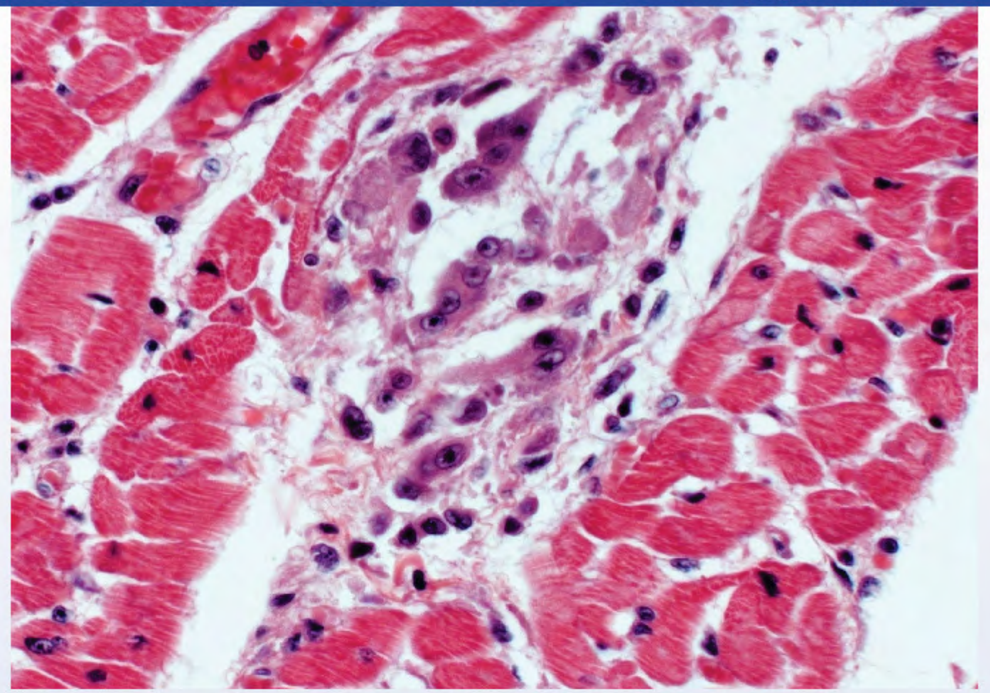

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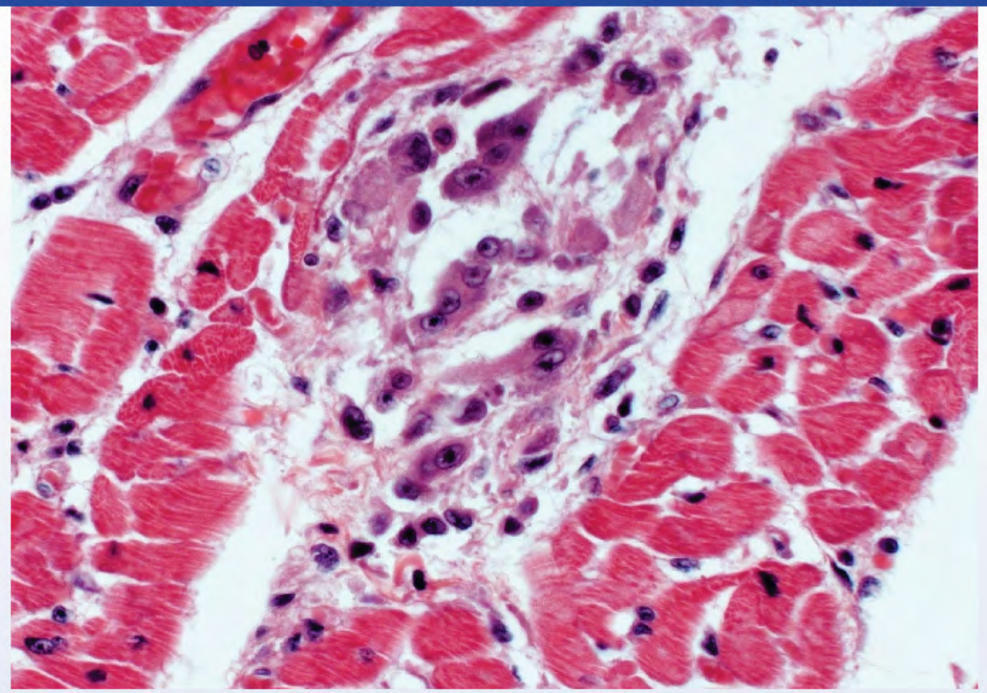

Which of the following most likely preceded development of this patient's current condition?

- A. Antibiotic exposure (0%)
- ✓ B. Bacterial infection (48%)
- ✗ C. Chemotherapy (1%)
- D. Genetic mutation (3%)
- E. Travel to South America (16%)
- F. Viral infection (29%)

Incorrect
Correct answer
B

48%
Answered correctly

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E. Travel to South America (16%)

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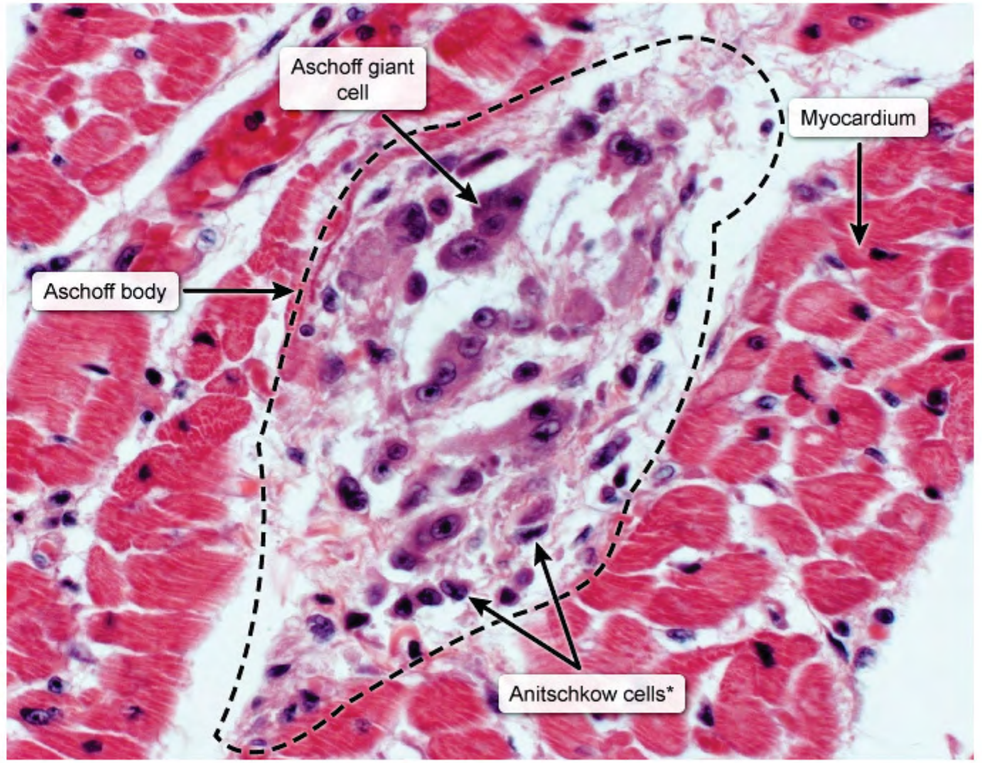
Acute rheumatic carditis



*Macrophages with characteristic "caterpillar" chromatin

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Acute rheumatic fever (ARF) is an immune-mediated complication of an **untreated group A streptococcal pharyngeal infection**. The most serious manifestation of ARF is **pancarditis**, which can cause nonspecific fever, fatigue, and anorexia as well as altered vital signs (tachycardia, tachypnea, hypotension). Endocardial involvement resulting in valvular dysfunction (specifically acute **mitral valve regurgitation**) is the most likely cause of the patient's new **holosystolic murmur**.

This patient's myocardial biopsy shows a lesion consisting of lymphocytes and macrophages as well as scattered multinucleated giant cells. This **interstitial myocardial granuloma**, or **Aschoff body**, is pathognomonic for ARF-related myocarditis. Plump macrophages with abundant cytoplasm and central, slender chromatin ribbons called Anitschkow (or caterpillar) cells are also often present. Over subsequent years, Aschoff bodies are replaced by **fibrous scar tissue**, leading to chronic **mitral valve stenosis** and regurgitation.

(Choice A) Hypersensitivity myocarditis, which results from an autoimmune reaction to a medication, is characterized by an interstitial infiltrate of eosinophils. Many classes of drugs, including diuretics (eg, furosemide, hydrochlorothiazide) and antibiotics (eg, ampicillin, azithromycin), can cause this hypersensitivity reaction.

(Choice C) Anthracyclines (eg, doxorubicin, daunorubicin) are cardiotoxic chemotherapeutic agents. These agents can cause dose-related acute and chronic cardiac damage (eg, dilated cardiomyopathy). On biopsy, patchy fibrosis with vacuolization and lysis of myocytes are evident.

(Choice D) A genetic mutation involving sarcomere genes can lead to the development of hypertrophic cardiomyopathy (HCM). HCM leads to left ventricular hypertrophy and, in turn, both systolic and diastolic dysfunction. The pathology involves disorganized, hypertrophied myocytes.

(Choice E) Recent travel to South America is associated with Chagas disease, which is caused by the protozoan parasite *Trypanosoma cruzi*. Chagas disease can result in a myocarditis characterized by distension of individual myofibers with intracellular trypanosomes.

(Choice F) Viral myocarditis produces a predominantly lymphocytic interstitial infiltrate with focal necrosis of myocytes. Aschoff bodies are not seen. Viral infection (eg, adenovirus, Coxsackie B virus, parvovirus B19) is the most common cause of myocarditis in the general population.

Educational objective:

Interstitial myocardial granulomas (Aschoff bodies) are found in carditis due to acute rheumatic fever, which

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involvement resulting in valvular dysfunction (specifically acute **mitral valve regurgitation**) is the most likely cause of the patient's new **holosystolic murmur**.

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Educational objective:
Interstitial myocardial granulomas (Aschoff bodies) are found in carditis due to acute rheumatic fever, which develops after an untreated group A streptococcal pharyngeal infection. Aschoff bodies contain plump macrophages with abundant cytoplasm and central, slender ribbons of chromatin (Anitschkow, or caterpillar, cells).

References

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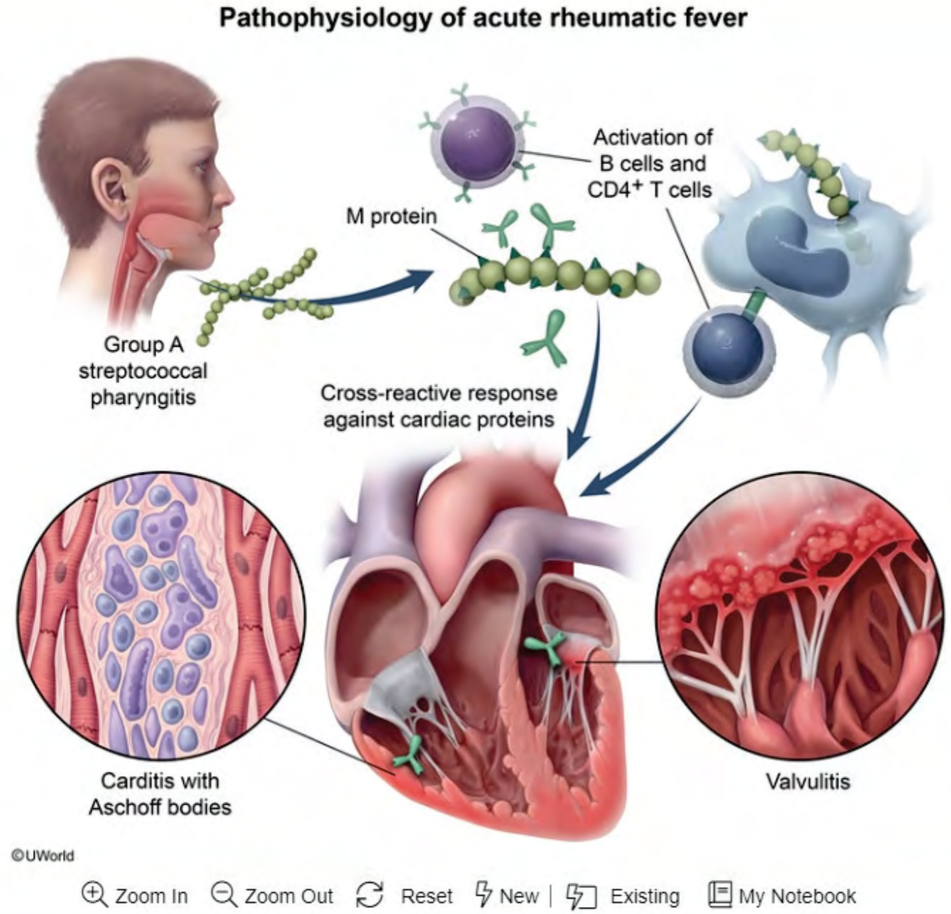

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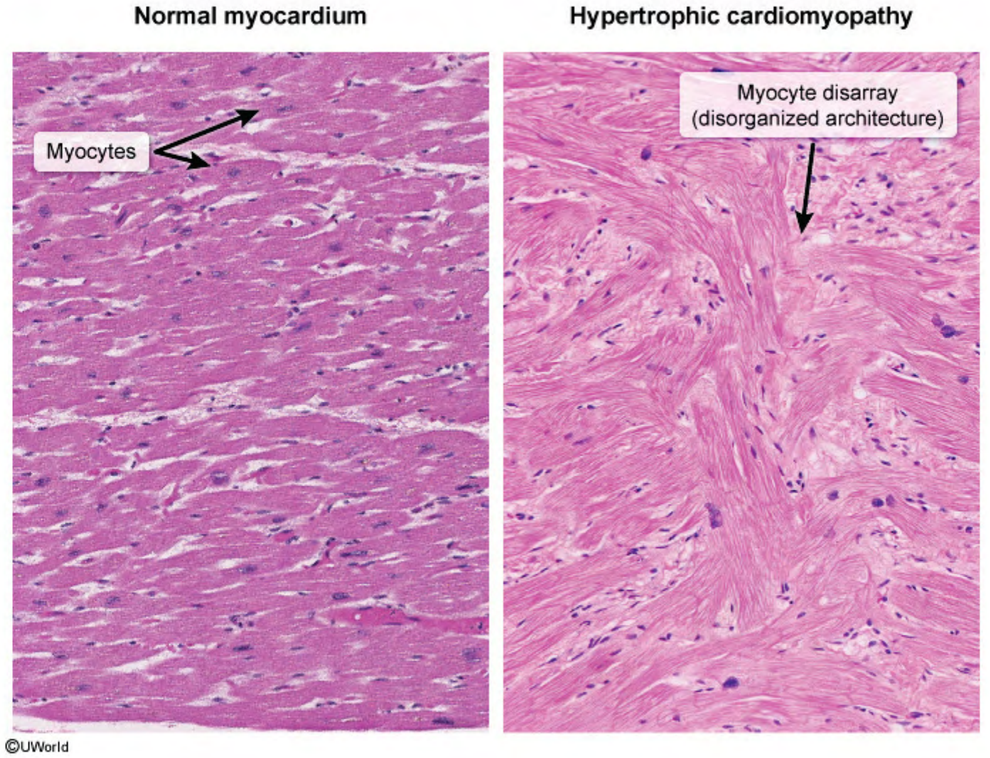

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Educational objective:
Interstitial myocardial granulomas (Aschoff bodies) are found in carditis due to acute rheumatic fever, which

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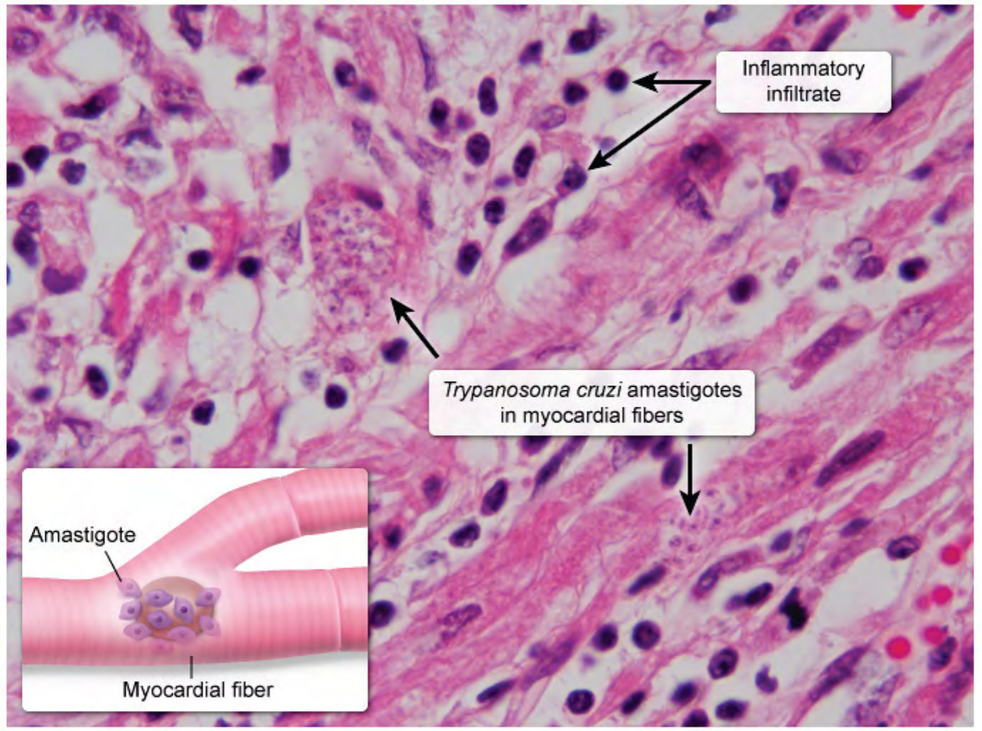
Chagas disease



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involvement resulting in valvular dysfunction (specifically acute **mitral valve regurgitation**) is the most likely cause of the patient's new **holosystolic murmur**.

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References

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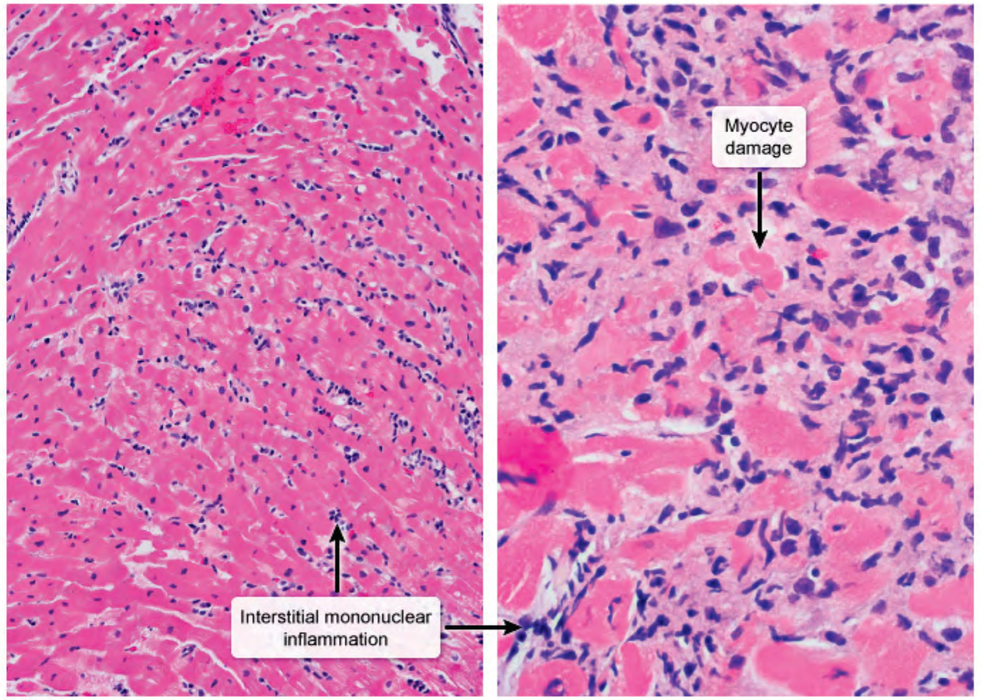
Myocarditis



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A 1-day-old infant in the newborn nursery is found to have a harsh, holosystolic murmur on physical examination. The neonate was born at 39 weeks gestation to a 36-year-old woman who opted to defer prenatal screening. Review of medical records shows no family history of genetic or chromosomal disorders. The infant's vital signs are appropriate for his age. The rest of the physical examination shows a flat facial profile, protruding tongue, and small ears. Which of the following most likely occurred prior to conception?

- A. Chromosomal deletion
- B. Chromosomal translocation
- C. Gene inactivation
- D. Meiotic nondisjunction
- E. Trinucleotide repeat expansion

Submit

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- ✗ A. Chromosomal deletion (5%)
- B. Chromosomal translocation (5%)
- C. Gene inactivation (1%)
- ✓ D. Meiotic nondisjunction (84%)
- E. Trinucleotide repeat expansion (3%)

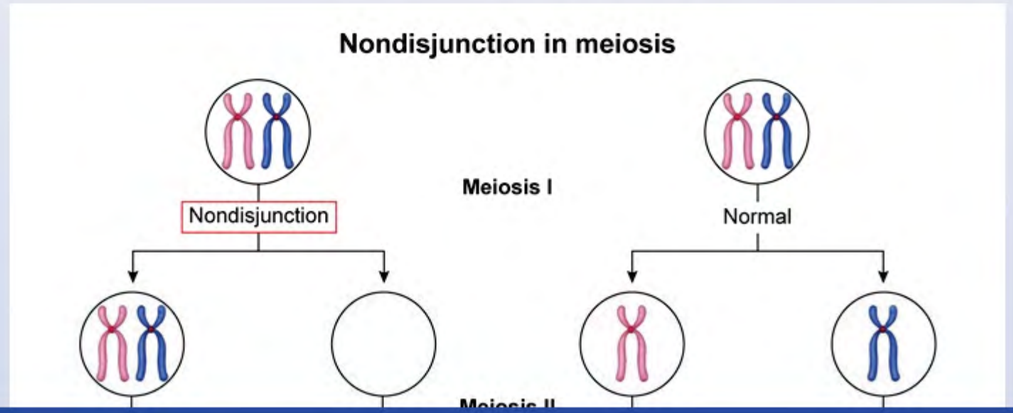
Incorrect
Correct answer
D

84%
Answered correctly

02 mins, 09 secs
Time Spent

2023
Version

Explanation



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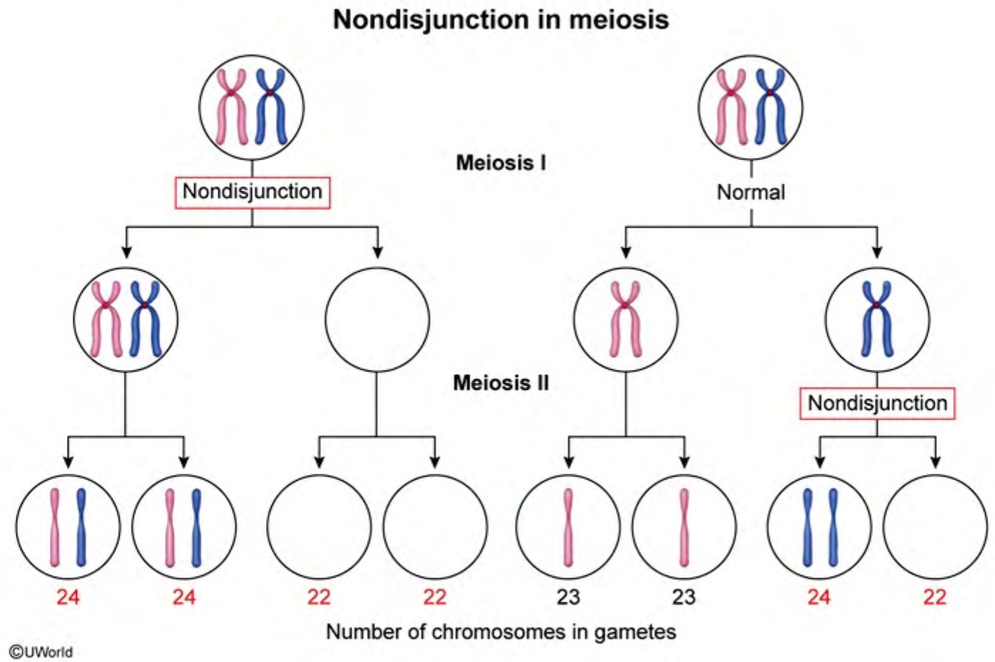

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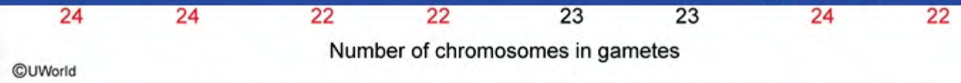

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During normal meiosis, homologous chromosomes separate to pass on individual chromatids to daughter cells. In **meiotic nondisjunction**, the chromosomes fail to separate, allowing one or more daughter cells to pass on with an **extra copy** of a chromosome. Increasing maternal age, particularly **age ≥35**, is associated with an elevated risk of nondisjunction that is likely due to cumulative oxidative stress, depletion of available mature oocytes, and shortening of oocyte telomeres. From highest to lowest prevalence, the most common consequences of maternal nondisjunction are **trisomies 21** (Down syndrome), 18 (Edwards syndrome), and 13 (Patau syndrome).

Less than 5% of Down syndrome cases are caused by a Robertsonian translocation **(Choice B)**, which results from a break near the centromeres of 2 chromosomes (eg, chromosomes 14 and 21) and subsequent transfer of genetic material between these chromosomes. This translocation results in 46 chromosomes in the fetus with 3 effective copies of chromosome 21.

Classic features of Down syndrome include a flat facial profile, epicanthal folds, upslanting palpebral fissures, protruding tongue, and small ears. Congenital heart defects (eg, complete atrioventricular defect, ventricular or atrial septal defect) are seen in 50% of patients.

(Choice A) Chromosomal deletions are associated with cri-du-chat syndrome (5p deletion), DiGeorge syndrome (22q11 microdeletion), and Prader-Willi syndrome (15q deletion). Deletions are not associated with increased maternal age.

(Choice C) Imprinting is the selective inactivation of a maternal or paternal allele. When a selectively expressed allele is abnormal (eg, gene deletion), conditions such as Prader-Willi syndrome (maternal inactivation/paternal deletion) or Angelman syndrome (paternal inactivation/maternal deletion) can result.

(Choice E) Trinucleotide repeat expansions within DNA can dysregulate protein production, causing disorders such as Huntington disease, myotonic dystrophy, and fragile X syndrome.

Educational objective:
Down syndrome is most commonly caused by maternal meiotic nondisjunction, a process by which the fetus receives 3 full copies of chromosome 21. Dysmorphic features (eg, flat facial profile, protruding tongue, small ears, upslanting palpebral fissures) and cardiac defects (eg, endocardial cushion defects) are characteristic.

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References

- Maternal age and risk for trisomy 21 assessed by the origin of chromosome nondisjunction: a report from the Atlanta and National Down Syndrome Projects

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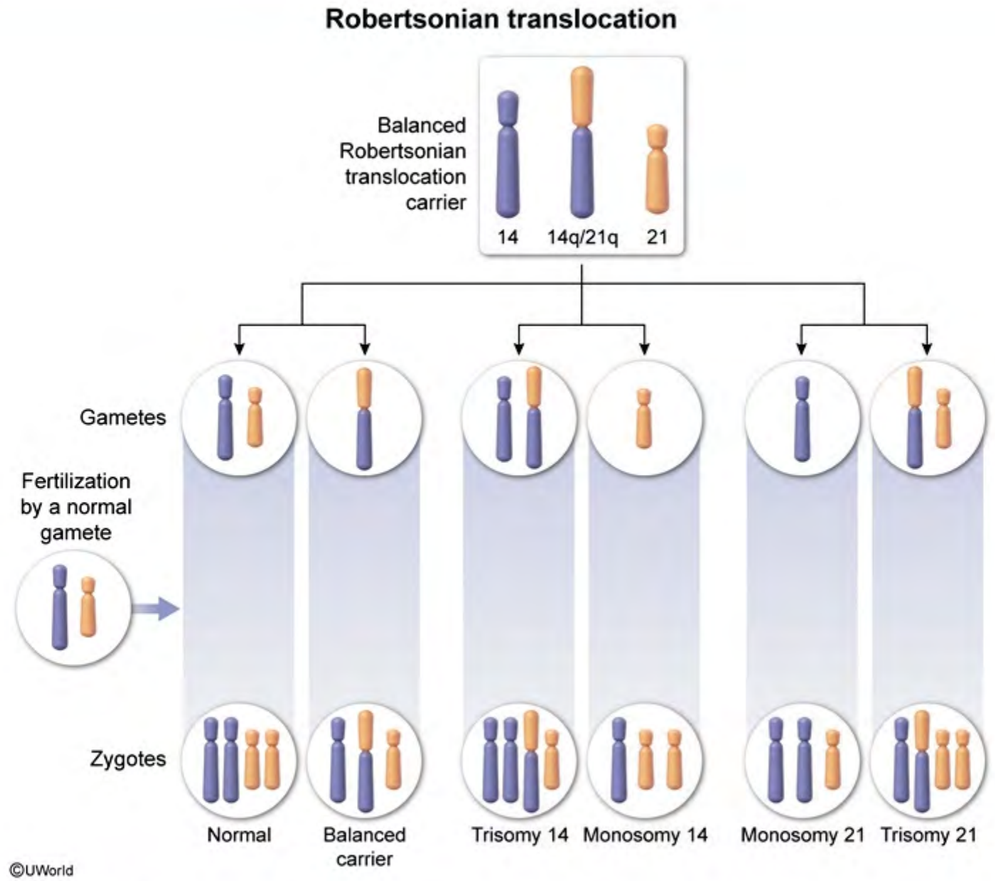

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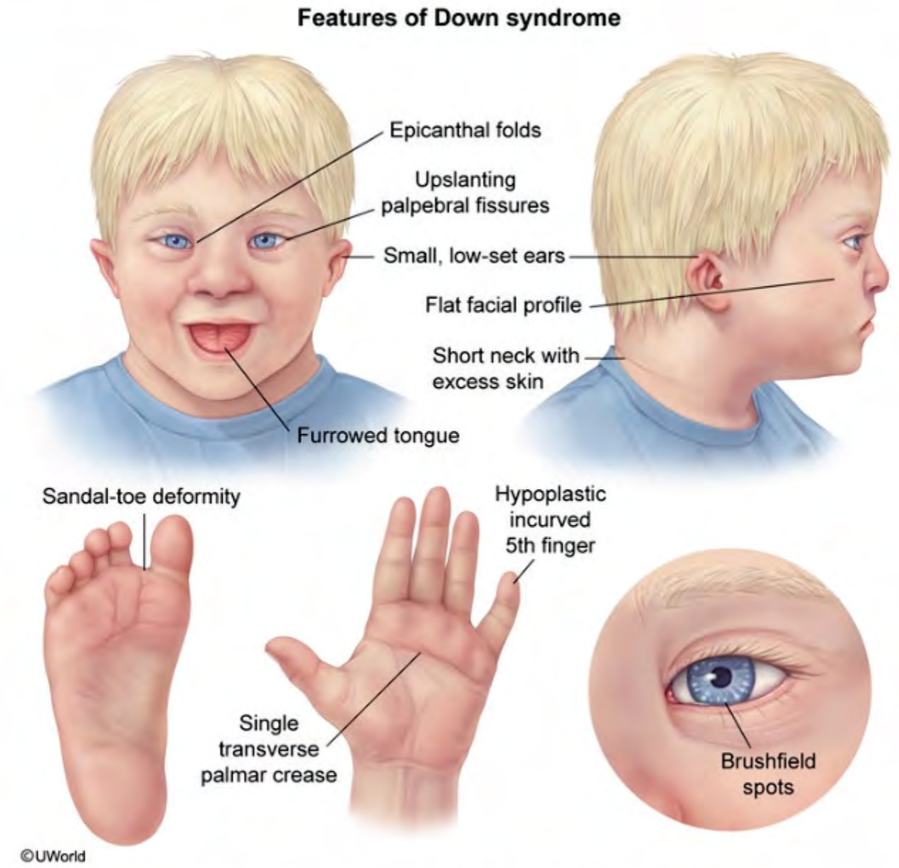

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(Choice E) Trinucleotide repeat expansions within DNA can dysregulate protein production, causing disorders such as Huntington disease, myotonic dystrophy, and fragile X syndrome.

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An 8-hour-old boy is being assessed for a murmur in the newborn nursery. The patient was born at 37 weeks via cesarean delivery. Apgar scores were 7 and 8 at 1 and 5 minutes, respectively. He was noted to have a harsh systolic ejection murmur of 4/6 intensity during the initial newborn examination. Brachial and femoral pulses are 2+ with no delay. While the patient is breastfeeding for the first time, his face, lips, hands, and feet become cyanotic. The murmur cannot be heard during the cyanotic episode. Which of the following oxygen saturation percentages are most likely present during the cyanotic episode?

	Right atrium	Right ventricle	Pulmonary artery	Pulmonary veins	Left atrium	Left ventricle
○ A.	75	75	75	98	98	80
○ B.	75	75	75	98	80	80
○ C.	75	85	85	98	98	98
○ D.	75	75	80	98	98	98

Submit

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	Right atrium	Right ventricle	Pulmonary artery	Pulmonary veins	Left atrium	Left ventricle
✓ A.	75	75	75	98	98	80
B.	75	75	75	98	80	80
C.	75	85	85	98	98	98
D.	75	75	80	98	98	98

Correct

Collecting Statistics

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Time Spent

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Explanation

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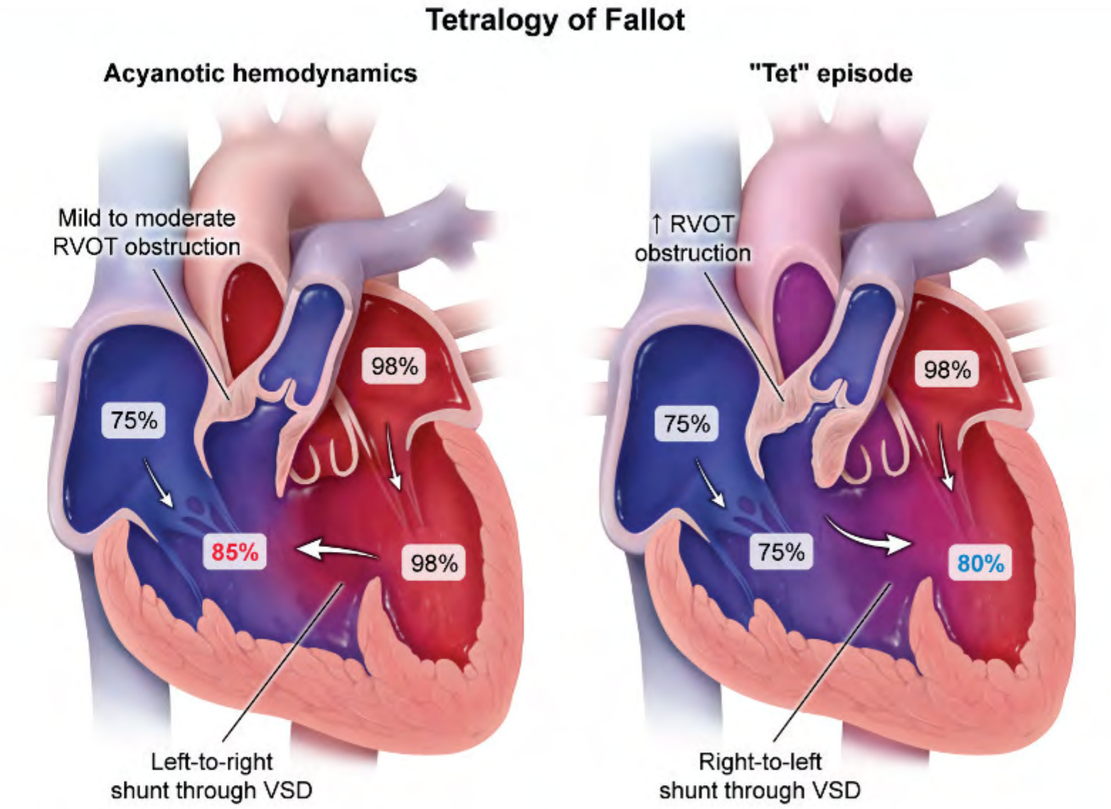

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dynamic and can change based on activity (eg, feeding, squatting) that alters the underlying hemodynamics. Many

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RVOT = right ventricular outflow tract; **VSD** = ventricular septal defect.

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This patient has a harsh **systolic ejection murmur** that disappears during an episode of **cyanosis**, a characteristic finding with **tetralogy of Fallot** (ToF). The major defects in ToF are right ventricular outflow tract (RVOT) obstruction and ventricular septal defect (VSD); the murmur is created by blood flow through the narrowed RVOT, not the VSD.

The degree of RVOT obstruction is the primary driver of symptoms in ToF because it determines the direction and magnitude of shunting through the VSD. A certain degree of RVOT obstruction is fixed, but some degree is dynamic and can change based on activity (eg, feeding, squatting) that alters the underlying hemodynamics. Many patients have left-to-right or neutral shunting at rest (and are acyanotic) but develop cyanosis **("tet" episodes)** when a **dynamic increase in RVOT obstruction** (eg, precipitated by crying or feeding) leads to **increased right-to-left shunting** through the VSD.

During such an episode, decreased blood flow through the RVOT causes the systolic murmur to disappear. In addition, oxygen saturation **(SaO_2) in the left ventricle is reduced** (eg, <90%) due to mixing with deoxygenated blood from the RV. The SaO_2 in the left atrium (LA) remains normal as the LA continues to receive oxygenated blood from the pulmonary veins.

(Choice B) These values show a step-down in SaO_2 at the LA (pulmonary vein SaO_2 > LA SaO_2), consistent with a right-to-left shunt through an atrial septal defect (ASD). Right-to-left shunting through an ASD (ie, Eisenmenger syndrome) takes years to develop and is not an expected cause of episodic cyanosis in infants.

(Choice C) These values show a step-up in SaO_2 at the RV (RV SaO_2 > right atrium SaO_2), consistent with left-to-right shunting through a VSD, as occurs during acyanotic periods in patients with ToF.

(Choice D) These values show a step-up in SaO_2 at the pulmonary artery (pulmonary artery SaO_2 > RV SaO_2), consistent with left-to-right shunting through a patent ductus arteriosus. Left-to-right shunting does not cause cyanosis.

Educational objective:
The major defects in tetralogy of Fallot are right ventricular outflow tract (RVOT) obstruction and ventricular septal defect (VSD). Cyanotic ("tet") episodes are commonly precipitated by a dynamic rise in RVOT obstruction that

https://t.me/USMLEWorldStep1

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Educational objective:
The major defects in tetralogy of Fallot are right ventricular outflow tract (RVOT) obstruction and ventricular septal defect (VSD). Cyanotic ("tet") episodes are commonly precipitated by a dynamic rise in RVOT obstruction that increases right-to-left shunting through the VSD. These episodes involve decreased blood flow through the RVOT, causing disappearance of the associated systolic murmur and reduced oxygen saturation of left ventricular blood.

References

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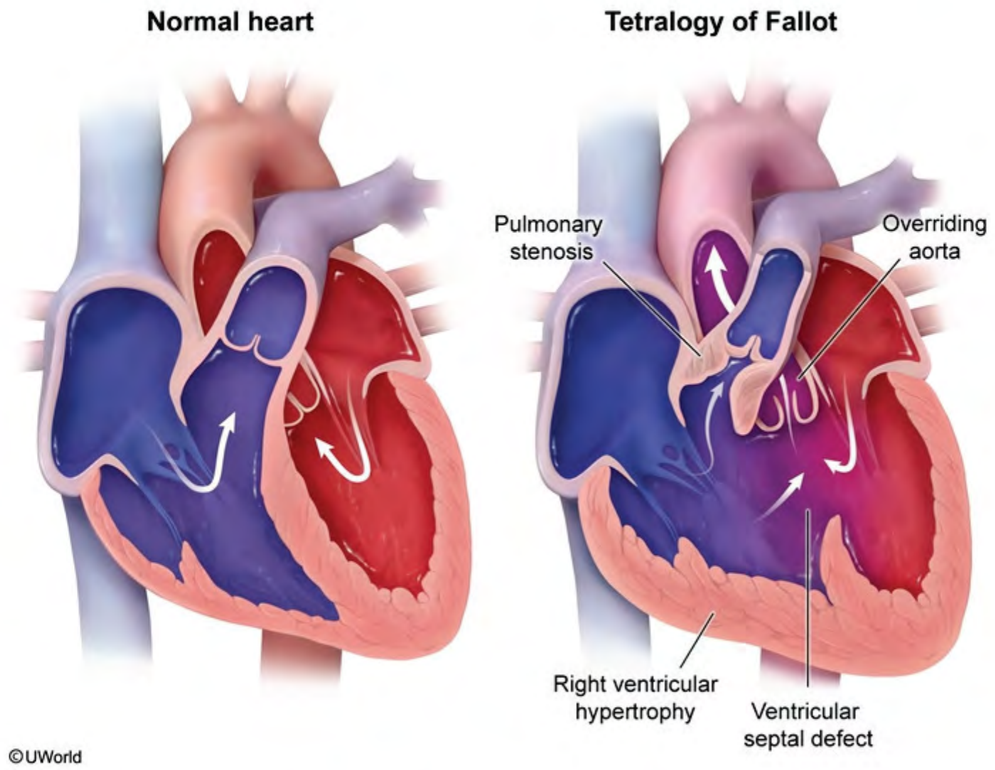

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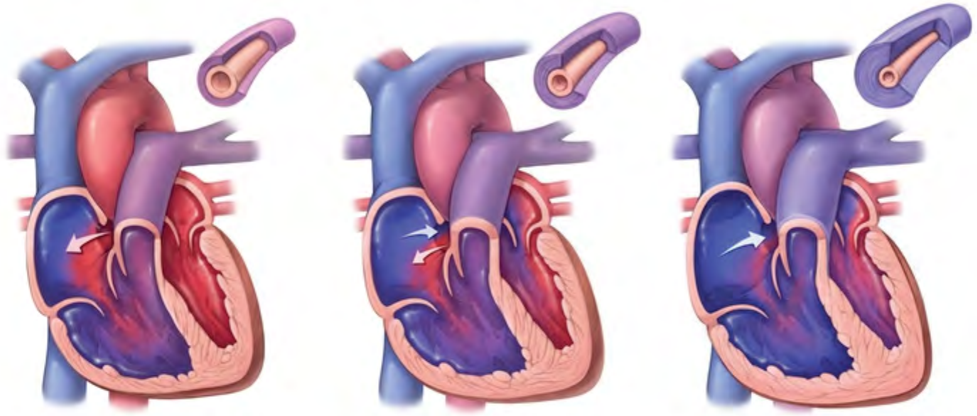
Eisenmenger syndrome



Congenital defect (eg, ASD) allows left-to-right shunt

Over time, ↑ PVR leads to bidirectional flow

PVR continues to increase with eventual continuous right-to-left shunt (Eisenmenger syndrome)

ASD = atrial septal defect; **PVR** = pulmonary vascular resistance.

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Educational objective:

The major defects in tetralogy of Fallot are right ventricular outflow tract (RVOT) obstruction and ventricular septal defect (VSD). Cyanotic ("tet") episodes are commonly precipitated by a dynamic rise in RVOT obstruction that

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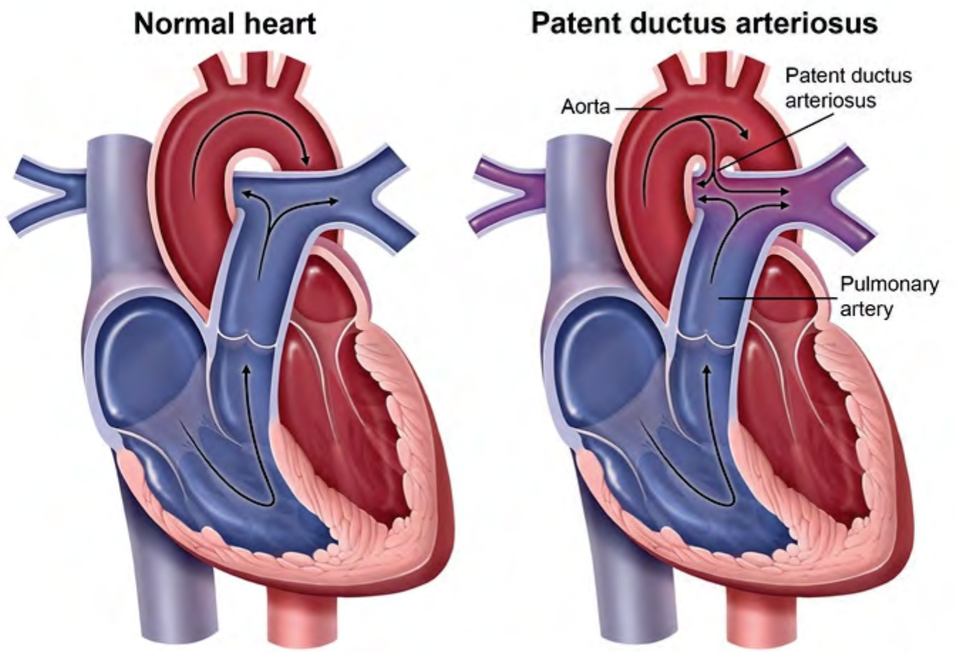

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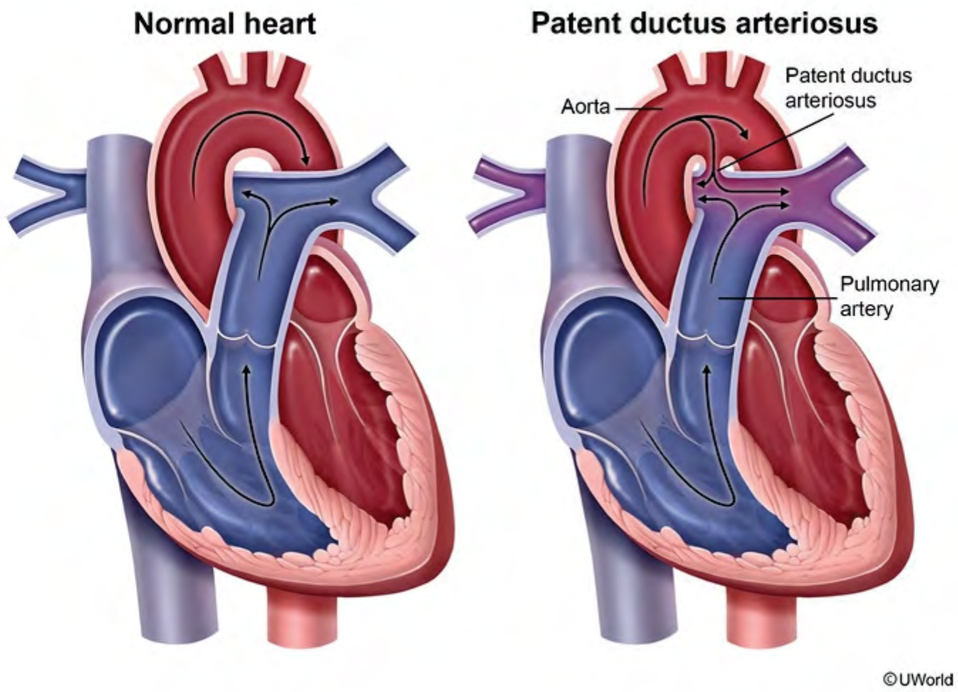

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A 4-year-old boy is being evaluated for failure to thrive, shortness of breath, and exercise intolerance. The parents first became aware of his symptoms after the patient started preschool a few weeks ago. His teacher mentioned that the patient seemed to tire faster than the other children when playing outside. Cardiac examination shows bounding peripheral pulses and a palpable thrill below the clavicle near the left upper sternal border. On auscultation, a continuous murmur is best heard over the same region. After discussion with the parents, a thoracotomy is scheduled to correct the patient's condition. During the procedure, the surgeon should plan on ligating a derivative of which of the following embryologic structures?

- A. Bulbus cordis
- B. Fourth aortic arch
- C. Primitive atria
- D. Sinus venosus
- E. Sixth aortic arch

Submit

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- ✗ A. Bulbus cordis (10%)
- B. Fourth aortic arch (28%)
- C. Primitive atria (5%)
- D. Sinus venosus (10%)
- ✓ E. Sixth aortic arch (45%)

Incorrect
Correct answer
E

45%
Answered correctly

09 mins, 28 secs
Time Spent

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Explanation

Aortic arch vascular derivatives	
Aortic arch	**Adult derivative**
First	• Part of maxillary artery
Second	• Hyoid artery • Stapedial artery
	• Common carotid artery

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Aortic arch vascular derivatives	
Aortic arch	**Adult derivative**
First	• Part of maxillary artery
Second	• Hyoid artery • Stapedial artery
Third	• Common carotid artery • Proximal internal carotid artery
Fourth	• On left → aortic arch • On right → proximal right subclavian artery
Sixth	• Proximal pulmonary arteries • On left → ductus arteriosus

This patient most likely has a **patent ductus arteriosus (PDA)**. The ductus arteriosus is an embryonic derivative of the **sixth aortic arch** that allows fetal blood to pass directly from the pulmonary artery to the proximal descending aorta (bypassing the pulmonary circulation). This vessel usually closes shortly after birth, secondary to decreased prostaglandin E_2 (PGE_2) levels and increased oxygen concentration. Patency of the ductus after birth results in a **left-to-right shunt** that can cause left ventricular volume overload and symptoms of **heart failure** (eg, failure to thrive, respiratory distress). Physical examination will show a **continuous "machinery-like" murmur** and palpable thrill over the **left infraclavicular region** due to turbulent blood flow through the PDA.

Pharmacologic closure of a PDA can be achieved by **PGE_2 synthesis inhibitors** (eg, indomethacin) in premature infants. However, older patients usually require **surgical ligation** or percutaneous PDA occlusion.

(Choice A) The bulbus cordis forms the beginning of the ventricular outflow tract in the embryonic heart. This structure forms the smooth portions of the left and right ventricles adjacent to the aorta and pulmonary artery, respectively.

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Sixth	• Proximal pulmonary arteries • On left → ductus arteriosus

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(Choice A) The bulbus cordis forms the beginning of the ventricular outflow tract in the embryonic heart. This structure forms the smooth portions of the left and right ventricles adjacent to the aorta and pulmonary artery, respectively.

(Choice B) The fourth aortic arch on the left forms the arch of the aorta between the left carotid artery and ductus arteriosus. Abnormalities during development can result in coarctation (narrowing) of the aorta, which can present with continuous murmurs over the back due to collateral formation.

(Choice C) The primitive atrium receives blood from the sinus venosus in the embryonic heart and transmits it to the primitive ventricle. The primitive atrium forms the rough portions of the left and right atria.

(Choice D) The sinus venosus is an embryologic structure within the heart that receives blood from the vena cava. In adults, this structure forms the smooth portion of the right atrium, known as the sinus venarum.

Educational objective:
The ductus arteriosus is derived from the sixth embryonic aortic arch. A patent ductus arteriosus (PDA) causes left-to-right shunting of blood that can be heard as a continuous murmur over the left infraclavicular region. Indomethacin (a PGE_2 synthesis inhibitor) can be used to close a PDA in premature infants, but surgical ligation is often necessary in older patients.

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of the **sixth aortic arch** that allows fetal blood to pass directly from the pulmonary artery to the proximal descending aorta (bypassing the pulmonary circulation). This vessel usually closes shortly after birth, secondary to decreased prostaglandin E_2 (PGE_2) levels and increased oxygen concentration. Patency of the ductus after birth results in a **left-to-right shunt** that can cause left ventricular volume overload and symptoms of **heart failure** (eg, failure to thrive, respiratory distress). Physical examination will show a **continuous "machinery-like" murmur** and palpable thrill over the **left infraclavicular region** due to turbulent blood flow through the PDA.

Pharmacologic closure of a PDA can be achieved by **PGE_2 synthesis inhibitors** (eg, indomethacin) in premature infants. However, older patients usually require **surgical ligation** or percutaneous PDA occlusion.

(Choice A) The bulbus cordis forms the beginning of the ventricular outflow tract in the embryonic heart. This structure forms the smooth portions of the left and right ventricles adjacent to the aorta and pulmonary artery, respectively.

(Choice B) The fourth aortic arch on the left forms the arch of the aorta between the left carotid artery and ductus arteriosus. Abnormalities during development can result in coarctation (narrowing) of the aorta, which can present with continuous murmurs over the back due to collateral formation.

(Choice C) The primitive atrium receives blood from the sinus venosus in the embryonic heart and transmits it to the primitive ventricle. The primitive atrium forms the rough portions of the left and right atria.

(Choice D) The sinus venosus is an embryologic structure within the heart that receives blood from the vena cava. In adults, this structure forms the smooth portion of the right atrium, known as the sinus venarum.

Educational objective:
The ductus arteriosus is derived from the sixth embryonic aortic arch. A patent ductus arteriosus (PDA) causes left-to-right shunting of blood that can be heard as a continuous murmur over the left infraclavicular region. Indomethacin (a PGE_2 synthesis inhibitor) can be used to close a PDA in premature infants, but surgical ligation is often necessary in older patients.

Anatomy	Cardiovascular System	Patent ductus arteriosus
Subject	System	Topic

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An 81-year-old woman undergoes transcatheter aortic valve implantation through femoral access. She was diagnosed with severe calcific aortic stenosis 2 months ago after she began having shortness of breath with mild exertion. The vascular access is gained without complication, and the transcatheter valve is deployed under fluoroscopic guidance. The following hemodynamic changes are observed:

	Before deployment	After deployment
Diastolic blood pressure	76 mm Hg	44 mm Hg
Left ventricular end-diastolic pressure	12 mm Hg	25 mm Hg

Which of the following complications should be suspected in this patient?

- A. Aortic regurgitation
- B. Mitral stenosis
- C. Prosthetic valve thrombosis
- D. Retroperitoneal bleeding
- E. Ventricular septal perforation

Submit

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	Before deployment	After deployment
Diastolic blood pressure	76 mm Hg	44 mm Hg
Left ventricular end-diastolic pressure	12 mm Hg	25 mm Hg

Which of the following complications should be suspected in this patient?

- ✓ A. Aortic regurgitation (65%)
- ✗ B. Mitral stenosis (6%)
- C. Prosthetic valve thrombosis (14%)
- D. Retroperitoneal bleeding (5%)
- E. Ventricular septal perforation (7%)

Incorrect
Correct answer
A

65%
Answered correctly

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Explanation

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An 81-year-old woman undergoes transcatheter aortic valve implantation through femoral access. She was
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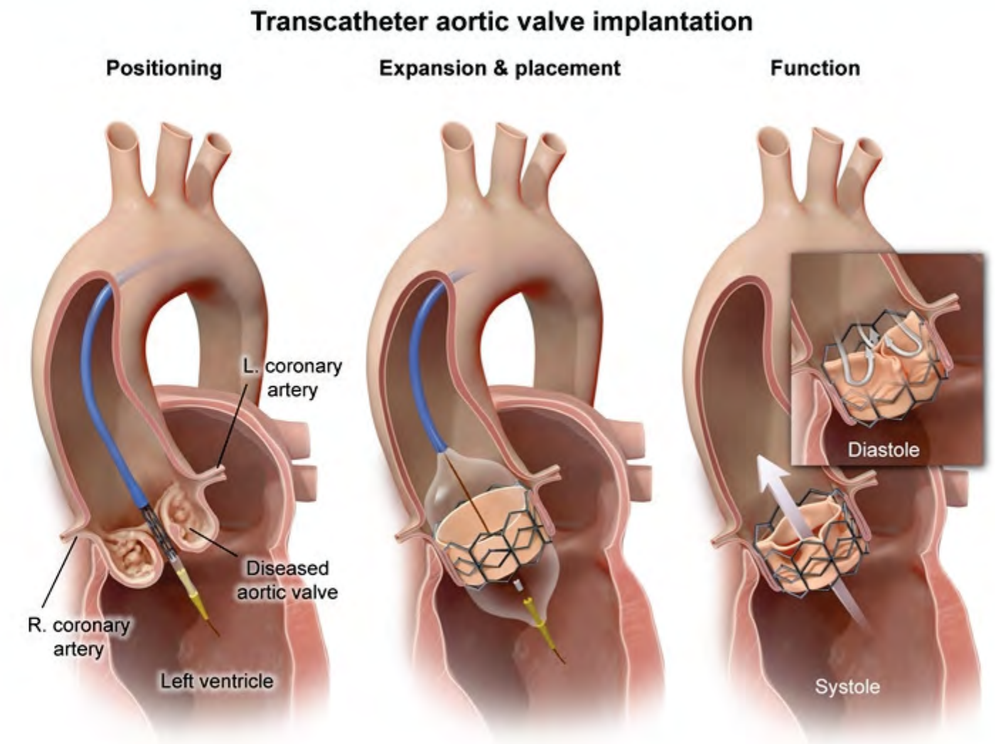

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The prevalence of **severe aortic stenosis** (AS) increases with age, and definitive management necessitates surgical correction of the stenotic valve. This often creates a complicated scenario because many **elderly patients** with severe AS have multiple other comorbidities, making them poor candidates for open surgical valve replacement due to unacceptably high operative risk. **Transcatheter aortic valve implantation** (TAVI) provides an alternative option for such patients; the catheter is advanced through the aorta to the aortic valve where a bioprosthetic valve is placed over the native aortic valve leaflets.

This patient's intraoperative pressure readings of **decreased diastolic blood pressure** and **increased LVEDP** (left ventricular end-diastolic pressure) are consistent with **aortic regurgitation**, a common complication of TAVI that results from improper sealing of the prosthetic valve to the native valve annulus, leading to paravalvular leak (blood leakage around the valve). Inappropriate valve size or improper valve positioning is often responsible.

Other intraoperative complications of TAVI include stroke (due to embolization of native valve calcification) and myocardial infarction (due to obstruction of coronary artery ostia by the implanted valve).

(Choice B) Worsening mitral stenosis can rarely occur following TAVI; such a disruption is expected to cause a decrease (rather than increase) in LVEDP due to obstructed diastolic filling.

(Choice C) Prosthetic valve thrombosis usually causes valvular obstruction but may sometimes cause valvular regurgitation with similar pressure findings to this patient. However, valvular thrombus requires time to develop and is not typically an immediate intraoperative complication.

(Choice D) Retroperitoneal bleeding can result from extension of a femoral artery hematoma and is a potential complication of any catheter-based procedure with femoral arterial access. The complication is likely to cause hemorrhagic shock with a drop in both systemic blood pressure and LVEDP.

(Choice E) Ventricular septal perforation is a potential complication of TAVI but is unlikely to cause the large and abrupt increase in LVEDP that occurred in this patient. Large perforation may eventually lead to biventricular failure and increased LVEDP, but no change or a small decrease in LVEDP is expected initially.

Educational objective:
Transcatheter aortic valve implantation (TAVI) allows for minimally invasive management of severe aortic stenosis in elderly patients who are unable to tolerate open surgical valve replacement. Paravalvular aortic regurgitation is

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replacement due to unacceptably high operative risk. **Transcatheter aortic valve implantation** (TAVI) provides an alternative option for such patients; the catheter is advanced through the aorta to the aortic valve where a bioprosthetic valve is placed over the native aortic valve leaflets.

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Educational objective:
Transcatheter aortic valve implantation (TAVI) allows for minimally invasive management of severe aortic stenosis in elderly patients who are unable to tolerate open surgical valve replacement. Paravalvular aortic regurgitation is a common complication of TAVI, resulting from improper sealing of the prosthetic valve to the native aortic valve annulus.

References

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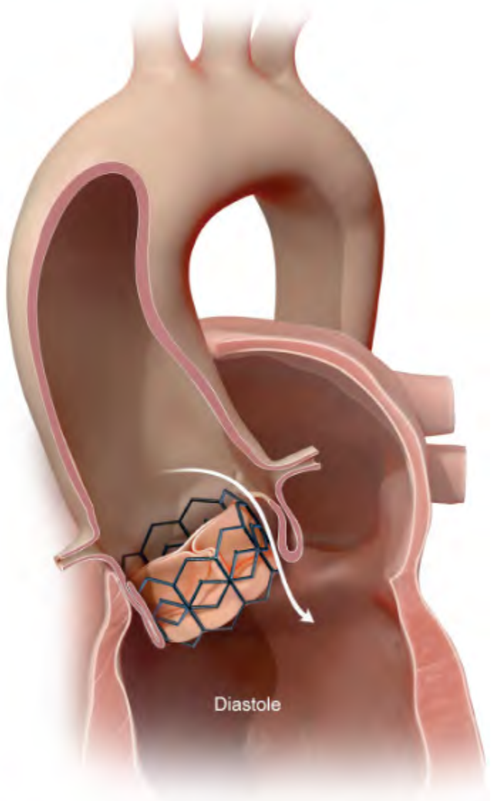
Aortic regurgitation due to paravalvular leak



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A 37-year-old woman is brought to the emergency department after developing sudden-onset right arm weakness and difficulty speaking. She has no significant medical history but takes oral contraceptives. Examination shows decreased right upper extremity strength and expressive aphasia. Cardiac auscultation is normal. MRI of the brain reveals acute infarction in the left frontal lobe. Further evaluation to determine an etiology is performed. During echocardiogram, agitated normal saline is injected into a peripheral vein and bubbles are seen passing into the left side of the heart. Which of the following is the most likely cause of the observed finding in this patient?

- A. Aplasia of the atrial septum secundum
- B. Failure of the aorticopulmonary septum to develop
- C. Incomplete closure of the interventricular foramen
- D. Incomplete fusion of atrial septum primum and secundum
- E. Persistent channel between aorta and pulmonary artery

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- ✗ A. Aplasia of the atrial septum secundum (9%)
- B. Failure of the aorticopulmonary septum to develop (3%)
- C. Incomplete closure of the interventricular foramen (16%)
- ✓ D. Incomplete fusion of atrial septum primum and secundum (66%)
- E. Persistent channel between aorta and pulmonary artery (4%)

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Correct answer
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66%
Answered correctly

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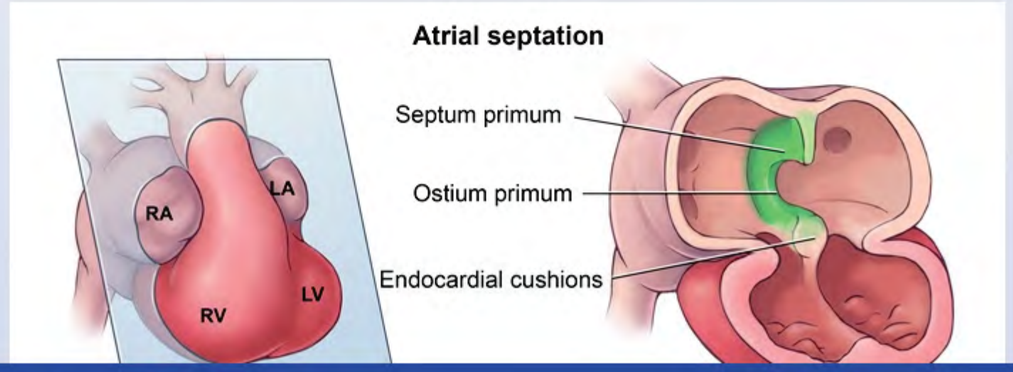

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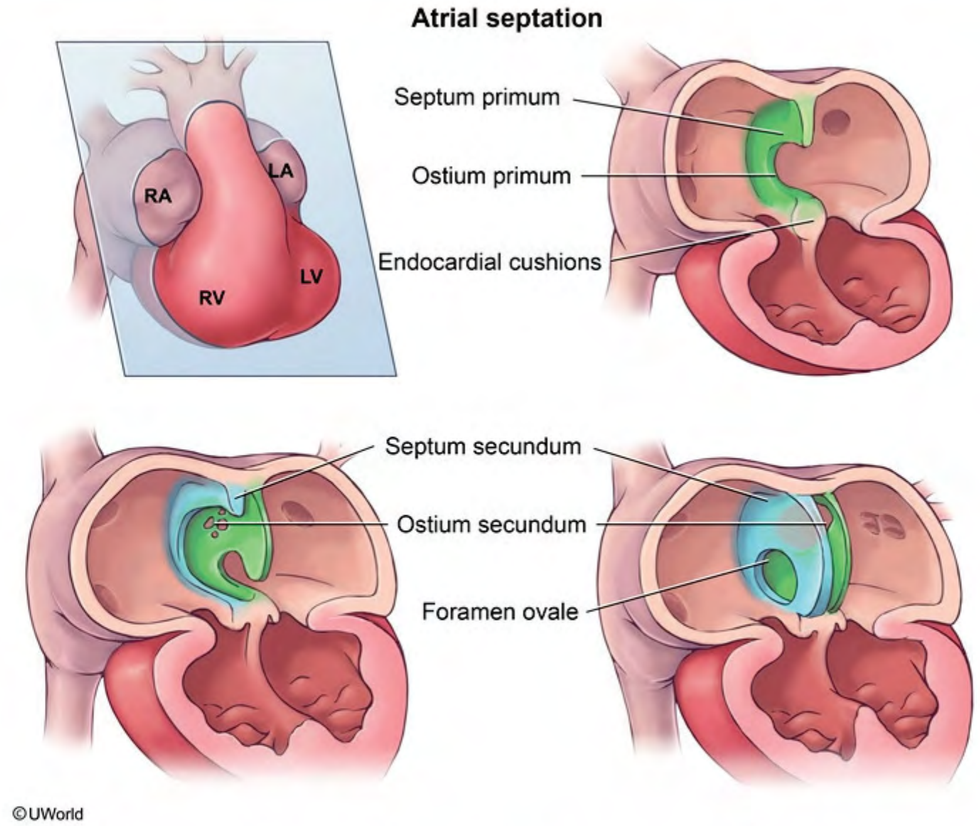

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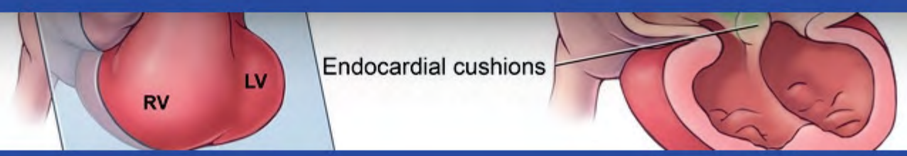

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This young patient had a **cryptogenic stroke**, which is evaluated with echocardiography with a "bubble study" to identify right-to-left intracardiac shunts. This study is performed by injecting agitated normal saline intravenously and observing for microbubbles in the left heart. Shunt detection is improved by having the patient release a sustained Valsalva maneuver, which increases right atrial pressure. Cryptogenic stroke is frequently associated with atrial septal abnormalities such as **patent foramen ovale** (PFO) and atrial septal defect (ASD).

During fetal development, the foramen ovale shunts oxygenated fetal blood across from the right to the left atrium. After delivery, umbilical cord clamping and decreased pulmonary vascular resistance lower right atrial pressure and raise left atrial pressure. This pushes the **septum primum** against the **septum secundum**, closing the foramen ovale. Over time, fibrosis and tissue remodeling fuse the flap closed. **Incomplete fusion** occurs in approximately 25% of normal adults, resulting in a PFO.

PFO usually remains functionally closed and asymptomatic after birth as left atrial pressure is greater than right atrial pressure. However, conditions that raise right atrial pressure above left atrial pressure (eg, Valsalva release) produce a transient right-to-left shunt across the PFO. This is particularly concerning in patients with hypercoagulability (eg, oral contraceptive use) as they are at increased risk for venous thrombosis and **paradoxical brain embolism**. Chronically elevated right heart pressure (eg, Eisenmenger syndrome) is not necessary for paradoxical embolism.

(Choice A) ASD results from aplasia of the septum primum or septum secundum during development. Although ASD may lead to paradoxical embolism, it is much less common than PFO and characterized by fixed splitting of S2. Unrepaired ASDs may lead to heart failure, pulmonary hypertension, or Eisenmenger syndrome.

(Choice B) Incomplete development of the aorticopulmonary septum results in truncus arteriosus, which causes cyanosis in newborns and requires early surgical repair.

(Choice C) Incomplete closure of the interventricular foramen results in ventricular septal defect (VSD), which causes a holosystolic murmur at the lower left sternal border. Large VSDs can cause significant left-to-right shunting, leading to heart failure in infancy; however, VSDs infrequently lead to paradoxical embolism.

(Choice E) The ductus arteriosus functionally closes within 24 hours after delivery. Persistent patent ductus arteriosus causes a continuous murmur and may eventually result in heart failure, pulmonary hypertension, or Eisenmenger syndrome.

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After delivery, umbilical cord clamping and decreased pulmonary vascular resistance lower right atrial pressure and raise left atrial pressure. This pushes the **septum primum** against the **septum secundum**, closing the foramen ovale. Over time, fibrosis and tissue remodeling fuse the flap closed. **Incomplete fusion** occurs in approximately 25% of normal adults, resulting in a PFO.

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Educational objective:
The foramen ovale is patent in approximately 25% of normal adults. Although the foramen ovale usually remains functionally closed, transient increases of right atrial pressure above left atrial pressure can produce a right-to-left shunt, leading to paradoxical embolism of venous clots into the arterial circulation.

References

- Contemporary approach to paradoxical embolism

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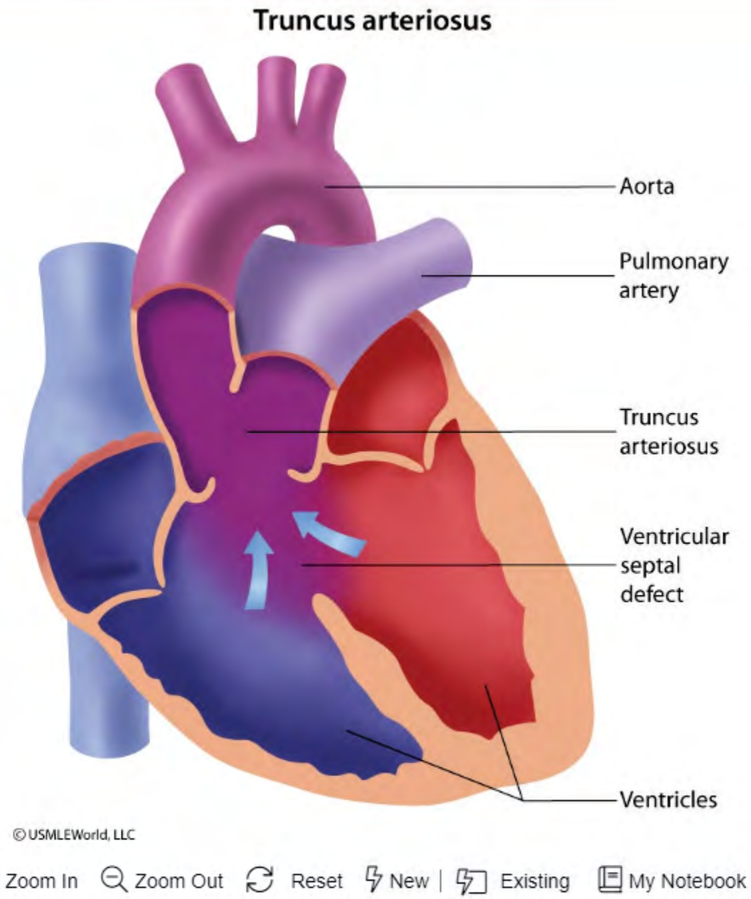

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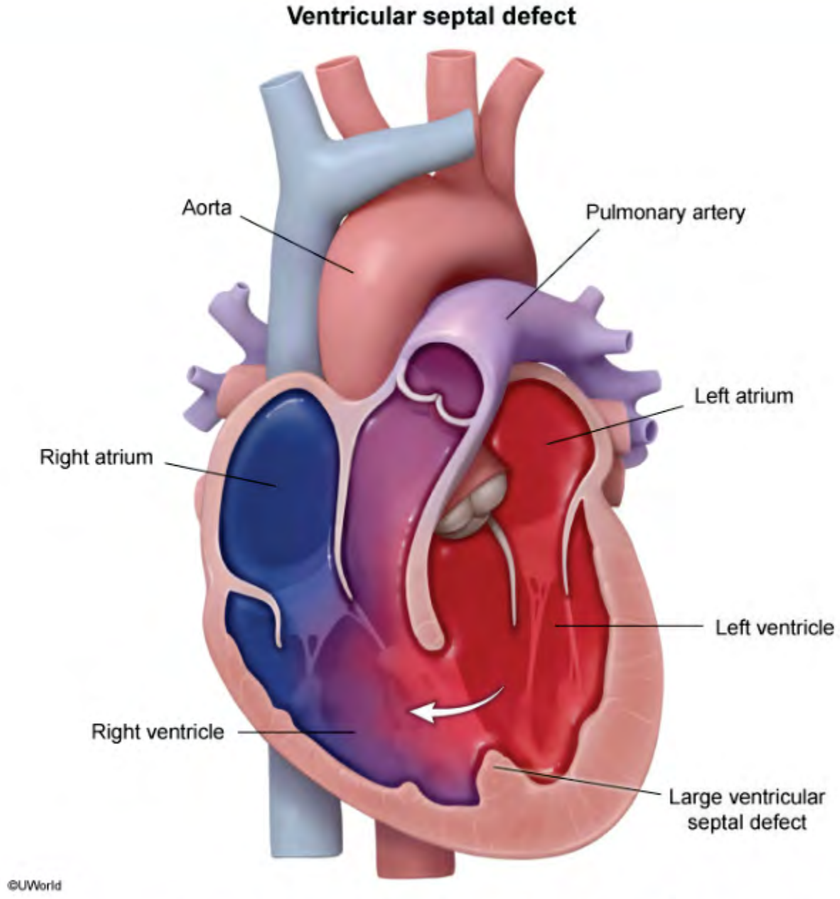

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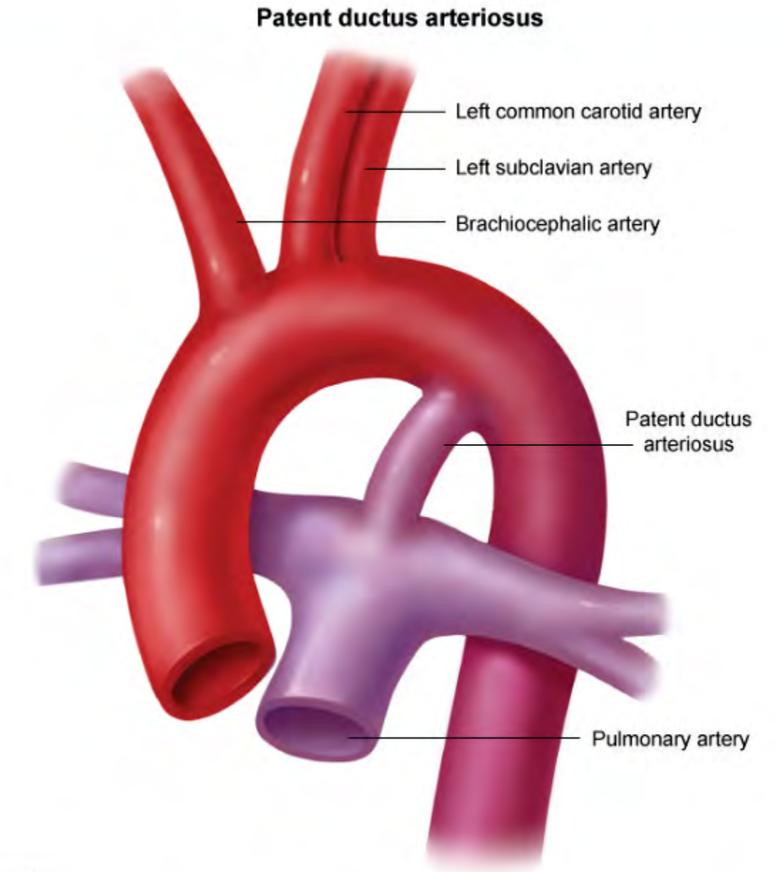

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References

- Contemporary approach to paradoxical embolism

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A 32-year-old man comes to the emergency room due to severe headaches and vomiting. Soon after, he becomes comatose and expires despite extensive resuscitation efforts. Autopsy shows a ruptured cerebral aneurysm with extensive intracranial hemorrhage and a congenital heart defect. This patient's presentation is most likely associated with which of the following abnormalities?

- A. Coarctation of the aorta
- B. Patent ductus arteriosus
- C. Primum-type atrial septal defect
- D. Secundum-type atrial septal defect
- E. Tetralogy of Fallot
- F. Ventricular septal defect

Submit

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A 32-year-old man comes to the emergency room due to severe headaches and vomiting. Soon after, he becomes comatose and expires despite extensive resuscitation efforts. Autopsy shows a ruptured cerebral aneurysm with extensive intracranial hemorrhage and a congenital heart defect. This patient's presentation is most likely associated with which of the following abnormalities?

- ✓ A. Coarctation of the aorta (60%)
- B. Patent ductus arteriosus (4%)
- ✗ C. Primum-type atrial septal defect (8%)
- D. Secundum-type atrial septal defect (14%)
- E. Tetralogy of Fallot (2%)
- F. Ventricular septal defect (9%)

Incorrect
Correct answer
A

60%
Answered correctly

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Time Spent

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Explanation

Coarctation of the aorta	
Pathology	• Thickening of tunica media of aortic arch • Can be associated with Turner syndrome or bicuspid aortic valve

Clinical features	• ↑ BP & strong pulses in upper extremities
	• ↓ BP & weak pulses in lower extremities
	• Neonates (severe narrowing)
	  ○ Differential cyanosis affecting lower extremities*
	  ○ Heart failure & shock after ductal closure

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Clinical features	• ↑ BP & strong pulses in upper extremities
	• ↓ BP & weak pulses in lower extremities
	• Neonates (severe narrowing)
	  ◦ Differential cyanosis affecting lower extremities*
	  ◦ Heart failure & shock after ductal closure
	• Children/adults (mild narrowing)
	  ◦ Lower extremity claudication
	  ◦ Palpable pulsations of intercostal vessels (collaterals)
	  ◦ Secondary hypertension (upper arms & head)

*Patent ductus arteriosus shunts blood into descending aorta. **BP** = blood pressure.	

This patient died from an intracranial hemorrhage due to a ruptured cerebral aneurysm. Hypertension, genetic conditions (eg, Ehlers-Danlos syndrome), and lifestyle factors (eg, cigarette smoking) increase the risk of aneurysm development. **Coarctation of the aorta** is a congenital cardiac defect associated with secondary hypertension and is a **risk factor** for **cerebral aneurysm** development.

Coarctation of the aorta is a narrowing of the aortic arch near the ligamentum arteriosum (remnant of ductus arteriosus) with localized medial and intimal hyperplasia. Critical coarctation (severe narrowing) often presents in the neonatal period with heart failure and cardiogenic shock. However, less severe narrowing allows for distal (but decreased) perfusion to the lower extremities and may present with only asymptomatic **upper extremity hypertension** in children or adults. Additional symptoms due to this differential in blood pressure include headache, epistaxis, chest pain, and lower extremity claudication.

Complications of aortic coarctation include cerebral and aortic aneurysm. Cerebral aneurysm with life-threatening rupture causing **subarachnoid hemorrhage** is likely due to chronic hypertension. Aortic aneurysms usually develop near the site of coarctation due to embryologic vessel wall abnormalities and may lead to dissection or

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(Choices B, C, D, and F) Patent ductus arteriosus and atrial and ventricular septal defects create a left-to-right intracardial shunt that, if untreated, may lead to right-to-left shunting (ie, Eisenmenger syndrome) due to pulmonary hypertension. This may increase risk of a paradoxical thromboembolism from the venous to arterial circulation directly to the brain, causing an ischemic stroke. However, neither condition is associated with cerebral aneurysm or intracranial hemorrhage.

(Choice E) Tetralogy of Fallot is characterized by pulmonary stenosis, ventricular septal defect, an overriding aorta, and right ventricular hypertrophy. Almost all patients have cyanosis in infancy or hypercyanotic episodes (ie, tet spells) in early childhood. This patient's age makes this diagnosis unlikely. In addition, risk of intracranial hemorrhage is not increased with tetralogy of Fallot, regardless of age.

Educational objective:

Adults with coarctation of the aorta often have chronic hypertension and are at increased risk of developing life-

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conditions (eg, Ehlers-Danlos syndrome), and lifestyle factors (eg, cigarette smoking) increase the risk of aneurysm development. **Coarctation of the aorta** is a congenital cardiac defect associated with secondary hypertension and is a **risk factor** for **cerebral aneurysm** development.

Coarctation of the aorta is a narrowing of the aortic arch near the ligamentum arteriosum (remnant of ductus arteriosus) with localized medial and intimal hyperplasia. Critical coarctation (severe narrowing) often presents in the neonatal period with heart failure and cardiogenic shock. However, less severe narrowing allows for distal (but decreased) perfusion to the lower extremities and may present with only asymptomatic **upper extremity hypertension** in children or adults. Additional symptoms due to this differential in blood pressure include headache, epistaxis, chest pain, and lower extremity claudication.

Complications of aortic coarctation include cerebral and aortic aneurysm. Cerebral aneurysm with life-threatening rupture causing **subarachnoid hemorrhage** is likely due to chronic hypertension. Aortic aneurysms usually develop near the site of coarctation due to embryologic vessel wall abnormalities and may lead to dissection or fatal rupture.

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Educational objective:
Adults with coarctation of the aorta often have chronic hypertension and are at increased risk of developing life-threatening aneurysms of the aorta (eg, dissection, rupture) and cerebral vessels (ie, intracranial hemorrhage).

References

- Coarctation of the aorta.

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- Differential cyanosis affecting lower extremities*

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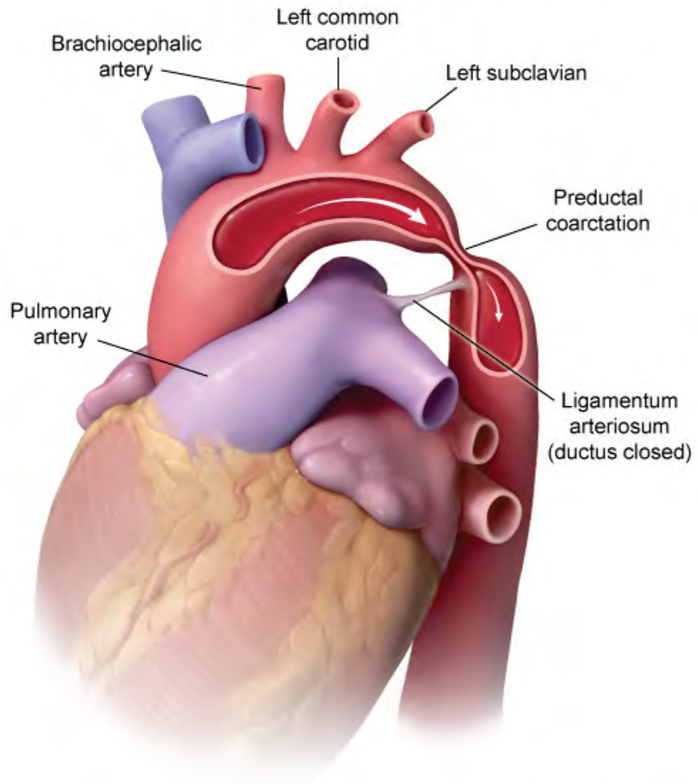
Aortic coarctation, closed ductus



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- Lower extremity claudication
- Palpable pulsations of intercostal vessels (collaterals)

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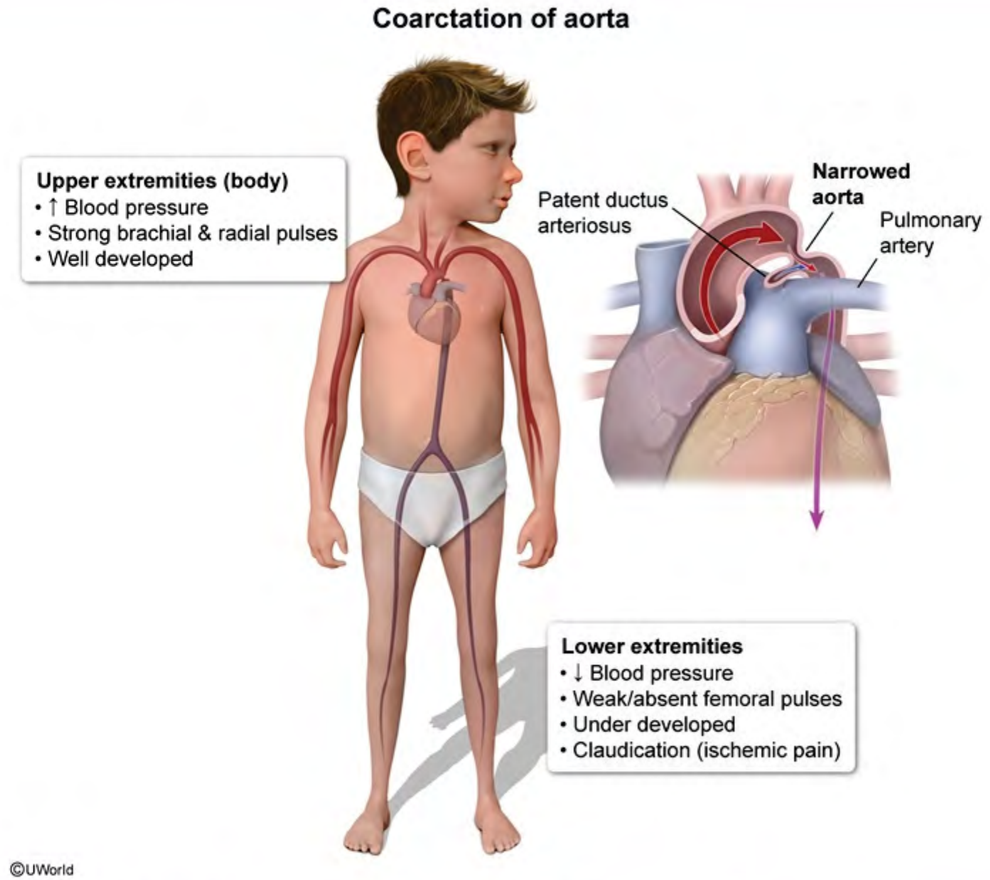

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*Patent ductus arteriosus shunts blood into descending aorta.

BP = blood pressure.

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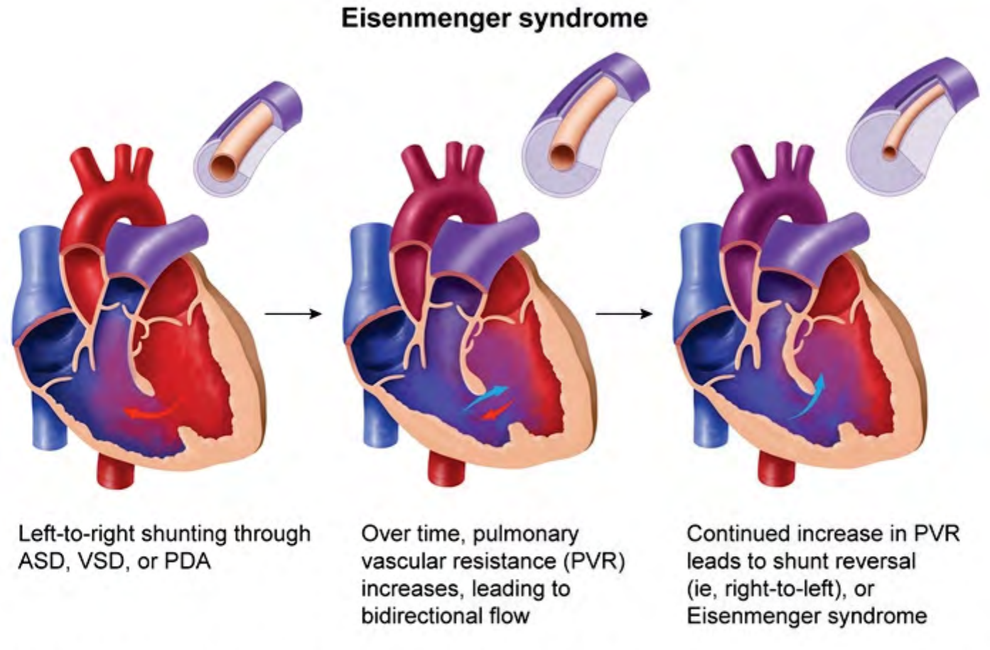

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Educational objective:

Adults with coarctation of the aorta often have chronic hypertension and are at increased risk of developing life-

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conditions (eg, Ehlers-Danlos syndrome), and lifestyle factors (eg, cigarette smoking) increase the risk of aneurysm development. **Coarctation of the aorta** is a congenital cardiac defect associated with secondary hypert

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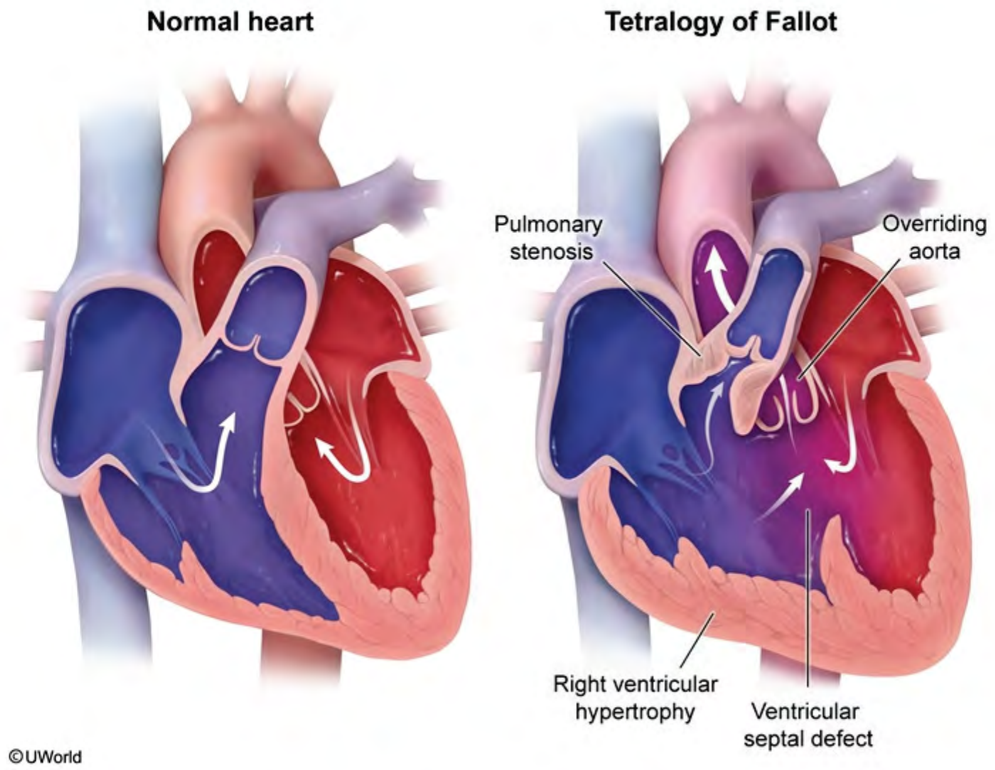

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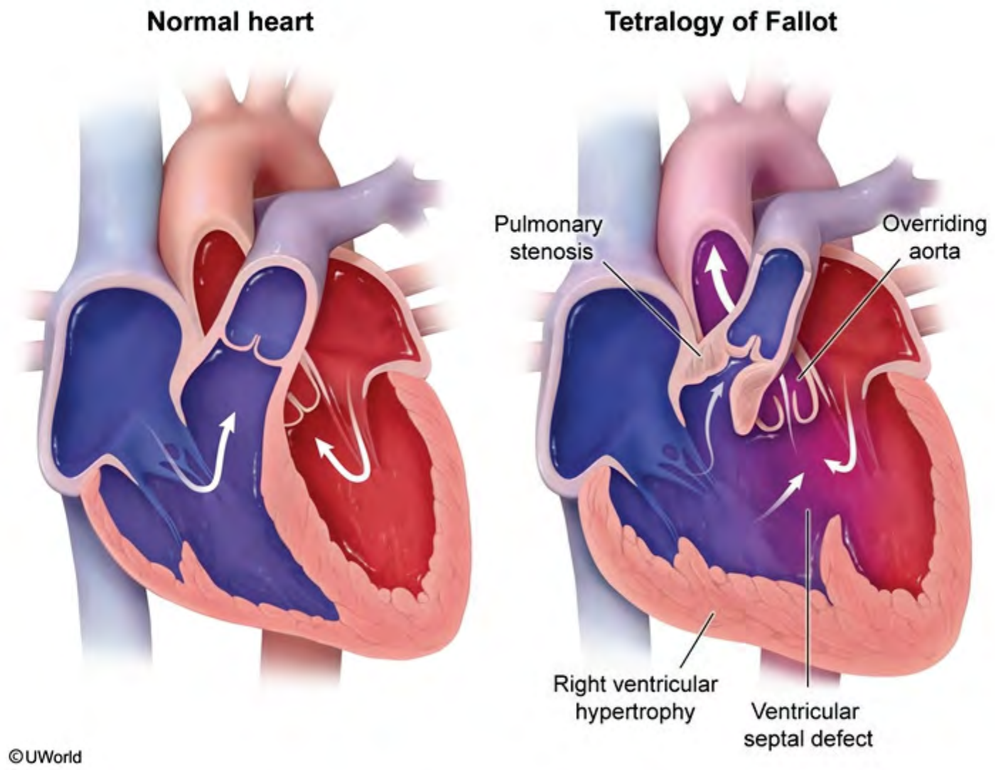

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A 34-year-old woman comes to the emergency department because of sharp chest pain that radiates to the left shoulder. The pain increases with inspiration and is partially relieved by sitting up and leaning forward. Review of her outpatient medical records shows that she was seen for a facial rash 6 months ago. She is also being evaluated for proteinuria that was identified during her last clinic appointment. Which of the following is the most likely cause of this patient's chest pain?

- A. Aortic dissection
- B. Cardiac tamponade
- C. Intimal hyperplasia of pulmonary arteries
- D. Non-infectious cardiac valve vegetations
- E. Pericardial inflammation

Submit

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A 34-year-old woman comes to the emergency department because of sharp chest pain that radiates to the left shoulder. The pain increases with inspiration and is partially relieved by sitting up and leaning forward. Review of her outpatient medical records shows that she was seen for a facial rash 6 months ago. She is also being evaluated for proteinuria that was identified during her last clinic appointment. Which of the following is the most likely cause of this patient's chest pain?

- A. Aortic dissection (1%)
- ✗ B. Cardiac tamponade (3%)
- C. Intimal hyperplasia of pulmonary arteries (1%)
- D. Non-infectious cardiac valve vegetations (9%)
- ✓ E. Pericardial inflammation (83%)

Incorrect
Correct answer
E

83%
Answered correctly

43 secs
Time Spent

2023
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Explanation

Manifestations of systemic lupus erythematosus	
Clinical symptoms	• Constitutional: fever, fatigue & weight loss • Symmetric, migratory arthritis • Skin: butterfly rash & photosensitivity • Serositis: pleurisy, pericarditis & peritonitis • Thromboembolic events (due to vasculitis & antiphospholipid antibodies) • Neurologic: cognitive dysfunction & seizures
	• Hemolytic anemia, thrombocytopenia & leukopenia

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Explanation

Manifestations of systemic lupus erythematosus	
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Laboratory findings	• Hemolytic anemia, thrombocytopenia & leukopenia
	• Hypocomplementemia (C3 & C4)
	• Antibodies:
	  ◦ Antinuclear antibodies (sensitive)
	  ◦ Anti-dsDNA & anti-Smith (specific)
	• Renal involvement: proteinuria & elevated creatinine

dsDNA = double-stranded DNA.	

This patient's chest pain is characteristic of **acute pericarditis**, which along with her facial rash and proteinuria is suggestive of underlying **systemic lupus erythematosus (SLE)**. SLE is a chronic autoimmune disease predominantly affecting women age 20-40 that causes **constitutional** and **multisystemic** symptoms. **Serosal inflammation** is common in SLE and most often manifests as pleuritis or pericarditis.

Pericarditis presents with severe and constant middle or left chest pain that may radiate to the neck and shoulders (particularly the trapezius ridge). The pain increases on **inspiration** (pleuritic) and is relieved by **sitting up and leaning forward** (postural). Auscultation of the chest reveals a scratchy sound called a **pericardial friction rub** that is best heard when the patient is leaning forward or lying prone. Additional cardiovascular manifestations in SLE include pericardial effusion, verrucous (Libman-Sacks) endocarditis, and increased risk of coronary artery disease.

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dsDNA = double-stranded DNA.

This patient's chest pain is characteristic of **acute pericarditis**, which along with her facial rash and proteinuria is suggestive of underlying **systemic lupus erythematosus (SLE)**. SLE is a chronic autoimmune disease predominantly affecting women age 20-40 that causes **constitutional** and **multisystemic** symptoms. **Serosal inflammation** is common in SLE and most often manifests as pleuritis or pericarditis.

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(Choice A) Aortic dissection occurs in patients with long-standing hypertension and those with Marfan and Ehlers-Danlos syndromes (the 2 latter conditions cause cystic medial necrosis of the aortic wall). It presents with abrupt-onset, severe, tearing chest pain that radiates to the back.

(Choice B) Cardiac tamponade is caused by accumulation of fluid in the pericardial space that prevents the heart from filling properly in diastole. It presents with dyspnea and tachypnea. Physical examination shows distended neck veins, hypotension, diminished heart sounds, and pulsus paradoxus (drop in systolic blood pressure $\geq$10 mm Hg on inspiration).

(Choice C) Intimal hyperplasia of the pulmonary arteries is characteristic of both primary and secondary pulmonary hypertension. It presents with dyspnea, malaise, and findings associated with right ventricular failure (eg, jugular venous distention, pedal edema, hepatomegaly). Severe symptomatic pulmonary hypertension is a rare complication of SLE.

(Choice D) Non-bacterial endocarditis (verrucous or Libman-Sacks endocarditis) refers to small, wart-like fibrinous lesions and generalized thickening that can affect the heart valves of patients with SLE. These are typically asymptomatic but may lead to valvular insufficiency or embolism.

Educational objective:

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suggestive of underlying **systemic lupus erythematosus (SLE)**. SLE is a chronic autoimmune disease predominantly affecting women age 20-40 that causes **constitutional** and **multisystemic** symptoms. **Serosal inflammation** is common in SLE and most often manifests as pleuritis or pericarditis.

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(Choice A) Aortic dissection occurs in patients with long-standing hypertension and those with Marfan and Ehlers-Danlos syndromes (the 2 latter conditions cause cystic medial necrosis of the aortic wall). It presents with abrupt-onset, severe, tearing chest pain that radiates to the back.

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(Choice C) Intimal hyperplasia of the pulmonary arteries is characteristic of both primary and secondary pulmonary hypertension. It presents with dyspnea, malaise, and findings associated with right ventricular failure (eg, jugular venous distention, pedal edema, hepatomegaly). Severe symptomatic pulmonary hypertension is a rare complication of SLE.

(Choice D) Non-bacterial endocarditis (verrucous or Libman-Sacks endocarditis) refers to small, wart-like fibrinous lesions and generalized thickening that can affect the heart valves of patients with SLE. These are typically asymptomatic but may lead to valvular insufficiency or embolism.

Educational objective:
Pericarditis is the most common cardiovascular manifestation associated with systemic lupus erythematosus. It presents with sharp pleuritic chest pain that is relieved by sitting up and leaning forward.

References

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A 10-day-old boy born at home is brought to the emergency department due to difficulty breathing and sweating with feeds. He was born at 38 weeks gestation via spontaneous vaginal delivery. The patient has been breastfeeding every 2 hours for 5-10 minutes per side. The mother notes that he sweats and his lips turn blue while he feeds. Weight is at the 5th percentile and length is at the 50th percentile. On examination, the patient is currently not cyanotic. There is a III/VI systolic crescendo-decrescendo murmur best heard along the left upper sternal border. Chest x-ray is shown below:

Which of the following pressure changes most likely occur in this patient during feedings?

Right ventricular	Pulmonary	Left atrial

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Right ventricular Pulmonary Left atrial

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Which of the following pressure changes most likely occur in this patient during feedings?

	Right ventricular pressure	Pulmonary arterial pressure	Left atrial pressure
A.	Decreased	Decreased	Decreased
B.	Decreased	Increased	Increased
C.	Increased	Decreased	Decreased
D.	Increased	Increased	Increased

Submit

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Which of the following pressure changes most likely occur in this patient during feedings?

	Right ventricular pressure	Pulmonary arterial pressure	Left atrial pressure	
✗ A.	Decreased	Decreased	Decreased	(9%)
B.	Decreased	Increased	Increased	(12%)
✓ C.	Increased	Decreased	Decreased	(39%)
D.	Increased	Increased	Increased	(36%)

Incorrect
Correct answer
C

39%
Answered correctly

03 mins, 49 secs
Time Spent

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Explanation

Tetralogy of Fallot	
Pathophysiology	• Embryonic deviation of infundibular septum creates 4 defects: ◦ RVOT obstruction ◦ Ventricular septal defect ◦ Overriding aorta ◦ Concentric RVH (secondary to RVOT obstruction)
Clinical presentation	• Based on severity of RVOT obstruction: ◦ Severe: profound neonatal cyanosis & hypoxemia ◦ Moderate: tet spells in infancy/childhood ◦ Mild: heart failure in childhood/adulthood • RVOT obstruction is dynamic (eg, worsens with feeding or crying) • Squatting can improve cyanosis via increased systemic afterload
Clinical findings	• Harsh systolic ejection murmur at LUSB (RVOT obstruction) • Soft or absent pulmonic component of S2 • Chest x-ray: boot-shaped heart caused by RVH
LUSB = left upper sternal border; **RVH** = right ventricular hypertrophy; **RVOT** = right ventricular outflow tract.	

This neonate has diaphoresis and episodic cyanosis with feeding, suggesting cyanotic congenital heart disease. The murmur of right ventricular outflow tract (RVOT) obstruction (ie, pulmonic stenosis) and evidence of right ventricular hypertrophy (ie, boot-shaped heart) on chest x-ray are consistent with **tetralogy of Fallot** (ToF). The anatomic defects of ToF are **RVOT obstruction** with an overriding aorta and large **ventricular septal defect** (VSD); concentric right ventricular hypertrophy develops due to increased right ventricular pressure load compared to normal.

The degree of RVOT obstruction is the major driver of symptoms in ToF because it determines how much blood flows through the pulmonary artery to the lungs and the magnitude and direction of shunting through the VSD.

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The degree of RVOT obstruction is the major driver of symptoms in ToF because it determines how much blood flows through the pulmonary artery to the lungs and the magnitude and direction of shunting through the VSD. Because some degree of RVOT obstruction is fixed and some degree is dynamic (ie, subject to an increase with activity [eg, crying, feeding]), many patients have left-to-right or neutral shunting through the VSD at baseline (and are acyanotic) but develop **cyanosis** when a dynamic **increase in RVOT obstruction** causes right-to-left shunting through the VSD (ie, **tet episode**).

During a tet episode, increased RVOT obstruction leads to **increased right ventricular pressure** and facilitates right-to-left shunting. There is also **decreased pulmonary arterial pressure** (blood cannot pass through the RVOT tract into the pulmonary artery, reducing pulmonary blood flow), and **decreased left atrial pressure** (the left atrium receives less blood returning from the lungs). Due to the large size of the VSD in ToF, left ventricular pressure usually closely matches right ventricular pressure (ie, the ventricles are somewhat like a single chamber).

A tet episode may be improved by bringing the knees to the chest. This maneuver increases systemic afterload, reducing right-to-left shunting through the VSD and forcing more blood through the RVOT.

Educational objective:
The major defects in tetralogy of Fallot are right ventricular outflow tract (RVOT) obstruction and a ventricular septal defect (VSD). In many patients, activities (eg, feeding, crying) can precipitate cyanotic episodes (tet episodes) by causing a dynamic increase in RVOT obstruction. During these episodes, increased right ventricular pressure results in right-to-left shunting through the VSD; because blood is shunted away from the pulmonary circulation, pulmonary arterial and left atrial pressures are decreased.

References

Tetralogy of Fallot

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Clinical findings	• Soft or absent pulmonic component of S2 • Chest x-ray: boot-shaped heart caused by RVH

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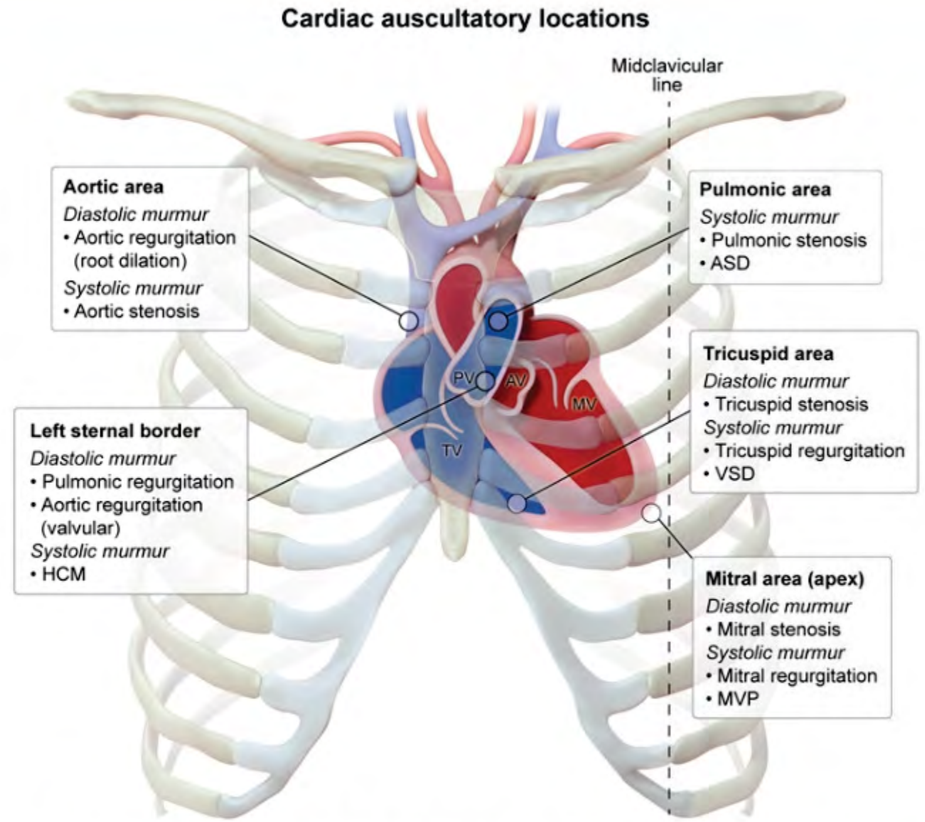

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Clinical findings	• Soft or absent pulmonic component of S2 • Chest x-ray: boot-shaped heart caused by RVH

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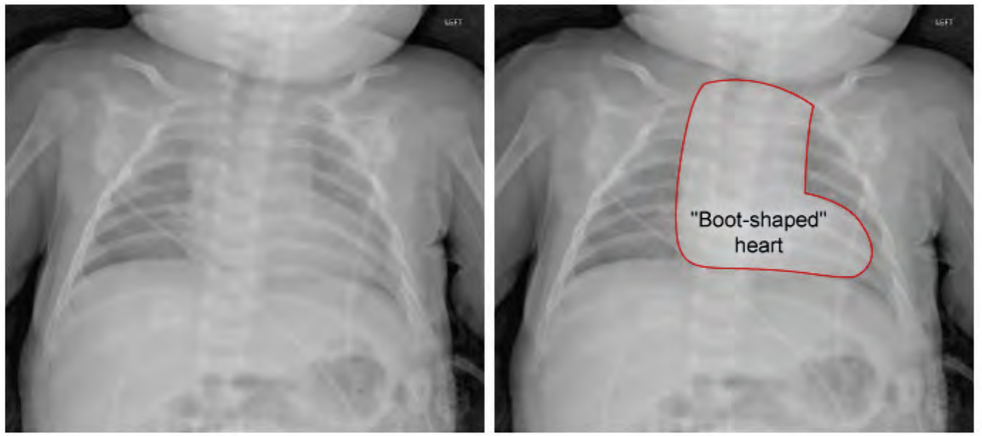
Tetralogy of Fallot



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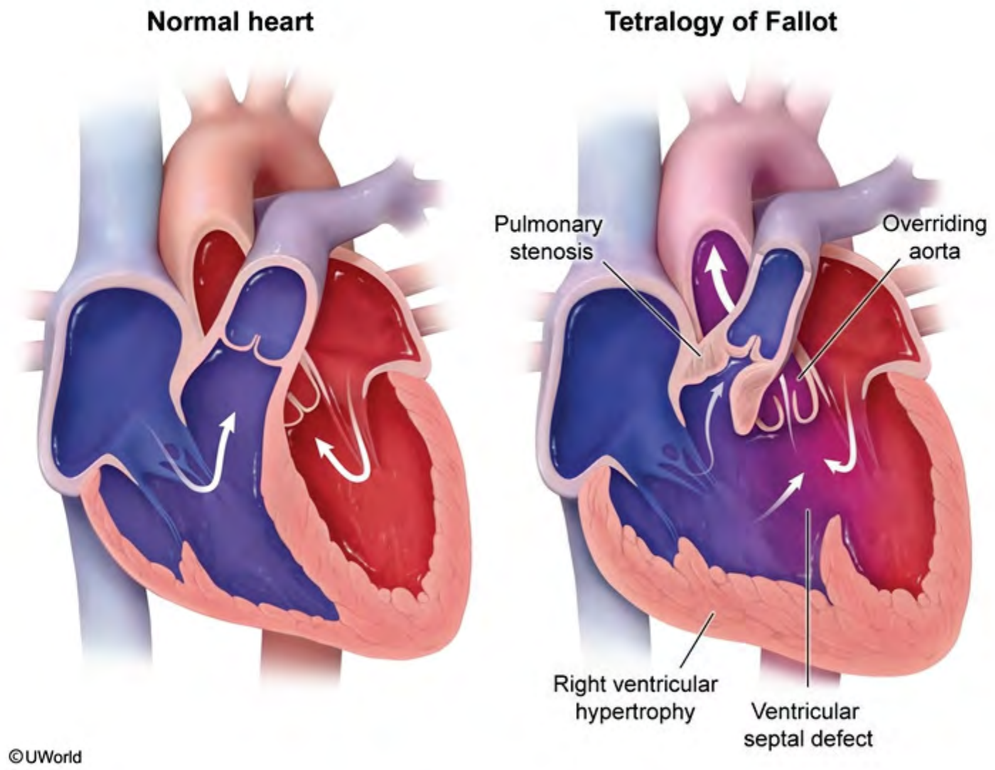

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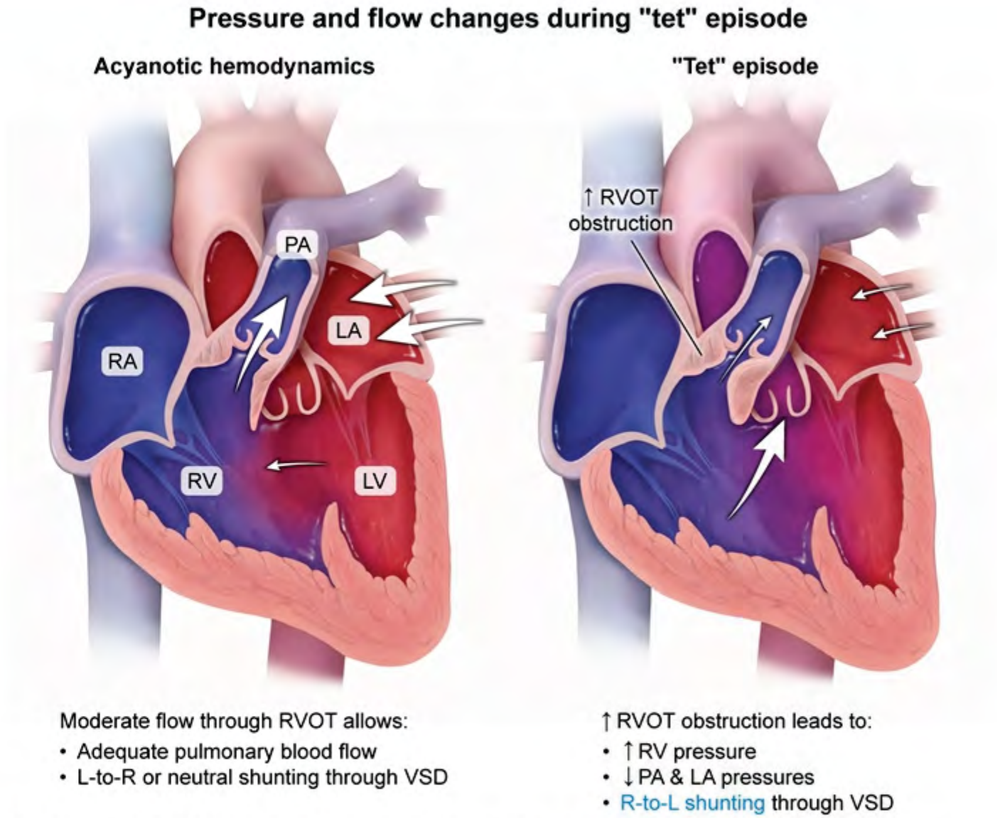

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Clinical findings	• Soft or absent pulmonic component of S2 • Chest x-ray: boot-shaped heart caused by RVH

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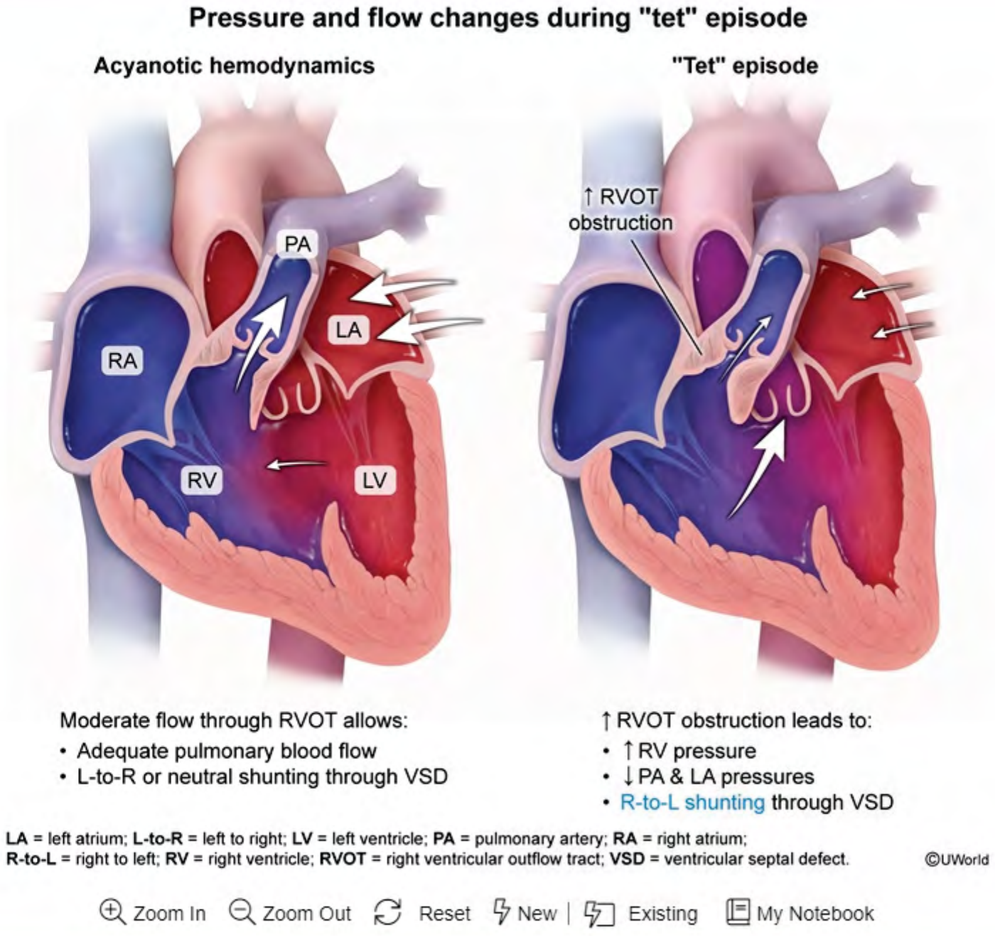

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A 46-year-old man comes to the office for an annual examination. He has an uncomfortable heartbeat sensation at night that he tries to decrease by sleeping on his right side. The patient has had mild shortness of breath with exertion over the last 6 months, but he has no chest pain. He was told during a wellness check approximately 2 years ago that he had a heart murmur. The patient has no other medical problems. Physical examination reveals bounding femoral pulses and carotid pulsations that are accompanied by head bobbing. Which of the following is the most likely diagnosis?

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Atrial septal defect
- D. Coarctation of the aorta
- E. Mitral regurgitation
- F. Mitral stenosis
- G. Pulmonary stenosis
- H. Tricuspid regurgitation

Submit

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- ✓ A. Aortic regurgitation (75%)
- B. Aortic stenosis (12%)
- C. Atrial septal defect (0%)
- D. Coarctation of the aorta (5%)
- E. Mitral regurgitation (2%)
- F. Mitral stenosis (0%)
- G. Pulmonary stenosis (0%)
- H. Tricuspid regurgitation (1%)

Correct

75% Answered correctly

03 secs Time Spent

2023 Version

Explanation

This presentation is most suggestive of **aortic regurgitation**. The inability of aortic valve leaflets to effectively close during diastole leads to regurgitation of blood back into the left ventricular (LV) cavity with an increase in LV end-diastolic volume and wall stress. The resultant chamber enlargement and eccentric hypertrophy increase total stroke volume, which is often felt as a sense of pounding or an uncomfortable feeling of heartbeat (especially when lying on the left side)

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This presentation is most suggestive of **aortic regurgitation**. The inability of aortic valve leaflets to effectively close during diastole leads to regurgitation of blood back into the left ventricular (LV) cavity with an increase in LV end-diastolic volume and wall stress. The resultant chamber enlargement and eccentric hypertrophy increase total stroke volume, which is often felt as a sense of pounding or an uncomfortable feeling of heartbeat (especially when lying on the left side).

Physical examination reveals an early "blowing" **diastolic decrescendo murmur** best heard at the left sternal border in the third or fourth intercostal space. The precordial impulse is hyperdynamic and displaced laterally and downward. **Bounding femoral and carotid pulses**, marked by abrupt distension and quick collapse ("water-hammer" pulses), are the result of the wide pulse pressure. Some patients exhibit **head bobbing** with carotid pulsations (de Musset sign) due to transfer of momentum from the large LV stroke volume to the head and neck. Significant systolic pulsations may also be noticed in other organs (eg, liver, spleen, retina) and the fingertips.

(Choice B) Patients with severe aortic stenosis have a characteristic arterial pulse – small pulse amplitude (pulsus parvus) with a delayed peak and slower upstroke of the arterial pulse (pulsus tardus) due to diminished stroke volume and prolonged ejection time.

(Choice C) The presence of atrial septal defect leads to left-to-right intracardiac shunting, which can cause a hyperdynamic right ventricular impulse. It does not cause any significant change in arterial pulse character.

(Choice D) Coarctation of the aorta causes systolic hypertension in the upper extremities along with characteristic diminished and/or delayed femoral pulses (brachial-femoral delay).

(Choice E) Arterial pulse, pulse pressure, and forward stroke volume remain normal in patients with chronic (compensated) mitral regurgitation.

(Choice F) Arterial pulses are reduced in volume and amplitude in patients with mitral stenosis due to decreased LV end-diastolic volume and stroke volume.

(Choices G and H) Tricuspid regurgitation and/or pulmonary stenosis are right-sided valvular lesions and do not cause any specific arterial pulse pattern on physical examination.

Educational objective:

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(Choices G and H) Tricuspid regurgitation and/or pulmonary stenosis are right-sided valvular lesions and do not cause any specific arterial pulse pattern on physical examination.

Educational objective:
Aortic regurgitation causes an increase in total stroke volume with abrupt distension and rapid falloff of peripheral arterial pulses, resulting in a wide pulse pressure. This leads to bounding peripheral pulses and head bobbing with each heartbeat.

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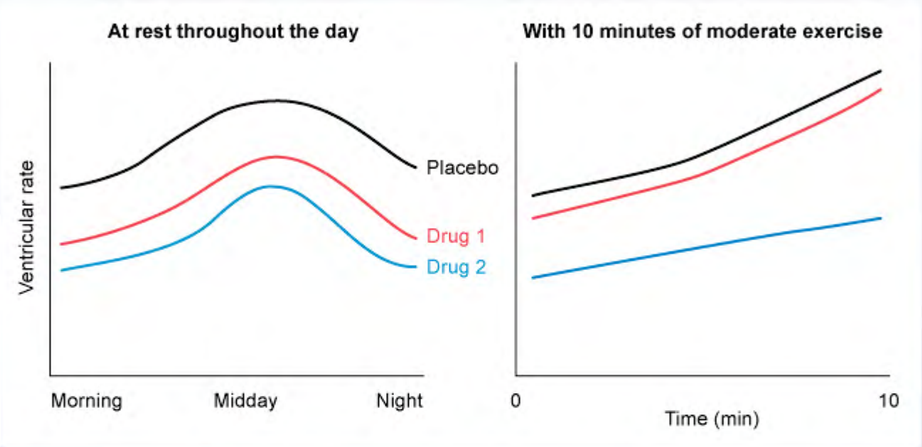
An investigator studies the effects of 2 different medications on the heart rate of patients with persistent atrial fibrillation. After participants receive therapeutic doses of medication for several days, their heart rates are measured for 24 hours at rest and during 10 minutes of moderate exercise. The results are compared against placebo and shown in the graphs below. Which of the following medications is most likely represented by the red line (drug 1)?



- A. Amlodipine
- B. Atenolol
- C. Digoxin
- D. Lisinopril

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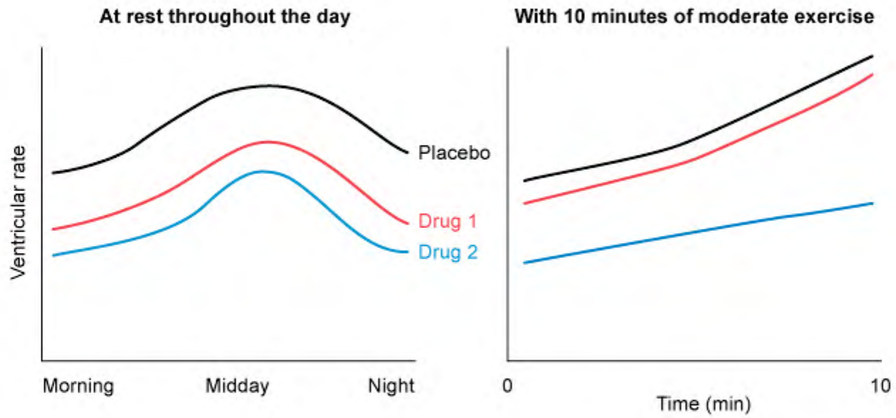

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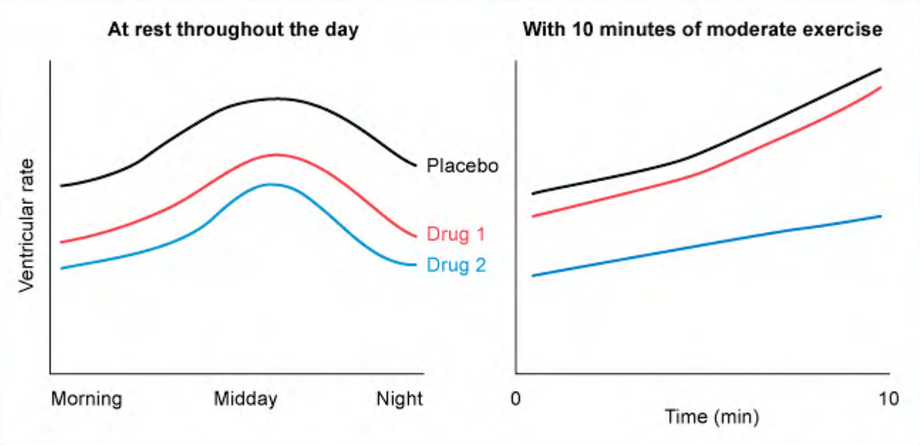

- ✗ A. Amlodipine (20%)
- B. Atenolol (36%)
- ✓ C. Digoxin (35%)
- D. Lisinopril (7%)

Incorrect
Correct answer
C

35%
Answered correctly

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Time Spent

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Version

Explanation

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✓ C. Digoxin (35%)

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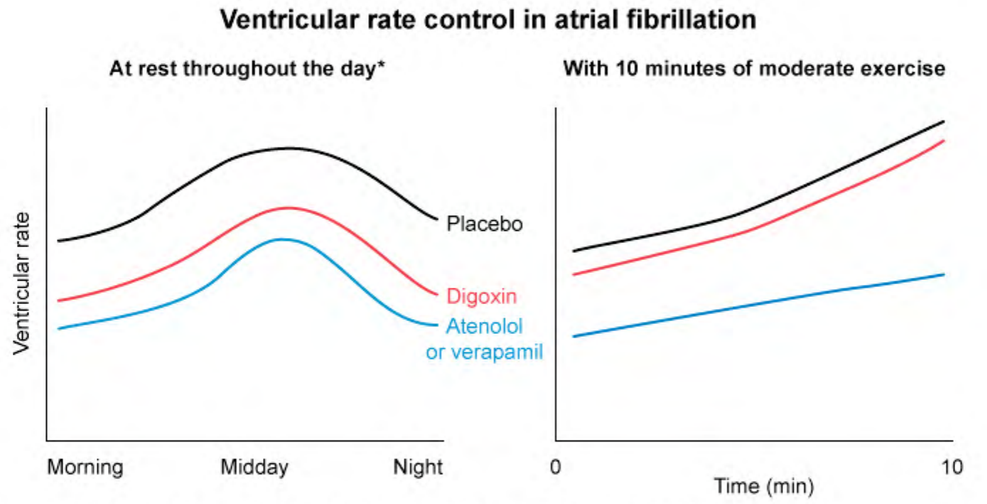

*Curves depict the natural variation of heart rate throughout the day, with a peak around midday.

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tone. This mechanism allows for effective rate control both during rest and exercise. For this reason, beta

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*Curves depict the natural variation of heart rate throughout the day, with a peak around midday.

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Persistent **atrial fibrillation** is usually managed with a **rate-control strategy** that focuses on maintaining an acceptable ventricular contraction rate during both rest (eg, <85/min) and moderate exercise (eg, <110-120/min) despite continued rapid and disorganized conduction of the atria. Pharmacologic agents that allow for rate control do so by **slowing conduction through the atrioventricular (AV) node** via various mechanisms:

- Beta blockers (eg, atenolol, metoprolol) **(Choice B)** slow AV node conduction via blockade of sympathetic tone. This mechanism allows for effective rate control both during rest and exercise. For this reason, beta blockers are often the preferred agent for rate control of atrial fibrillation.
- Nondihydropyridine calcium channel blockers (eg, verapamil, diltiazem) block AV nodal calcium channels to slow AV node conduction both at rest and during exercise. These drugs have similar rate control efficacy to that of beta blockers, although beta blockers are slightly superior at controlling heart rate during exercise.
- **Digoxin** slows AV node conduction via **increased parasympathetic tone**. This mechanism is effective at rest, when sympathetic tone is low, but it **poorly controls ventricular rate during exercise**, when high levels of sympathetic tone accelerate AV node conduction. For this reason, digoxin is often used only as an adjunctive agent for rate control in atrial fibrillation.

(Choices A and D) Dihydropyridine calcium channel blockers (eg, amlodipine, nifedipine) and ACE inhibitors (eg, lisinopril) are vasodilators with no significant effect on the AV node. These drugs are not expected to slow heart rate either at rest or with exercise (ie, both resting and exercise curves are expected to closely match placebo).

Educational objective:
Digoxin slows atrioventricular node conduction by increasing parasympathetic tone. This mechanism is effective at controlling ventricular rate in atrial fibrillation at rest, but it poorly controls ventricular rate during exercise, when parasympathetic output is low relative to high levels of sympathetic tone.

Pharmacology	Cardiovascular System	Atrial fibrillation
Subject	System	Topic

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A 35-year-old previously healthy man is evaluated for several episodes of syncope in the past 6 weeks. Physical examination is unremarkable. Echocardiogram shows no structural heart defect. An electrophysiologic study is performed during which catheters are passed into the patient's right and left atrium to record atrial electric potentials. Cardiac monitor currently shows normal sinus rhythm. Which of the following is the most likely earliest site of electric activation?

- A. Junction of left atrium and atrial appendage
- B. Left atrium near the opening of pulmonary veins
- C. Right atrium near the opening of inferior vena cava
- D. Right atrium near the opening of superior vena cava
- E. Right atrium near the septal cusp of tricuspid valve

Submit

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- ✗ A. Junction of left atrium and atrial appendage (2%)
- B. Left atrium near the opening of pulmonary veins (2%)
- C. Right atrium near the opening of inferior vena cava (6%)
- ✓ D. Right atrium near the opening of superior vena cava (80%)
- E. Right atrium near the septal cusp of tricuspid valve (7%)

Incorrect
Correct answer
D

80%
Answered correctly

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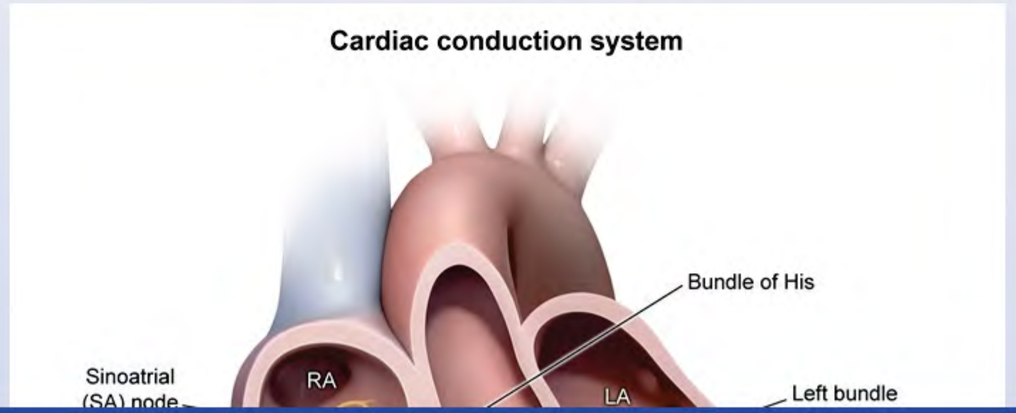
Explanation



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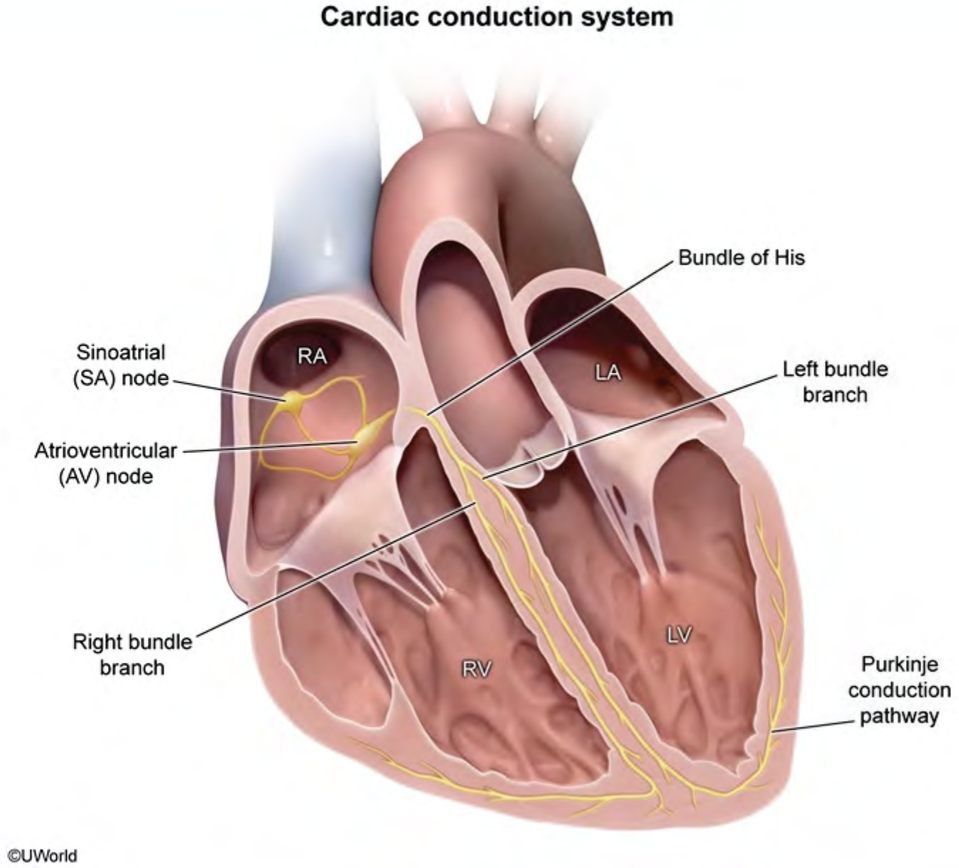

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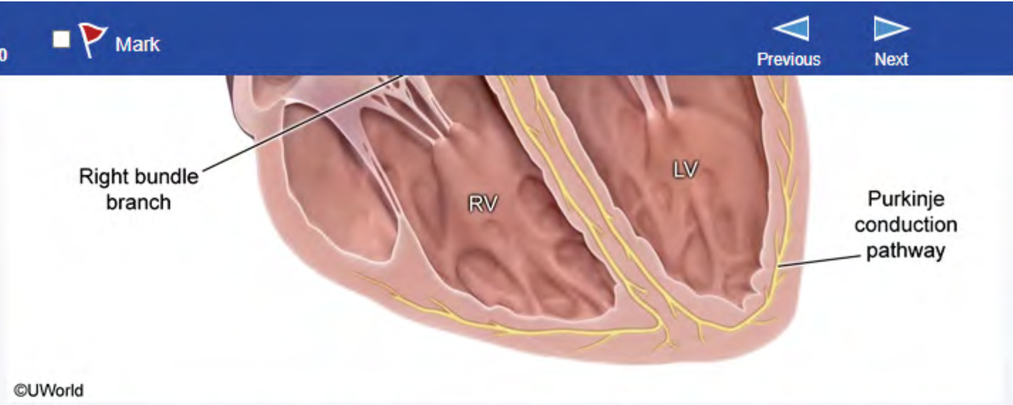

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The cardiac conduction system consists of the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, right and left bundle branches, and the Purkinje system (which directly activates the ventricular myocardium). In patients **with normal sinus rhythm** (as in this patient), the **SA node** acts as the dominant pacemaker site and is the site of earliest electrical activation. The SA node is a compact subepicardial structure that consists of specialized pacemaker cells located at the **junction of the right atrium and superior vena cava**.

(Choices A and B) The SA node is located in the right, not the left, atrium. The area around the opening of the pulmonary veins in the left atrium is frequently involved in the pathogenesis of atrial fibrillation.

(Choice C) The junction of the right atrium and inferior vena cava is not a major site of electrical activity.

(Choice E) The AV node is located in the right atrium near the septal cusp of the tricuspid valve.

Educational objective:
The sinoatrial node consists of specialized pacemaker cells located at the junction of the right atrium and superior vena cava. It is the site of earliest electrical activation in patients with sinus rhythm.

References

- The sinoatrial node, a heterogeneous pacemaker structure.
- The anatomy of the cardiac conduction system.

Anatomy	Cardiovascular System	Sinoatrial node
Subject	System	Topic

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A 54-year-old man comes to the office due to 2 days of redness and pain in his right arm. He was recently diagnosed with superficial thrombophlebitis involving the left lower and upper extremities. His symptoms then subsided within days of nonsteroidal anti-inflammatory drug therapy only. The patient has no other past medical history. He has some abdominal discomfort that he attributes to gastroesophageal reflux disease. Temperature is 37.6 C (99.7 F). On examination, there is erythema and tenderness extending linearly from the right forearm to just anterior to the right antecubital fossa. No fluid collection is palpable. Complete blood count shows mild leukocytosis. The patient's symptoms may indicate the presence of which of the following?

- A. Celiac sprue
- B. Chlamydial infection
- C. Hyperthyroidism
- D. Polycystic kidneys
- E. Visceral cancer

Submit

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- ✗ A. Celiac sprue (22%)
- B. Chlamydial infection (8%)
- C. Hyperthyroidism (3%)
- D. Polycystic kidneys (5%)
- ✓ E. Visceral cancer (60%)

Incorrect
Correct answer
E

60%
Answered correctly

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Explanation

Migratory thrombophlebitis should raise suspicion for **cancer**. Hypercoagulability is a very common paraneoplastic syndrome seen most frequently in **visceral** adenocarcinomas of the **pancreas**, colon, and lung. **Hypercoagulability** develops because adenocarcinomas produce a thromboplastin-like substance capable of causing chronic intravascular coagulations that can disseminate and tend to migrate.

Migratory superficial thrombophlebitis, known as **Trousseau syndrome**, is named after Armand Trousseau, a well-respected French physician who first described the association with cancer. Trousseau later diagnosed his own visceral cancer after developing the syndrome.

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Migratory thrombophlebitis should raise suspicion for **cancer**. Hypercoagulability is a very common paraneoplastic syndrome seen most frequently in **visceral** adenocarcinomas of the **pancreas**, colon, and lung. **Hypercoagulability** develops because adenocarcinomas produce a thromboplastin-like substance capable of causing chronic intravascular coagulations that can disseminate and tend to migrate.

Migratory superficial thrombophlebitis, known as **Trousseau syndrome**, is named after Armand Trousseau, a well-respected French physician who first described the association with cancer. Trousseau later diagnosed his own visceral cancer after developing the syndrome.

Trousseau also described the Trousseau sign (hand/forearm muscle spasms on sphygmomanometric measurement) associated with hypocalcemia.

(Choice A) Celiac sprue has been associated with a hemorrhagic diathesis (rather than abnormal thrombosis). The malabsorption resulting from celiac sprue can result in vitamin K deficiency.

(Choices B and D) Polycystic kidney disease is not among the conditions commonly associated with an acquired prothrombotic state. Disseminated chlamydia infections are classically associated with reactive arthritis (not thrombophlebitis), and infections in general can create a systemic inflammatory state that favors thrombosis. However, malignancy would be a more likely explanation for migratory superficial thrombophlebitis accompanied by vague abdominal pain.

(Choice C) Hyperthyroidism has been associated with a hypercoagulable state and is occasionally reported in cases of cerebral venous thrombosis, but there is no evidence of a significant association with venous thrombotic events.

Educational objective:
A paraneoplastic syndrome of hypercoagulability may be seen in some patients with cancer, especially adenocarcinomas of the pancreas, colon, or lung. Superficial venous thromboses may therefore appear in one site and then resolve, only to recur in another site. This is known as Trousseau syndrome (migratory superficial thrombophlebitis), an indication of visceral cancer.

Pathology Cardiovascular System Thrombophlebitis

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An 18-year-old man comes to the clinic due to hematuria and intermittent left flank pain of several months duration. He has no history of trauma or sexually transmitted diseases and no associated fever or dysuria. Examination reveals a soft abdomen with normal bowel sounds and no localized tenderness. Urinalysis confirms 3+ blood but no white blood cells, crystals, or organisms. Contrast-enhanced CT scan shows no abnormalities in the ureters or kidneys but does reveal compression of the left renal vein between the superior mesenteric artery and the aorta. Which of the following is most likely to develop due to the vascular abnormality seen in this patient?

- A. Esophageal varices
- B. Left-sided ankle swelling
- C. Periumbilical venous distension
- D. Rectal varices
- E. Varicocele

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- ✗ A. Esophageal varices (2%)
- B. Left-sided ankle swelling (3%)
- C. Periumbilical venous distension (6%)
- D. Rectal varices (2%)
- ✓ E. Varicocele (84%)

Incorrect
Correct answer
E

84%
Answered correctly

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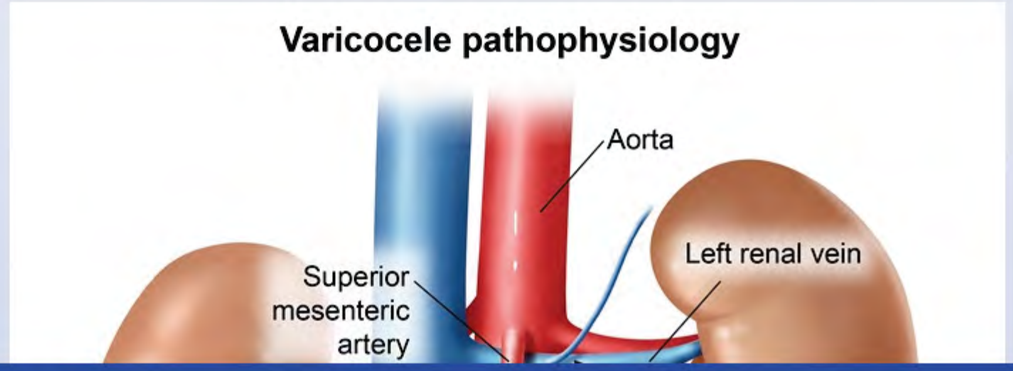
Explanation



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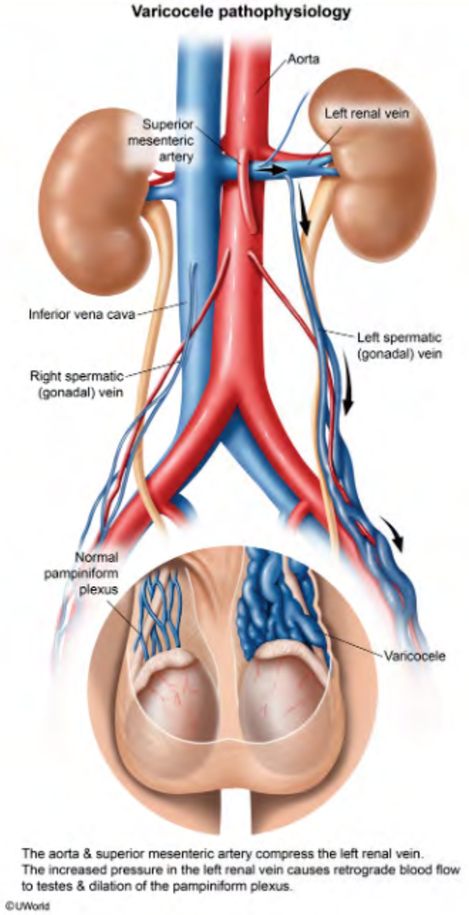

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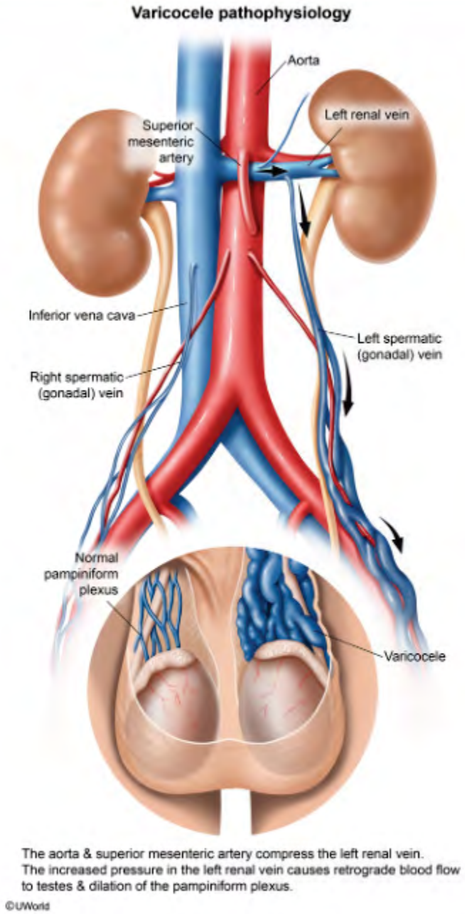

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common iliac vein, left external iliac vein, left femoral vein, or any of the other major veins of the left leg.

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to testes & dilation of the pampiniform plexus.

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The right renal vein is a relatively short structure and runs anterior to the right renal artery before joining the inferior vena cava (IVC). The right gonadal vein also drains directly to the IVC. In contrast, the **left renal vein** is significantly longer and runs posterior to the splenic vein before crossing the aorta beneath the superior mesenteric artery. The left gonadal vein joins the left renal vein upstream of where it crosses the aorta and does not enter the IVC directly.

The pressure within the left renal vein is often higher than on the right due to compression between the aorta and the superior mesenteric artery ("**nutcracker effect**"). Pressure in the left renal vein can also be elevated due to compression from a left-sided abdominal or retroperitoneal mass. Persistently elevated pressure in the left renal vein can cause flank or abdominal pain, along with gross or microscopic hematuria (left renal vein entrapment syndrome). Increased pressure in the left gonadal vein results in valve leaflet failure and varices of the testicular pampiniform plexus (**varicocele**).

(Choices A, C, and D) Esophageal varices, rectal varices, and periumbilical venous distension are signs of portal venous hypertension. The renal veins are not part of the portal system.

(Choice B) Unilateral left-sided ankle swelling can result from many causes, including obstruction of the left common iliac vein, left external iliac vein, left femoral vein, or any of the other major veins of the left leg.

Educational objective:
Pressure in the left renal vein may become elevated due to compression where the vein crosses the aorta beneath the superior mesenteric artery. This "nutcracker effect" can cause hematuria and flank pain. Pressure can also be elevated in the left gonadal vein, leading to formation of a varicocele.

References

- Nutcracker syndrome.

Anatomy	Cardiovascular System	Varicocele
Subject	System	Topic

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A 62-year-old man with a long history of hypertension comes to the clinic for a routine physical examination. Blood pressure is 150/90 mm Hg and pulse is 74/min and regular. Cardiac auscultation reveals a low-frequency, presystolic sound that immediately precedes S1 and is best heard during expiration when the patient is lying on his left side. Chest x-ray reveals extensive calcification around the mitral and aortic valves. Which of the following is the most likely explanation for the additional heart sound?

- A. Increased flow velocity through the aortic valve
- B. Increased stiffness of the left ventricular wall
- C. Rapid passive filling of the ventricles
- D. Restricted motion of the aortic valve cusps
- E. Systolic anterior motion of the mitral valve

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A 62-year-old man with a long history of hypertension comes to the clinic for a routine physical examination. Blood pressure is 150/90 mm Hg and pulse is 74/min and regular. Cardiac auscultation reveals a low-frequency, presystolic sound that immediately precedes S1 and is best heard during expiration when the patient is lying on his left side. Chest x-ray reveals extensive calcification around the mitral and aortic valves. Which of the following is the most likely explanation for the additional heart sound?

- ✗ A. Increased flow velocity through the aortic valve (4%)
- ✓ B. Increased stiffness of the left ventricular wall (67%)
- C. Rapid passive filling of the ventricles (8%)
- D. Restricted motion of the aortic valve cusps (11%)
- E. Systolic anterior motion of the mitral valve (8%)

Incorrect
Correct answer
B

67%
Answered correctly

04 secs
Time Spent

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Explanation

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive diastolic filling	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation
S4	• Heard just before S1 • Caused by blood striking a stiff LV wall	• Concentric LV hypertrophy • Restrictive cardiomyopathy

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Explanation

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive diastolic filling	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation
S4	• Heard just before S1 • Caused by blood striking a stiff LV wall during atrial contraction	• Concentric LV hypertrophy • Restrictive cardiomyopathy • Acute myocardial infarction
*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70). **EF** = ejection fraction; **LV** = left ventricular.		

This patient has an **S4** (low-frequency sound occurring just before S1), which is most likely secondary to concentric left ventricular hypertrophy (LVH) from longstanding hypertension (**hypertensive heart disease**). The calcifications revealed on chest x-ray likely represent degenerative mitral and aortic valve calcification, which is age related and typically accelerated by systemic hypertension.

An S4 is a sign of **diastolic dysfunction**. It occurs due to blood striking a stiffened ventricular wall during **atrial contraction** at the end of diastole. An S4 may be present in any condition that causes **reduced ventricular compliance** (eg, hypertensive heart disease, restrictive cardiomyopathy, acute myocardial infarction). A left-sided S4 is best heard with the bell of the stethoscope over the cardiac apex with the patient in the left lateral decubitus position; it will intensify during expiration due to increased blood flow from the lungs to the left atrium.

(Choices A and D) Age-related aortic valve calcification can cause restricted motion of the aortic valve cusps and lead to aortic stenosis. There may be a high-frequency ejection click heard just ***after*** S1, followed by the crescendo-decrescendo systolic murmur of aortic stenosis created by increased flow velocity through the aortic

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*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70).
EF = ejection fraction; **LV** = left ventricular.

This patient has an **S4** (low-frequency sound occurring just before S1), which is most likely secondary to concentric left ventricular hypertrophy (LVH) from longstanding hypertension (**hypertensive heart disease**). The calcifications revealed on chest x-ray likely represent degenerative mitral and aortic valve calcification, which is age related and typically accelerated by systemic hypertension.

An S4 is a sign of **diastolic dysfunction**. It occurs due to blood striking a stiffened ventricular wall during **atrial contraction** at the end of diastole. An S4 may be present in any condition that causes **reduced ventricular compliance** (eg, hypertensive heart disease, restrictive cardiomyopathy, acute myocardial infarction). A left-sided S4 is best heard with the bell of the stethoscope over the cardiac apex with the patient in the left lateral decubitus position; it will intensify during expiration due to increased blood flow from the lungs to the left atrium.

(Choices A and D) Age-related aortic valve calcification can cause restricted motion of the aortic valve cusps and lead to aortic stenosis. There may be a high-frequency ejection click heard just *after* S1, followed by the crescendo-decrescendo systolic murmur of aortic stenosis created by increased flow velocity through the aortic valve (and best heard at the right upper sternal border). Aortic stenosis is also a potential cause of concentric LVH and an S4, but it is a less common cause than chronic hypertension.

(Choice C) An S3 is caused by blood filling an enlarged (or overfull) ventricular cavity during early passive diastolic filling. It occurs shortly after S2 and is often heard in patients with heart failure with reduced ejection fraction or other causes of ventricular volume overload (eg, mitral or aortic regurgitation).

(Choice E) Systolic anterior motion of the mitral valve can exacerbate left ventricular outflow tract obstruction in patients with hypertrophic cardiomyopathy (HCM). HCM is expected to cause a systolic ejection murmur best heard at the left sternal border. An S4 is also sometimes heard with HCM.

Educational objective:
An S4 is a low-frequency, late diastolic sound that immediately precedes S1. It is caused by blood striking a stiffened ventricular wall during atrial contraction; concentric left ventricular hypertrophy due to chronic hypertension (or less commonly due to aortic stenosis) is a common cause of an S4.

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*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70).

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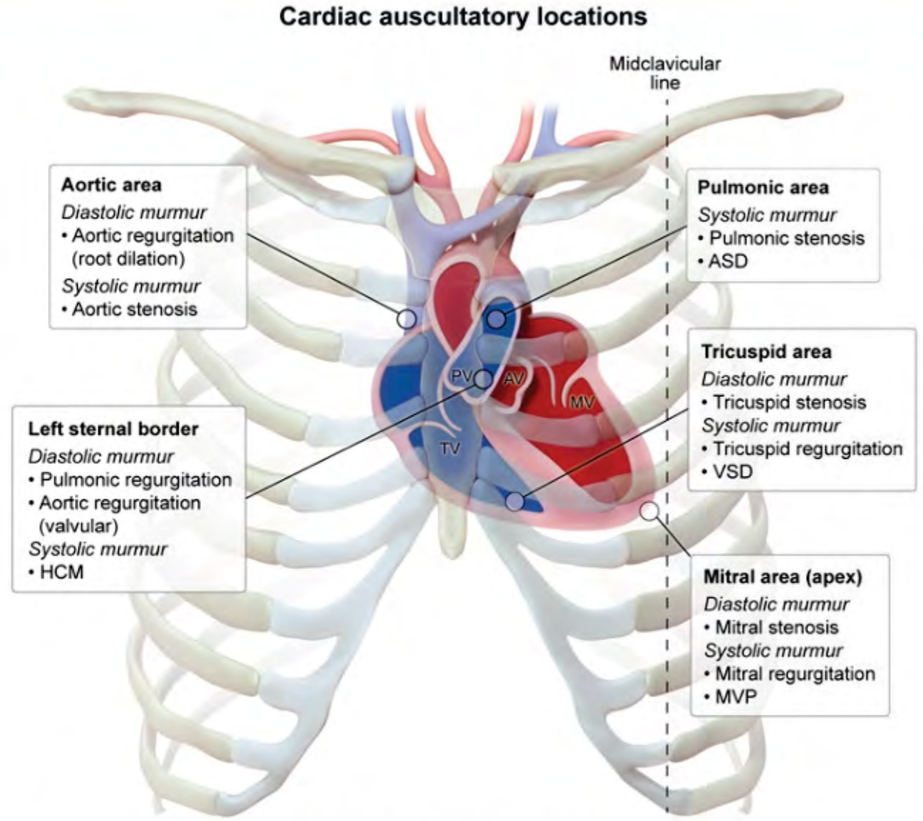

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A 45-year-old man comes to the hospital with acute onset of severe chest pain and diaphoresis. The patient describes the pain as "squeezing," different from any discomfort he has ever had. He has a past medical history of hypertension, and his father underwent coronary artery bypass grafting at age 50. The patient is diagnosed with acute ST-elevation myocardial infarction and undergoes an urgent coronary intervention with stent placement into the right coronary artery. He is also started on high-intensity atorvastatin therapy, along with antiplatelet therapy and appropriate medications to control blood pressure. Four weeks later, the patient's total cholesterol level is 140 mg/dL, down from 200 mg/dL before discharge. Which of the following has most likely increased as the result of the therapy?

- A. ApoB-100 concentration
- B. Biliary excretion of cholesterol
- C. LDL receptor density
- D. Plasma free fatty acids
- E. VLDL concentration

Submit

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- ✗ A. ApoB-100 concentration (8%)
- B. Biliary excretion of cholesterol (8%)
- ✓ C. LDL receptor density (74%)
- D. Plasma free fatty acids (5%)
- E. VLDL concentration (3%)

Incorrect
Correct answer
C

74%
Answered correctly

04 secs
Time Spent

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Explanation

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B. Biliary excretion of cholesterol (8%)

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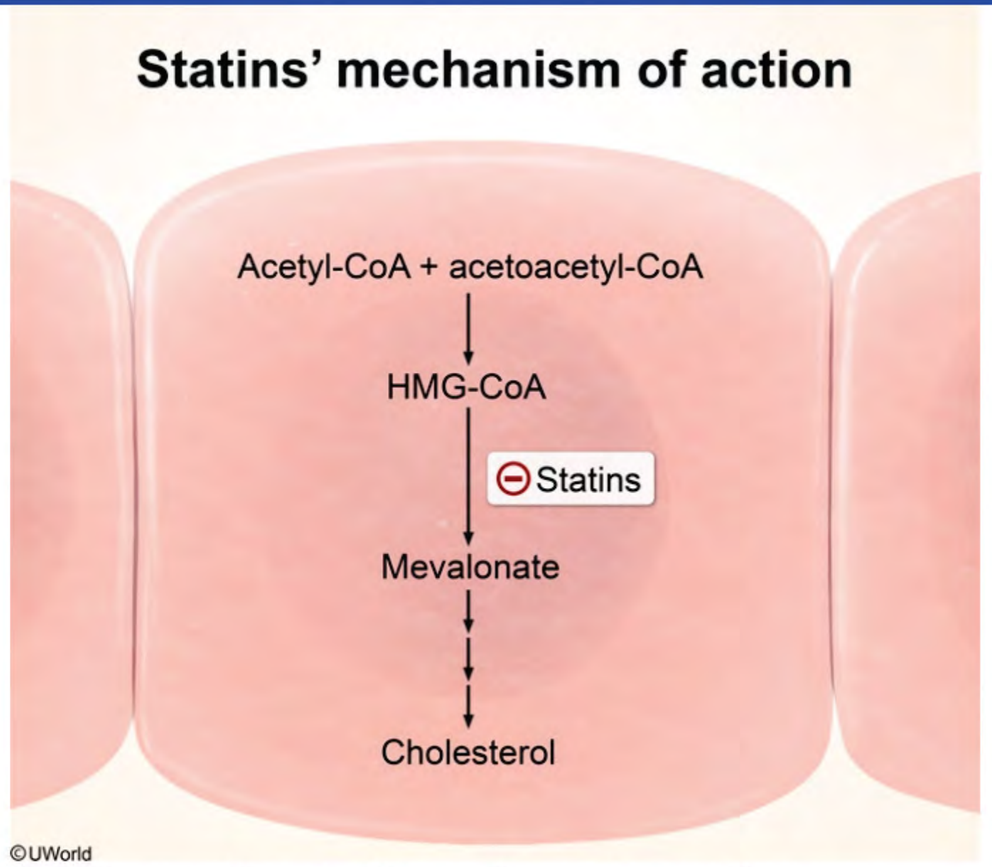

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HMG-CoA reductase inhibitors (**statins**) inhibit the rate-limiting enzyme in hepatic cholesterol synthesis. In response to these changes, hepatocytes increase their surface expression of the **LDL receptor** to increase uptake of circulating LDL. Treatment generally produces a 20%-50% reduction in serum cholesterol and LDL concentration.

High-intensity statin therapy reduces the risk of **acute coronary events**. This risk reduction is greater than that seen with other classes of lipid-lowering medications and is likely due to additional properties of statins. Besides effects on lipids, statins have anti-inflammatory properties, improve endothelial dysfunction, and appear to stabilize atherosclerotic plaques.

(Choice A) ApoB-100 is an apoprotein present in VLDL and LDL. Decreases in circulating LDL and VLDL will cause the ApoB-100 concentration to decrease as well.

(Choice B) The biliary excretion of cholesterol decreases in patients on statins due to decreased de novo hepatic synthesis of cholesterol.

(Choice D) Statins lower serum triglycerides modestly and have only a minimal effect on plasma free fatty acid concentrations.

(Choice E) Statins produce small decreases in VLDL. Fibrates and nicotinic acid therapy decrease hepatic production of VLDL and are the mainstay of treatment for primary hypertriglyceridemia (increased VLDL).

Educational objective:
Treatment with statins causes hepatocytes to increase their LDL receptor density, leading to increased uptake of circulating LDL.

References

- Statins: a new insight into their mechanisms of action and consequent pleiotropic effects.

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A 69-year-old man is brought to the emergency department with sudden onset palpitations and dyspnea. His medical history is significant for hypertension and gastroesophageal reflux disease. ECG reveals a heart rate of 120/min with an irregularly irregular rhythm, narrow QRS complexes, and no organized P waves. Which of the following physiologic factors most likely determines the ventricular contraction rate in this patient?

- A. Atrial muscle depolarization rate
- B. Atrioventricular node refractory period
- C. Bundle branch conductivity
- D. Purkinje system pacemaker activity
- E. Sinoatrial node discharge rate
- F. Ventricular muscle refractory period

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- A. Atrial muscle depolarization rate (9%)
- ✓ B. Atrioventricular node refractory period (48%)
- ✗ C. Bundle branch conductivity (5%)
- D. Purkinje system pacemaker activity (16%)
- E. Sinoatrial node discharge rate (6%)
- F. Ventricular muscle refractory period (13%)

Incorrect
Correct answer
B

48%
Answered correctly

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Explanation

This patient's ECG findings are diagnostic for **atrial fibrillation** with rapid ventricular response. Atrial fibrillation is the most common cardiac arrhythmia and is characterized by **absent P waves, irregularly irregular R-R intervals**, and **narrow QRS complexes**. Rapid, irregular, low amplitude fibrillatory waves are also frequently seen and represent chaotic, continuous atrial depolarizations.

Atrial fibrillation is initiated by aberrant electrical impulses that arise within regions of heightened atrial excitability (most often the pulmonary veins). Once triggered, atrial fibrillation induces electrical remodeling of the atria with the development of shortened refractory periods and increased conductivity. This facilitates the creation and

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Explanation

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Atrial fibrillation is initiated by aberrant electrical impulses that arise within regions of heightened atrial excitability (most often the pulmonary veins). Once triggered, atrial fibrillation induces electrical remodeling of the atria with the development of shortened refractory periods and increased conductivity. This facilitates the creation and persistence of multiple ectopic foci and reentrant impulses within the atria, increasing the risk and chronicity of subsequent episodes (atrial fibrillation begets atrial fibrillation).

Ventricular response in atrial fibrillation is dependent on the transmission rate of abnormal atrial impulses through the **atrioventricular (AV) node**. Each time the AV node is excited, it enters a **refractory period** during which additional atrial impulses cannot be transmitted to the ventricles; consequently, the majority of atrial impulses never reach the ventricles **(Choice A)**. The average ventricular rate in atrial fibrillation usually ranges between 90-170/min. Because atrial excitation is chaotic, the ventricular rate is irregular with no set intervals between contractions.

(Choice C) Bundle branch conductivity determines the duration of the QRS complexes, which are widened when there is bundle branch block. It does not affect the ventricular contraction rate.

(Choice D) The Purkinje system can assume pacemaker activity in patients with severe bradycardia (<40 beats per minute), but it would not generate impulses in atrial fibrillation since the ventricular rate is typically elevated.

(Choice E) During atrial fibrillation, the atria are in a state of chaotic, continuous depolarization that is independent of sinoatrial node activity.

(Choice F) The ventricular muscle refractory period does not limit the ventricular contraction rate during AF (the

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(Choice E) During atrial fibrillation, the atria are in a state of chaotic, continuous depolarization that is independent of sinoatrial node activity.

(Choice F) The ventricular muscle refractory period does not limit the ventricular contraction rate during AF (the ventricular heart rate can approach 300/min in ventricular tachycardia).

Educational objective:
Atrial fibrillation occurs due to irregular, chaotic electrical activity within the atria and presents with absent P waves, irregularly irregular R-R intervals, and narrow QRS complexes. The atrioventricular node refractory period regulates the number of atrial impulses that reach the ventricles and determines the ventricular contraction rate in conditions where the atria undergo rapid depolarization.

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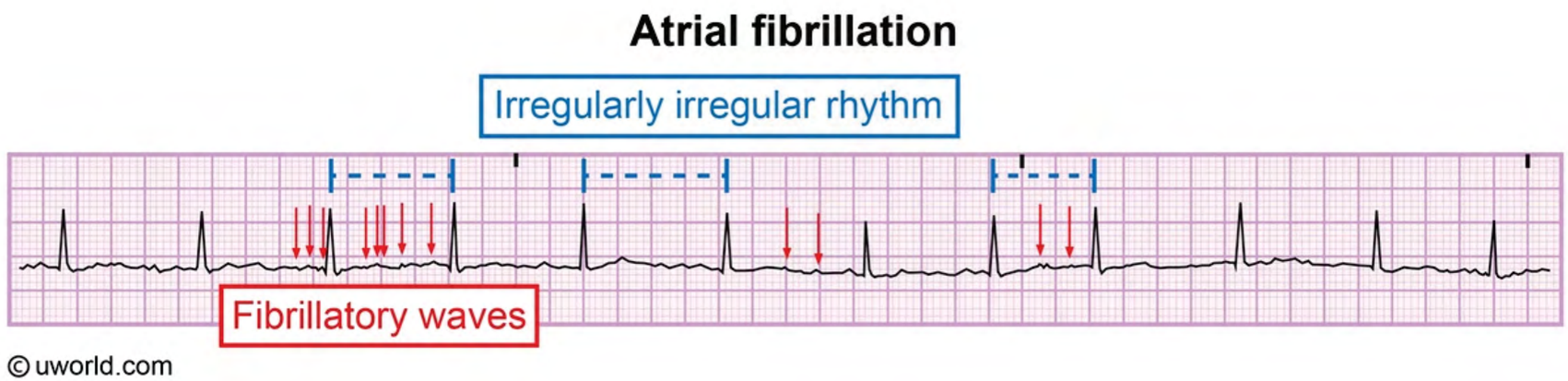

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A 35-year-old man is evaluated for progressive fatigue and shortness of breath. Recently, he has noticed bilateral leg swelling and abdominal distension despite overall weight loss. He does not use tobacco, alcohol, or illicit drugs. Despite treatment, the patient dies several weeks later. Autopsy reveals significant endocardial thickening with dense fibrous deposits around the tricuspid and pulmonary valves as well as moderate pulmonary valve stenosis. The left-sided cardiac chambers and valves are normal. Measuring the levels of which of the following substances would have helped in diagnosing this patient?

- A. Plasma cortisol
- B. Plasma homocysteine
- C. Plasma phenylalanine
- D. Urinary 5-hydroxyindoleacetic acid
- E. Urinary porphobilinogen
- F. Urinary vanillylmandelic acid

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- A. Plasma cortisol (6%)
- ✗ B. Plasma homocysteine (17%)
- C. Plasma phenylalanine (2%)
- ✓ D. Urinary 5-hydroxyindoleacetic acid (57%)
- E. Urinary porphobilinogen (6%)
- F. Urinary vanillylmandelic acid (9%)

Incorrect
Correct answer
D

57%
Answered correctly

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Explanation

Features of carcinoid syndrome	
Clinical manifestations	• Skin: flushing, telangiectasias, cyanosis • Gastrointestinal: watery diarrhea, cramping • Pulmonary: bronchospasm, dyspnea, wheezing

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Features of carcinoid syndrome	
Clinical manifestations	• Skin: flushing, telangiectasias, cyanosis • Gastrointestinal: watery diarrhea, cramping • Pulmonary: bronchospasm, dyspnea, wheezing • Cardiac: valvular fibrous plaques (right > left)
Diagnosis	• Elevated 24-hr urinary excretion of 5-HIAA • CT scan/MRI of abdomen & pelvis to localize tumor
Treatment	• Octreotide for symptomatic patients • Surgery for liver metastases
5-HIAA = 5-hydroxyindoleacetic acid.	

The autopsy findings - endocardial thickening and fibrosis of tricuspid and pulmonary valves - are characteristic of **carcinoid heart disease**. Carcinoids are well-differentiated neuroendocrine tumors found most commonly in the distal small intestine and proximal colon, with a strong propensity for metastasis to the liver. These tumors secrete several products (including histamine, serotonin, and vasoactive intestinal peptide) that are metabolized in the liver. In patients with liver metastasis, these hormones are released directly into the systemic circulation, leading to carcinoid syndrome.

Carcinoid heart disease is caused by excessive secretion of **serotonin**, which stimulates fibroblast growth and fibrogenesis. Pathognomonic plaque-like **deposits of fibrous tissue** occur most commonly on the **endocardium**, leading to **tricuspid regurgitation**, pulmonic valvulopathy, and right-sided heart failure (eg, ascites, peripheral edema). Endocardial fibrosis and thickening are generally limited to the right heart as vasoactive products are inactivated distally by pulmonary vascular endothelial monoamine oxidase.

5-hydroxyindoleacetic acid (5-HIAA) is an end product of serotonin metabolism, and elevated 24-hour urinary 5-

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5-hydroxyindoleacetic acid (5-HIAA) is an end product of serotonin metabolism, and elevated 24-hour urinary 5-HIAA levels are helpful in diagnosing suspected carcinoid syndrome.

(Choice A) Measurement of plasma cortisol levels is used for diagnosing adrenal insufficiency and Cushing syndrome.

(Choice B) Inherited or acquired elevation of plasma homocysteine levels may contribute to arterial and venous thrombosis and to the development of atherosclerosis.

(Choice C) Plasma phenylalanine may be elevated in patients with phenylalanine hydroxylase deficiency (phenylketonuria), resulting in central nervous system damage/intellectual disability.

(Choice E) Urinary porphobilinogen may be elevated in the porphyrias, which are caused by deficiencies of the heme synthesis enzymes. The porphyrias may produce cutaneous lesions, skin photosensitivity, or attacks of abdominal pain and neurological disturbances (acute intermittent porphyria).

(Choice F) Vanillylmandelic acid is a byproduct of norepinephrine and epinephrine, and can be used to detect neuroblastoma and other tumors of neural crest origin.

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Educational objective:
Carcinoid syndrome typically presents with episodic flushing, secretory diarrhea, and wheezing. It can lead to pathognomonic plaque-like deposits of fibrous tissue on the right-sided endocardium, causing tricuspid regurgitation and right-sided heart failure. Elevated 24-hour urinary 5-hydroxyindoleacetic acid can confirm the diagnosis.

References

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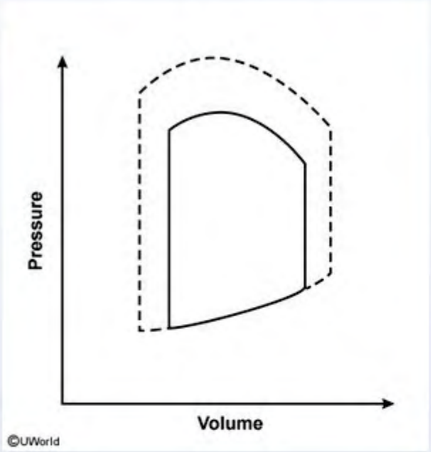
Researchers conduct an experiment to determine hemodynamic changes during exercise. Invasive monitoring of various parameters is performed to obtain a left ventricular pressure-volume loop. Compared to at rest, which of the following changes is most likely to be observed during moderate to heavy exercise?

(solid line: at rest; dashed line: during exercise)

A.



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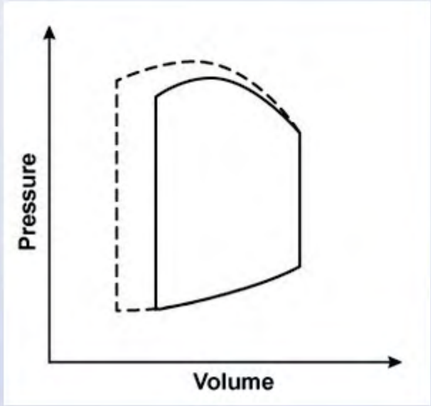

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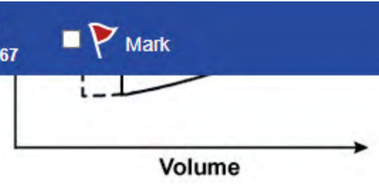

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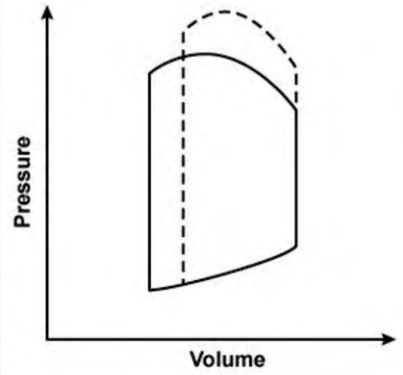

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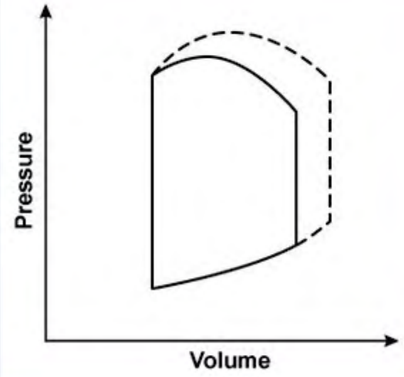

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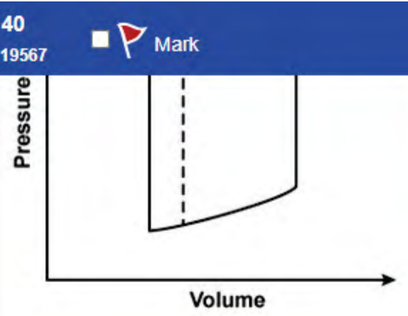

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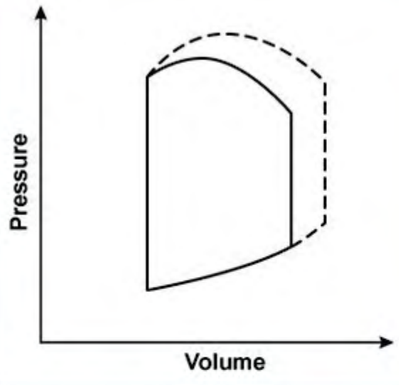

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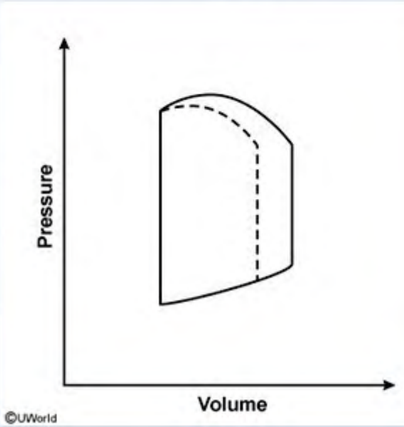

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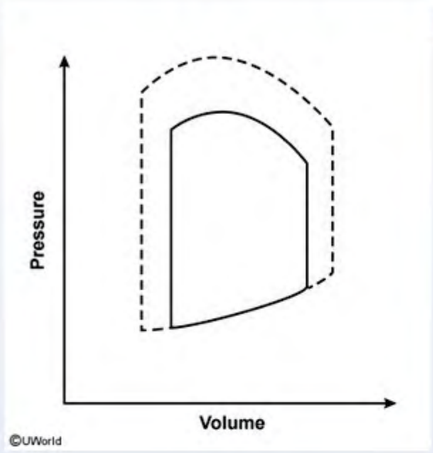
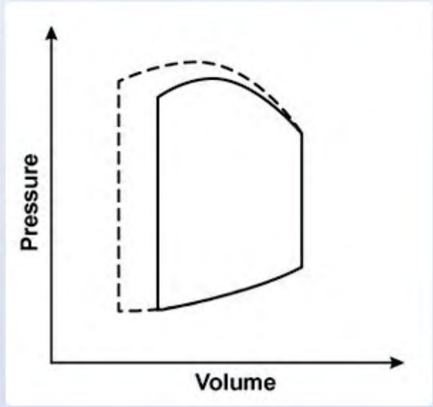
various parameters is performed to obtain a left ventricular pressure-volume loop. Compared to at rest, which of the following changes is most likely to be observed during moderate to heavy exercise?

(solid line: at rest; dashed line: during exercise)

✓ A. (33%)



B. (29%)



C. (10%)

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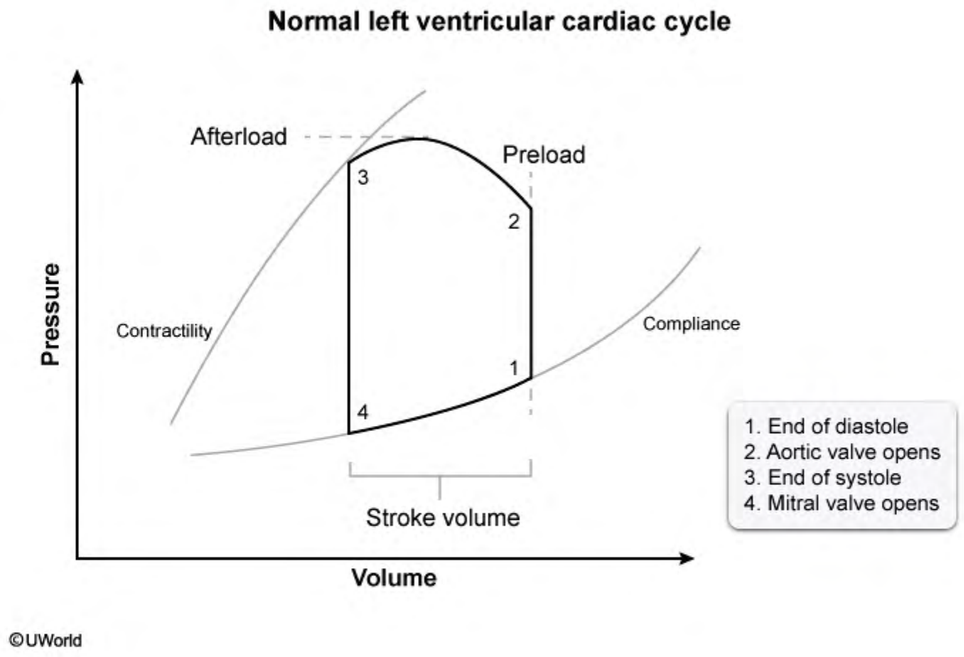

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During moderate to heavy **exercise**, several **cardiovascular changes** occur to facilitate adequate oxygen delivery to skeletal muscle. These changes and their effects on the left ventricular (LV) pressure-volume loop are as follows:

- Vasoconstriction of capacitance veins mobilizes blood from the legs and increases venous return (ie, **increased preload**), which shifts LV end-diastolic volume to the right. There is also vasoconstriction of the splanchnic circulation and a higher degree of vasodilation in skeletal muscle, which shunts blood away from the gastrointestinal tract toward skeletal muscle and causes overall **reduced systemic vascular resistance** (SVR). The reduced SVR also contributes to increased preload by allowing cardiac output to return to the right atrium more quickly and easily.
- **Myocardial contractility** is markedly **increased**, shifting point 3 up and to the left. Both the increased contractility and the increased preload contribute to **increased stroke volume** (ie, the horizontal distance between points 1 and 3 is increased).
- Although reduced SVR typically leads to a slight decrease in diastolic pressure during exercise, the increase in myocardial contractility and stroke volume causes systolic pressure to rise. This results in an overall **increase in afterload** due to increased peak LV and aortic pressure during LV contraction.

The increase in stroke volume, combined with an increase in heart rate, **increases cardiac output** to meet the metabolic demands of exercise.

(Choice B) This graph shows an increase in contractility that leads to increased stroke volume and a slight increase in afterload. Such changes are expected with a positive inotropic agent such as digoxin.

(Choice C) This graph shows an increase in afterload with a consequent reduction in stroke volume (end-systolic volume is increased). Such changes are expected with the obstruction to LV outflow that occurs with aortic stenosis.

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(Choice C) This graph shows an increase in afterload with a consequent reduction in stroke volume (end-systolic volume is increased). Such changes are expected with the obstruction to LV outflow that occurs with aortic stenosis.

(Choice D) This graph shows an increase in preload (end-diastolic volume) with a consequent increase in stroke volume and slight increase in afterload. Such changes are expected with the increase in intravascular volume that occurs with the infusion of normal saline.

(Choice E) This graph shows decreased preload with a consequent decrease in stroke volume and slight reduction in afterload. Such changes are expected with the selective venous vasodilation that occurs with nitroglycerin.

Educational objective:
With moderate to heavy exercise, the changes to the left ventricular pressure-volume loop include increased preload and increased contractility that both contribute to increased stroke volume. Although reduced systemic vascular resistance decreases afterload, the increase in systolic pressure caused by increased preload and contractility override the decrease, resulting in overall increased afterload.

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1. End of diastole

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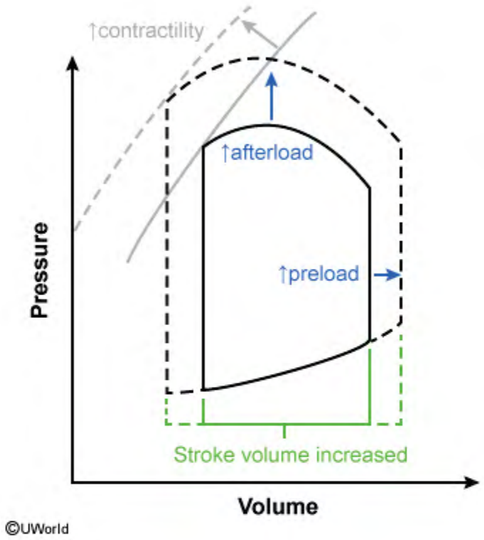

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During moderate to heavy **exercise**, several **cardiovascular changes** occur to facilitate adequate oxygen delivery to skeletal muscle. These changes and their effects on the left ventricular (LV) pressure-volume loop are as follows:

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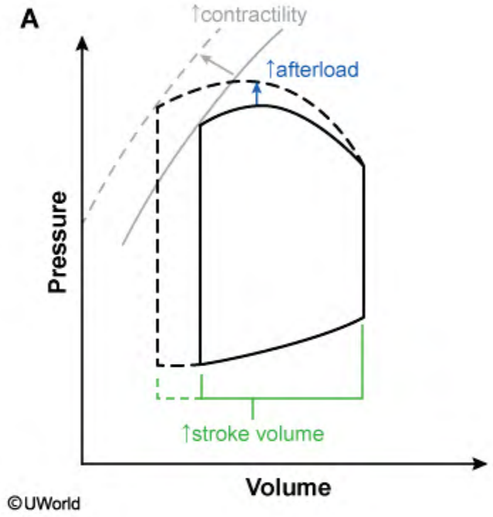

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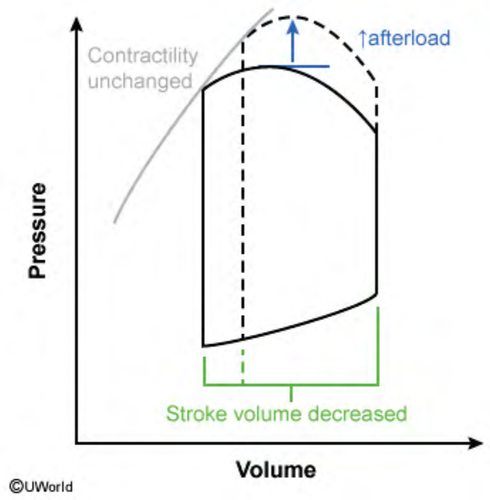

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- Vasoconstriction of capacitance veins mobilizes blood from the legs and increases venous return (ie, **increased preload**), which shifts LV end-diastolic volume to the right. There is also vasoconstriction of the

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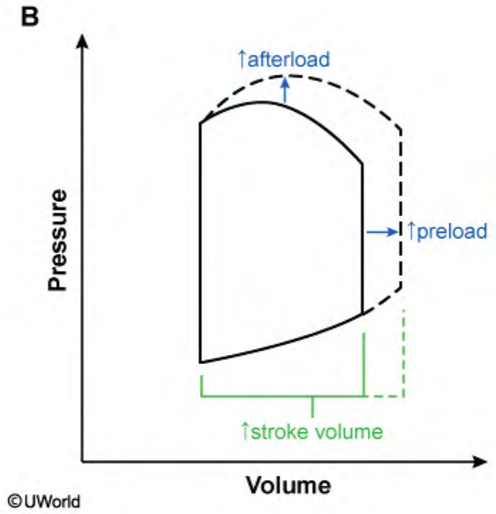

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reduction in afterload. Such changes are expected with the selective venous vasodilation that occurs with nitroglycerin.

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(SVR). The reduced SVR also contributes to increased preload by allowing cardiac output to return to the ri

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With moderate to heavy exercise, the changes to the left ventricular pressure-volume loop include increased preload and increased contractility that both contribute to increased stroke volume. Although reduced systemic

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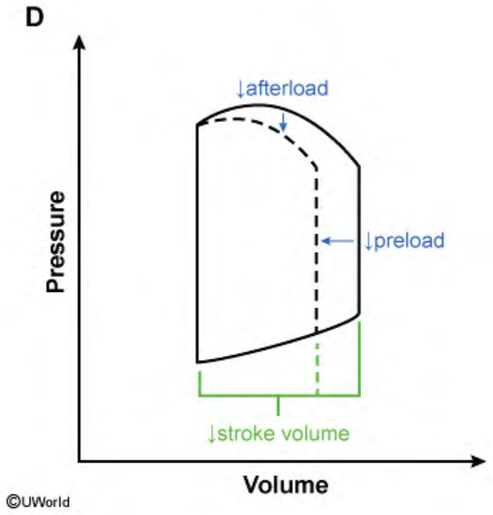

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A 62-year-old man is hospitalized for chest pain and palpitations. His past medical history is significant for coronary artery disease, ischemic cardiomyopathy, paroxysmal atrial fibrillation, and hypertension. Blood pressure on admission is 135/78 mm Hg and pulse is 78/min. During hospitalization, the patient has 2 episodes of brief loss of consciousness. ECG recording during the episodes shows polymorphic QRS complexes that change in amplitude and cycle length. Between the episodes, his ECG shows QT interval prolongation but is otherwise unremarkable. Which of the following medications is most likely responsible for this patient's condition?

- A. Digoxin
- B. Diltiazem
- C. Lidocaine
- D. Metoprolol
- E. Sotalol

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- ✗ A. Digoxin (16%)
- B. Diltiazem (8%)
- C. Lidocaine (10%)
- D. Metoprolol (2%)
- ✓ E. Sotalol (62%)

Incorrect
Correct answer
E

62%
Answered correctly

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Explanation

This patient's presentation (syncope and ECG findings) is suggestive of **long QT syndrome** with associated torsades de pointes (TdP, "twisting of points"). TdP is a form of polymorphic ventricular tachycardia characterized by QRS complexes of varying amplitude and cycle length giving the appearance that the tip of the QRS complex is "twisting" around the ECG baseline. TdP is differentiated from other forms of polymorphic ventricular tachycardia because it is always associated with a prolonged QT interval. It may terminate spontaneously or can degenerate into ventricular fibrillation and sudden cardiac death.

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QT prolongation may be acquired or congenital. **Acquired QT prolongation** is most frequently caused by electrolyte imbalances (hypokalemia, hypomagnesemia) and pharmacologic agents such as **class IA and III antiarrhythmics** (eg, quinidine, sotalol), antibiotics (eg, macrolides, fluoroquinolones), methadone, and antipsychotics (eg, haloperidol). Sotalol is a class III antiarrhythmic agent (K^+ channel blocking) used for treatment of atrial fibrillation. It prolongs action potential duration, resulting in QT interval prolongation.

(Choice A) Digoxin slows conduction through the atrioventricular node by augmenting vagal parasympathetic tone, and it increases cardiac contractility by inhibiting Na^+/K^+-ATPase (increasing intracellular calcium). It also decreases the action potential duration and can cause QT interval shortening.

(Choices B and D) Diltiazem (nondihydropyridine calcium channel blocker) and metoprolol (beta-1 selective antagonist) slow the sinus node discharge rate and atrioventricular nodal conduction. These agents prolong the PR interval but do not affect the QT interval.

(Choice C) Lidocaine is a class IB antiarrhythmic drug that acts by blocking sodium channels and inhibiting phase 0 depolarization. It does not prolong the QT interval.

Educational objective:
Torsades de pointes (TdP) refers to polymorphic ventricular tachycardia that occurs in the setting of a congenital or acquired prolonged QT interval. TdP is most commonly precipitated by medications that prolong the QT interval such as certain antiarrhythmics (sotalol, quinidine), antipsychotics (haloperidol), and antibiotics (macrolides, fluoroquinolones).

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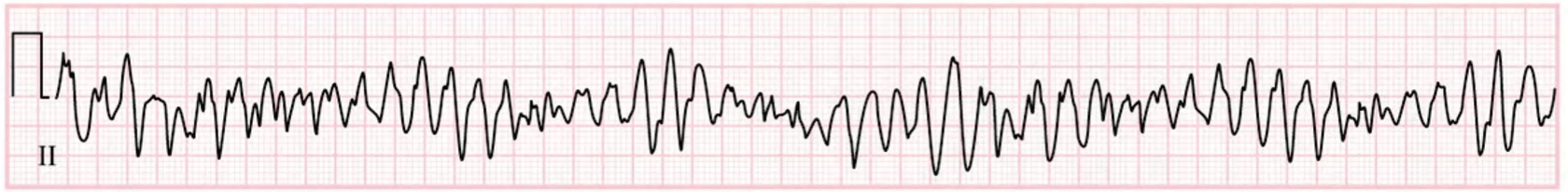

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A 72-year-old man with long-standing dyspnea was seen in the clinic after experiencing an episode of syncope. Physical examination showed weak and slowly rising arterial pulses. Cardiac auscultation showed a harsh midsystolic murmur best heard at the second right intercostal space with decreased intensity of the second heart sound. Electrocardiogram and echocardiogram confirmed the diagnosis of severe aortic stenosis. Two months later, the patient comes to the emergency department with palpitations and increased shortness of breath. His blood pressure is 90/60 mm Hg and his heart rate is 130/min with an irregularly irregular rhythm. Electrocardiogram shows new-onset atrial fibrillation without significant ST-segment or T-wave changes. Chest x-ray shows bilateral pulmonary edema. Which of the following hemodynamic changes is most likely associated with this patient's current presentation?

- A. Insidious right ventricular failure
- B. Sudden decrease in left ventricular preload
- C. Sudden decrease in left ventricular systolic function
- D. Sudden increase in left ventricular afterload
- E. Sudden increase in left ventricular filling

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- ✗ A. Insidious right ventricular failure (10%)
- ✓ B. Sudden decrease in left ventricular preload (28%)
- C. Sudden decrease in left ventricular systolic function (39%)
- D. Sudden increase in left ventricular afterload (15%)
- E. Sudden increase in left ventricular filling (5%)

Incorrect
Correct answer
B

28%
Answered correctly

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Explanation

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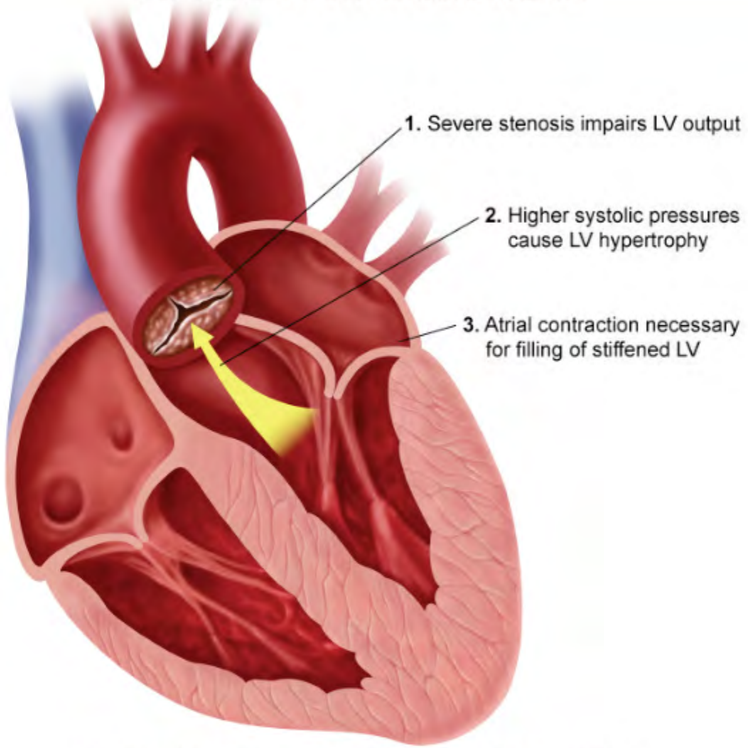
Hemodynamics of severe aortic stenosis



Atrial fibrillation: Loss of contraction reduces LV filling, causing hypotension & pulmonary edema

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This patient's sudden-onset heart failure was most likely precipitated by acute **atrial fibrillation**, a condition that occurs in up to 10% of patients with **severe aortic stenosis** (AS). Patients with severe AS already have reduced cardiac output due to significant valvular obstruction, which can be exacerbated by the sudden **loss of normal atrial contraction** that contributes to ventricular filling. Atrial contraction is especially important for these patients as many have concentric left ventricular (LV) hypertrophy and therefore reduced LV compliance. As a result, they become dependent on atrial contraction to maintain adequate LV filling. Without atrial contraction, LV preload can decrease to the point of producing severe **hypotension**. In addition, loss of the atrial kick can result in significantly increased mean pulmonary venous pressure due to buildup of blood in the left atrium and pulmonary veins, leading to **acute pulmonary edema**. As a result, cardioversion is indicated for acute atrial fibrillation in patients with severe AS.

(Choice A) Cardiogenic pulmonary edema is characteristic of left, not right, heart failure. When the forward pumping function of the left heart is impaired, pressure can build up in the left atrium. This rise in pressure is transmitted to the pulmonary veins and capillaries, resulting in increased fluid transudation into the alveoli and pulmonary interstitium.

(Choices C and E) This patient has no evidence of myocardial ischemia on electrocardiogram, meaning that LV systolic function is most likely unchanged. Under these circumstances, an increase in LV filling (preload) would increase cardiac output and mean arterial pressure due to the Frank-Starling mechanism. This patient's decreased cardiac output is most likely due to decreased LV preload.

(Choice D) In a patient with AS, LV afterload is determined by the mean systolic blood pressure and the degree of transvalvular obstruction. In degenerative calcific AS and most other forms of adult AS, the transvalvular obstruction gradually increases over years to decades, making an acute increase in LV afterload unlikely. In addition, this patient's acute hypotension would actually cause a reduction in afterload.

Educational objective:
In patients with chronic aortic stenosis and concentric left ventricular hypertrophy, atrial contraction contributes significantly to left ventricular filling. Loss of atrial contraction due to atrial fibrillation can reduce left ventricular

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Educational objective:
In patients with chronic aortic stenosis and concentric left ventricular hypertrophy, atrial contraction contributes significantly to left ventricular filling. Loss of atrial contraction due to atrial fibrillation can reduce left ventricular preload and cardiac output sufficiently to cause systemic hypotension. Decreased forward filling of the left ventricle can also result in backup of blood in the left atrium and pulmonary veins, leading to acute pulmonary edema.

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A 10-year-old boy is brought to the physician by his parents due to restlessness and involuntary jerking. He takes no medications and his vaccinations are up-to-date. His parents do not recall any recent injuries or illnesses other than a sore throat 3 months ago. On examination, the patient has rapid, irregular jerking movements involving his face, arms, and legs. This patient is at greatest risk for developing which of the following conditions?

- A. Deforming polyarthritis
- B. Early dementia
- C. Parkinson disease
- D. Renal failure
- E. Valvular heart disease

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- A. Deforming polyarthritis (4%)
- ✗ B. Early dementia (9%)
- C. Parkinson disease (3%)
- D. Renal failure (8%)
- ✓ E. Valvular heart disease (75%)

Incorrect
Correct answer
E

75%
Answered correctly

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Explanation

Acute rheumatic fever	
Epidemiology	• Endemic in developing countries
Pathogenesis	• Occurs 2-4 weeks after acute group A streptococcal pharyngitis • Molecular mimicry: Anti-streptococcal antibodies attack cardiac & neuronal antigens
Clinical features	• Acute/subacute ◦ Migratory arthritis ◦ Pancarditis (mitral regurgitation)

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Clinical features	• Acute/subacute
	  ◦ Migratory arthritis
	  ◦ Pancarditis (mitral regurgitation)
	  ◦ Sydenham chorea
	• Chronic
	  ◦ Mitral stenosis

Prevention	• Prompt treatment of streptococcal pharyngitis with penicillin

This patient's **restlessness and purposeless jerking movements** 3 months after having a **sore throat** are consistent with **Sydenham chorea**, a hyperkinetic extrapyramidal movement disorder that is the most common acquired chorea of childhood. Sydenham chorea is a neurologic manifestation of **acute rheumatic fever** that occurs 1-8 months after group A β-hemolytic streptococcal infection. It is caused by a delayed onset autoimmune reaction involving anti-streptococcal antibodies that cross-react with the **basal ganglia**. Because Sydenham chorea is a manifestation of acute rheumatic fever, patients with this disorder are at risk for **chronic rheumatic heart disease**.

(Choice A) Although migratory polyarthritis is an early manifestation of acute rheumatic fever, it is generally transient and rarely, if ever, deforming.

(Choice B) Early-onset Alzheimer dementia is associated with Down syndrome (trisomy 21). Increased expression of the amyloid precursor protein gene located on chromosome 21 is thought to be the cause.

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Prevention	• Prompt treatment of streptococcal pharyngitis with penicillin

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(Choice B) Early-onset Alzheimer dementia is associated with Down syndrome (trisomy 21). Increased expression of the amyloid precursor protein gene located on chromosome 21 is thought to be the cause.

(Choice C) Parkinson disease is an extrapyramidal hypokinetic movement disorder consisting of tremor, rigidity, akinesia, and postural instability. Jerking extremity movements are generally not seen in Parkinson disease, except in cases of levodopa overdose.

(Choice D) Poststreptococcal glomerulonephritis occurs following infection with specific nephritogenic strains of group A *Streptococcus*. However, the nephritis manifests within 1-6 weeks and is typically self-limited. Renal failure is not part of the clinical syndrome associated with acute rheumatic fever.

Educational objective:
Sydenham chorea presents with involuntary, rapid, irregular jerking movements involving the face, arms, and legs. It occurs months after group A streptococcal infection and is one of the major clinical manifestations of acute rheumatic fever. Patients with this condition carry a high risk of chronic valvular disease.

References

- Diagnostic criteria of acute rheumatic fever.

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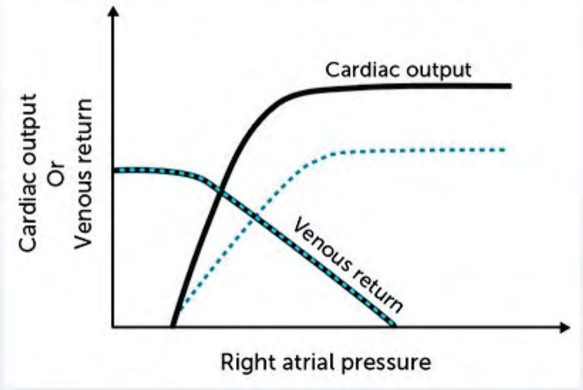
The cardiac output and venous return curves of a healthy person are shown below with solid lines.



Which of the following is the most likely cause of the change depicted by the dashed lines?

- A. Excessive hydration
- B. Acute hemorrhage
- C. Chronic anemia
- D. Myocardial infarction
- E. Anaphylaxis

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The cardiac output and venous return curves of a healthy person are shown below with solid lines

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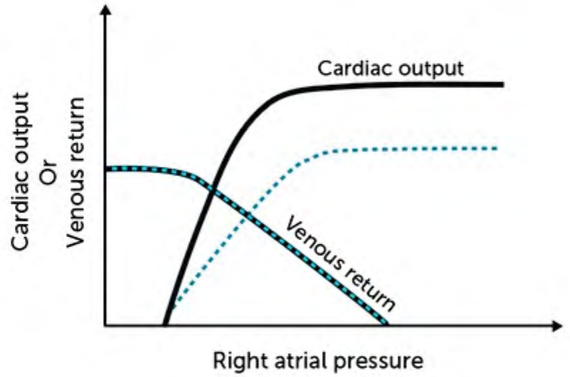

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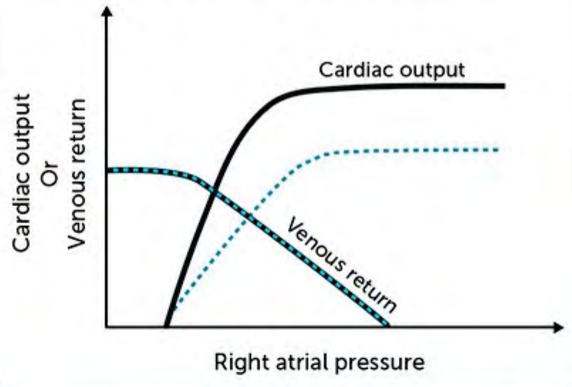

Which of the following is the most likely cause of the change depicted by the dashed lines?

- A. Excessive hydration (2%)
- B. Acute hemorrhage (13%)
- C. Chronic anemia (4%)
- ✓ D. Myocardial infarction (76%)
- ✗ E. Anaphylaxis (3%)

Incorrect
Correct answer
D

76%
Answered correctly

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Explanation

The graph above combines cardiac and vascular function curves. The cardiac function curve, labeled as cardiac

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Explanation

The graph above combines cardiac and vascular function curves. The cardiac function curve, labeled as cardiac output, illustrates the Frank-Starling effect. The Frank-Starling effect states that as cardiac muscle is increasingly stretched, the cardiac output increases (up to a limit, as illustrated by the flat portion of the curve). This is essentially the same length-tension relationship that is seen in skeletal muscle.

The venous return curve represents how venous return changes with variations in right atrial pressure. Where the venous return curve intersects with the x-axis, it indicates a venous return of zero. At this point, all circulatory flow ceases, and the pressure throughout the circulatory system equalizes. This average pressure is termed the mean systemic filling pressure, which is a measure of the degree of filling of the circulatory system relative to the circulatory capacity. Increases in blood volume (such as with excessive hydration, dashed line) shift the x-intercept to the right, while decreased blood volume (acute hemorrhage, dashed line) shifts it to the left **(Choices A and B)**. Total peripheral resistance (TPR) affects the slope of both the venous return and cardiac output curves. Increases in the TPR decrease the slope of both curves, as higher vascular resistance restricts venous return and increases afterload.

The dashed lines in the question image depict decreased cardiac output and an unchanged venous return (unchanged blood volume and TPR). An isolated decrease in cardiac output indicates decreased contractility that is not the result of decreased preload (because the venous return line is unchanged). This indicates inhibited contraction either from the action of a negative inotropic drug or an injury to the myocardium, such as a myocardial infarction.

(Choice C) Chronic anemia causes an increase in cardiac output in an effort to meet the metabolic demands of the tissues. This causes an increase in the slope and height of the cardiac output graph. Venous return also increases somewhat due to decreased blood viscosity.

(Choice E) Anaphylaxis causes widespread venous and arteriolar dilation along with increased capillary permeability and third-spacing of fluids. This results in a serious drop in venous return (shifted down and

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(Choice E) Anaphylaxis causes widespread venous and arteriolar dilation along with increased capillary permeability and third-spacing of fluids. This results in a serious drop in venous return (shifted down and leftward). Cardiac contractility also increase as the body attempts to maintain blood pressure.

Educational objective:
Myocardial infarction causes a sharp decrease in cardiac output due to loss of function of a zone of myocardium. On a cardiac function curve, myocardial infarction would decrease both the slope and the maximal height of the line.

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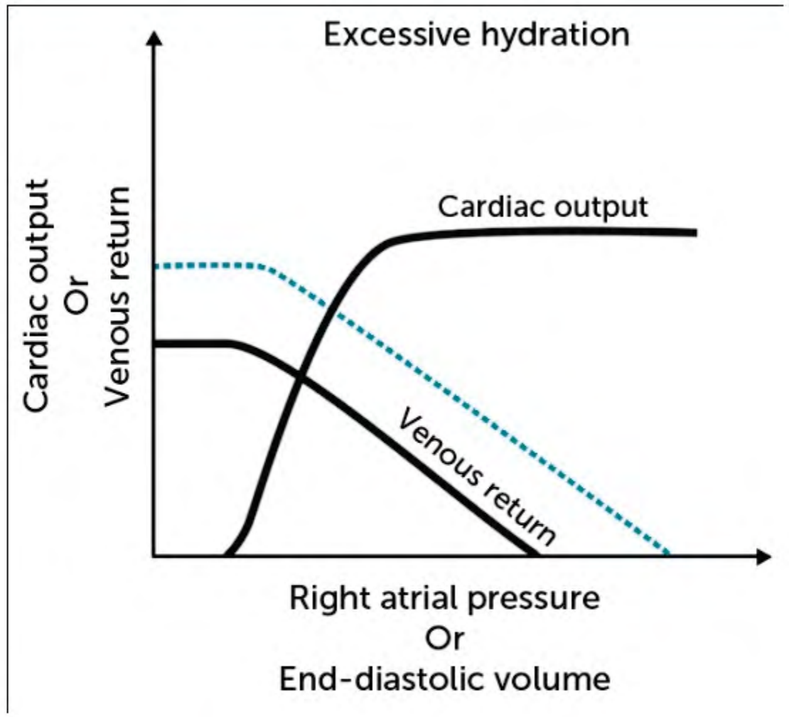

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(Choice E) Anaphylaxis causes widespread venous and arteriolar dilation along with increased capillary

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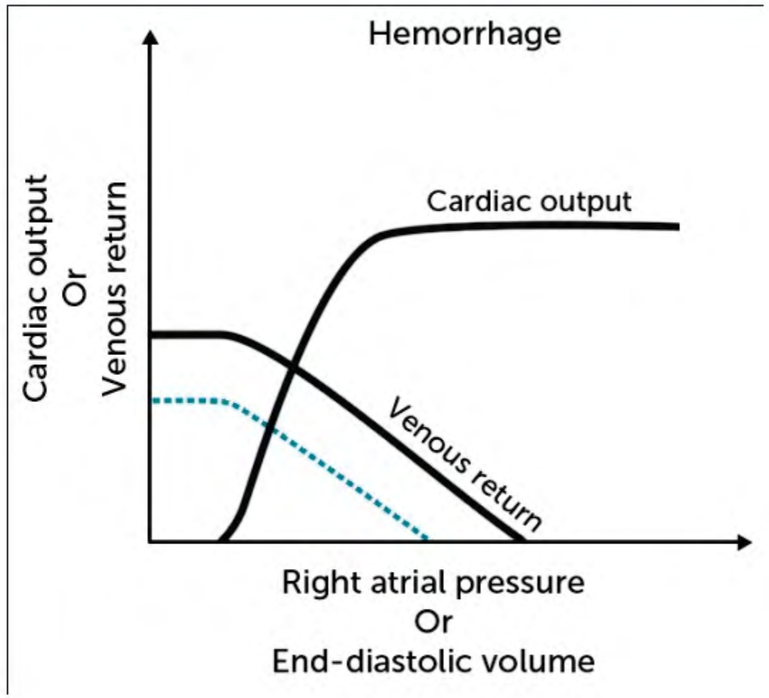

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The graph above combines cardiac and vascular function curves. The cardiac function curve, labeled as cardiac

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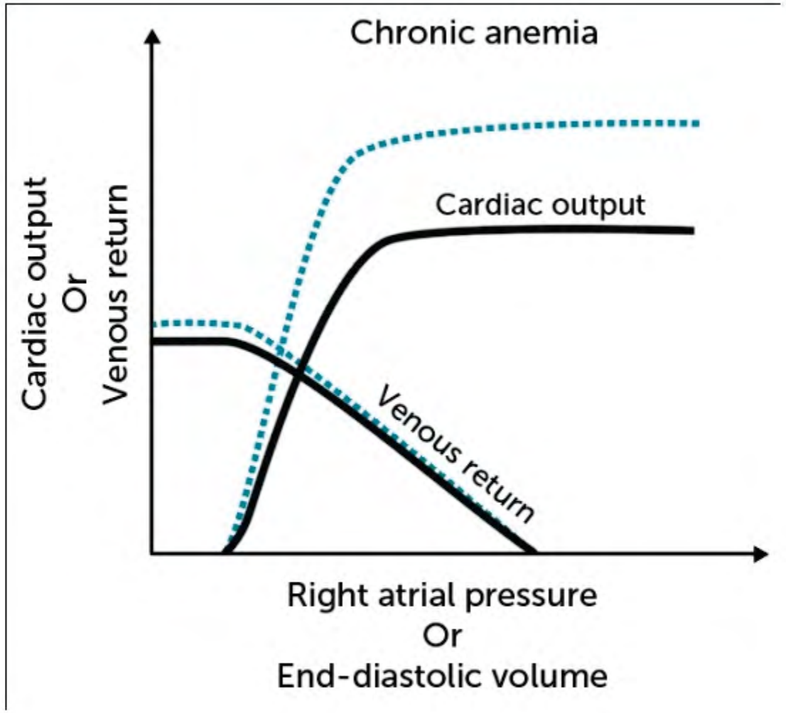

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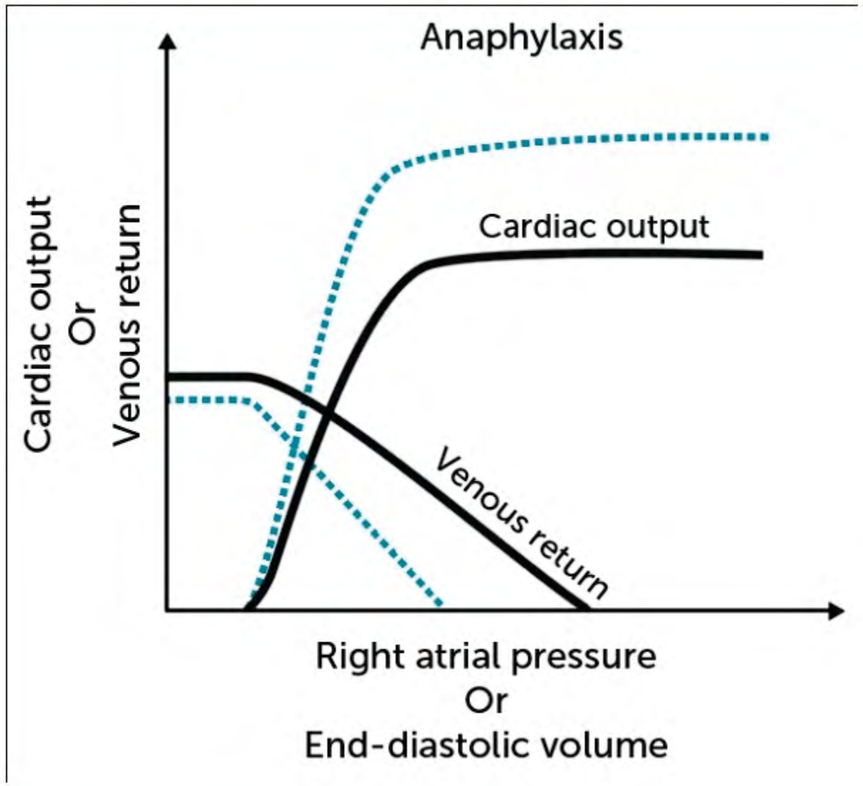

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A 53-year-old man comes to the office with progressive exertional dyspnea. He has smoked 2 packs of cigarettes per day for the last 35 years. Physical examination shows increased anteroposterior diameter of his chest. Auscultation reveals decreased breath sounds and scattered wheezes throughout his lungs. Examination of his extremities is unremarkable. Echocardiography reveals moderate dilation of the right ventricle and increased central venous pressure. The absence of peripheral edema in this patient is best explained by which of the following compensatory mechanisms?

- A. Decreased capillary permeability
- B. Decreased circulating aldosterone levels
- C. Decreased interstitial fluid pressure
- D. Increased plasma oncotic pressure
- E. Increased tissue lymphatic drainage

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- ✗ A. Decreased capillary permeability (12%)
- B. Decreased circulating aldosterone levels (25%)
- C. Decreased interstitial fluid pressure (8%)
- D. Increased plasma oncotic pressure (22%)
- ✓ E. Increased tissue lymphatic drainage (31%)

Incorrect
Correct answer
E

31%
Answered correctly

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Explanation

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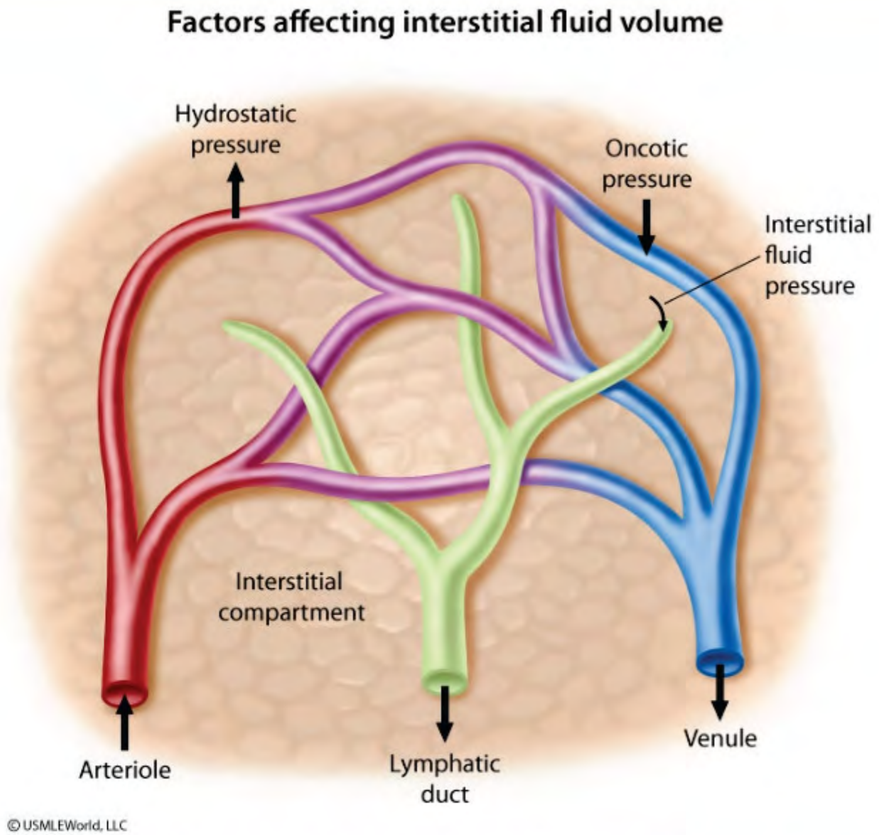

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This patient has dyspnea, signs of lung hyperinflation, and a heavy smoking history, suggesting chronic obstructive pulmonary disease (COPD). Chronic hypoxia in COPD can lead to pulmonary vasoconstriction, increased pulmonary artery pressure, and right heart failure (ie, cor pulmonale), which is confirmed by this patient's echocardiogram. In turn, right heart failure leads to increased central venous pressure and elevated peripheral capillary hydrostatic pressure, encouraging the development of **peripheral edema**.

Fluid movement across the capillary wall (**filtration**) into the interstitium is dependent on hydrostatic pressure, oncotic pressure, and capillary permeability. It is tightly **balanced by lymphatic drainage**, which returns interstitial fluid to the vasculature. Peripheral edema (accumulation of excess fluid in the interstitial space) develops when transcapillary plasma filtration exceeds the resorptive capacity of the lymphatics. Factors that favor development of edema include:

- **Elevated capillary hydrostatic pressure:** Higher capillary pressures increase plasma filtration into the interstitium, particularly in dependent tissues. Causes include sodium and water retention (eg, heart failure, renal failure), arteriolar dilation (eg, dihydropyridine calcium channel blockers), and impaired venous return (eg, venous thrombosis).
- **Decreased plasma oncotic pressure:** Plasma proteins (eg, albumin) generate oncotic pressure that pulls interstitial fluid back into the capillary bed venules. Edema develops in conditions with decreased albumin levels (eg, nephrotic syndrome, cirrhosis, malnutrition).
- **Lymphatic obstruction:** Decreased lymphatic return impairs removal of excess interstitial fluid. Common causes of lymphatic obstruction include filariasis, invasive malignancies, and iatrogenic etiologies (eg, surgical lymph node dissection and radiation therapy).

The peripheral lymphatics can significantly increase lymphatic drainage from baseline, allowing compensation for increased capillary fluid filtration that can **temporarily delay** the development of peripheral edema. However,

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levels (eg, nephrotic syndrome, cirrhosis, malnutrition).

- **Lymphatic obstruction:** Decreased lymphatic return impairs removal of excess interstitial fluid. Common causes of lymphatic obstruction include filariasis, invasive malignancies, and iatrogenic etiologies (eg, surgical lymph node dissection and radiation therapy).

The peripheral lymphatics can significantly increase lymphatic drainage from baseline, allowing compensation for increased capillary fluid filtration that can **temporarily delay** the development of peripheral edema. However, because the peripheral lymphatics offload fluid to the central venous system (via the thoracic duct), which in heart failure is already volume overloaded, the overall ability to compensate is limited.

(Choices A and D) Although increased plasma oncotic pressure and decreased capillary permeability oppose edema formation, neither is associated with COPD. Severe COPD may cause polycythemia due to hypoxemia, but erythrocyte concentration does not affect plasma oncotic pressure.

(Choice B) In patients with heart failure, activation of the renin-angiotensin-aldosterone system (RAAS) leads to increased aldosterone levels that cause sodium and water retention (exacerbating edema formation). Volume expansion causes increased atrial/brain natriuretic peptide release, which can partially counteract the effects of RAAS activation; however, aldosterone levels remain elevated.

(Choice C) Decreased interstitial fluid pressure favors flow into the interstitium and would contribute to the development of peripheral edema.

Educational objective:
Peripheral edema results from the accumulation of fluid in the interstitial spaces. Factors that promote edema include elevated capillary hydrostatic pressure, decreased plasma oncotic pressure, and impaired lymphatic drainage. In chronic heart failure, increased lymphatic drainage initially offsets factors favoring edema, temporarily delaying edema development.

Pathophysiology	Cardiovascular System	Cor pulmonale
Subject	System	Topic

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A 64-year-old man comes to the emergency department due to worsening dizziness and shortness of breath for the past several days. The patient was diagnosed with advanced stage lung cancer 6 months ago and is receiving palliative care. The patient has no other medical history. Blood pressure is 85/45 mm Hg, pulse is 122/min and regular, and respirations are 22/min. Pulse oximetry is 94% on room air. There are undulations in blood pressure at different phases of respiration. On physical examination, the point of maximal impulse is not palpable, and heart sounds are distant. Chest x-ray reveals diffuse pulmonary metastatic disease and new enlargement of the cardiac silhouette. Which of the following hemodynamic changes are most likely present in this patient due to his current cardiac condition?

	Central venous pressure	Right atrial cavity size	Left atrial pressure	Stroke volume
○ A.	Decreased	Increased	Normal	Increased
○ B.	Decreased	Normal	Decreased	Decreased
○ C.	Increased	Decreased	Decreased	Increased
○ D.	Increased	Decreased	Increased	Decreased
○ E.	Increased	Increased	Increased	Decreased

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		Central venous pressure	Right atrial cavity size	Left atrial pressure	Stroke volume	
✗	A.	Decreased	Increased	Normal	Increased	(3%)
	B.	Decreased	Normal	Decreased	Decreased	(14%)
	C.	Increased	Decreased	Decreased	Increased	(3%)
✓	D.	Increased	Decreased	Increased	Decreased	(41%)
	E.	Increased	Increased	Increased	Decreased	(37%)

Incorrect
Correct answer
D

41%
Answered correctly

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Explanation

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Explanation

Cardiac tamponade	
Etiology	• Blood in pericardial space (eg, LV rupture, cardiac surgery) • Pericardial effusion (eg, malignancy, infection, uremia)
Clinical signs	• Beck triad: hypotension, JVD, ↓ heart sounds • Pulsus paradoxus: SBP ↓10 mm Hg during inspiration
Diagnosis	• ECG: low-voltage QRS, electrical alternans • Chest x-ray: enlarged cardiac silhouette*, clear lungs • Echocardiogram: right atrial & ventricular collapse, IVC plethora
*Seen with subacute but not acute tamponade. **IVC** = inferior vena cava; **JVD** = jugular venous distension; **LV** = left ventricular; **SBP** = systolic blood pressure.	

This patient's hypotension, distant heart sounds, and new **enlargement of the cardiac silhouette** on chest x-ray in the setting of advanced malignancy are consistent with **cardiac tamponade**. Cardiac tamponade results from an accumulation of fluid within the relatively noncompliant pericardium, leading to an increase in pericardial pressure. The increased pericardial pressure compresses the relatively low-pressure, right-sided heart chambers and restricts diastolic filling of the right side of the heart to cause **obstructive shock**. In addition to Beck's triad (ie, hypotension, distant heart sounds, jugular venous distension), other common signs of tamponade include an absent point of maximum impulse and pulsus paradoxus (ie, >10 mm Hg drop in systolic blood pressure with inspiration).

In tamponade, the compressed **right atrium** is **decreased in size**, and elevated right atrial pressures are transmitted backward to the vena cava, causing **increased central venous pressure**. With decreased right-sided filling, less blood is pumped through the pulmonary circulation to the left atrium and left ventricle, resulting in **decreased stroke volume** and cardiac output (and causing hypotension and shock). Although the volume of

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in the setting of advanced malignancy are consistent with **cardiac tamponade**. Cardiac tamponade results from an accumulation of fluid within the relatively noncompliant pericardium, leading to an increase in pericardial pressure. The increased pericardial pressure compresses the relatively low-pressure, right-sided heart chambers and restricts diastolic filling of the right side of the heart to cause **obstructive shock**. In addition to Beck's triad (ie, hypotension, distant heart sounds, jugular venous distension), other common signs of tamponade include an absent point of maximum impulse and pulsus paradoxus (ie, >10 mm Hg drop in systolic blood pressure with inspiration).

In tamponade, the compressed **right atrium** is **decreased in size**, and elevated right atrial pressures are transmitted backward to the vena cava, causing **increased central venous pressure**. With decreased right-sided filling, less blood is pumped through the pulmonary circulation to the left atrium and left ventricle, resulting in **decreased stroke volume** and cardiac output (and causing hypotension and shock). Although the volume of blood reaching the left atrium is reduced, **left atrial pressure is increased** due to external compression by the excessive pericardial fluid **(Choices A, B, C, and E)**.

As tamponade progresses, diastolic pressures in **all 4 cardiac chambers** (ie, right atrium, right ventricle, left atrium [estimated by pulmonary capillary wedge pressure], left ventricle) increase and **equalize with pericardial pressure** (and with one another), eliminating the normal pressure gradients and stagnating blood flow within the heart.

Educational objective:
Cardiac tamponade involves increased pericardial pressure that restricts diastolic filling of the right-sided heart chambers, leading to obstructive shock with increased central venous pressure and decreased stroke volume and cardiac output. As tamponade progresses, diastolic pressures in all 4 cardiac chambers increase and equalize with pericardial pressure (and with one another), stagnating blood flow within the heart.

Pathophysiology	Cardiovascular System	Cardiac tamponade
Subject	System	Topic

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- ECG: low-voltage QRS, electrical alternans

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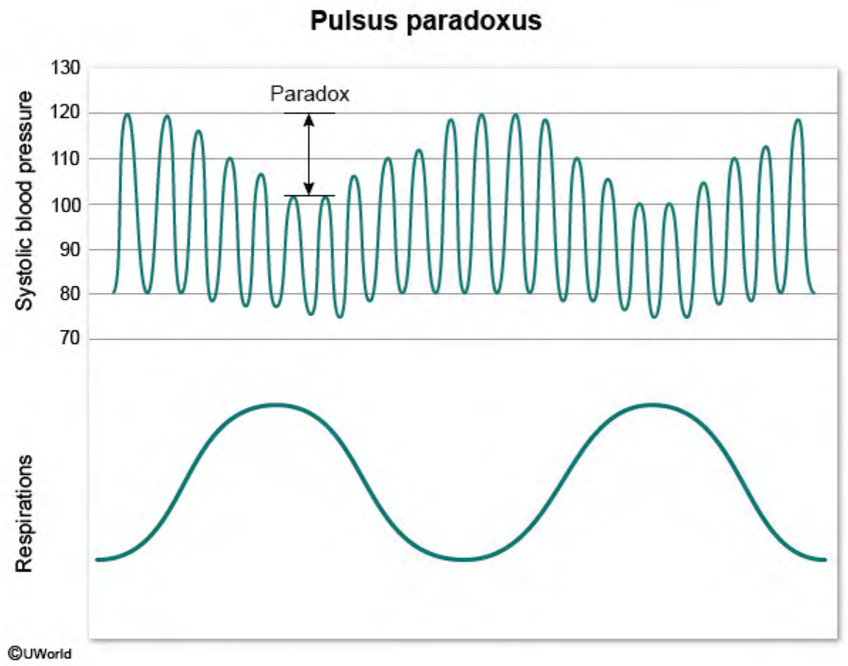

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Cardiac tamponade involves increased pericardial pressure that restricts diastolic filling of the right-sided heart

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- ECG: low-voltage QRS, electrical alternans

Diagnosis

- Chest x-ray: enlarged cardiac silhouette*, clear lungs

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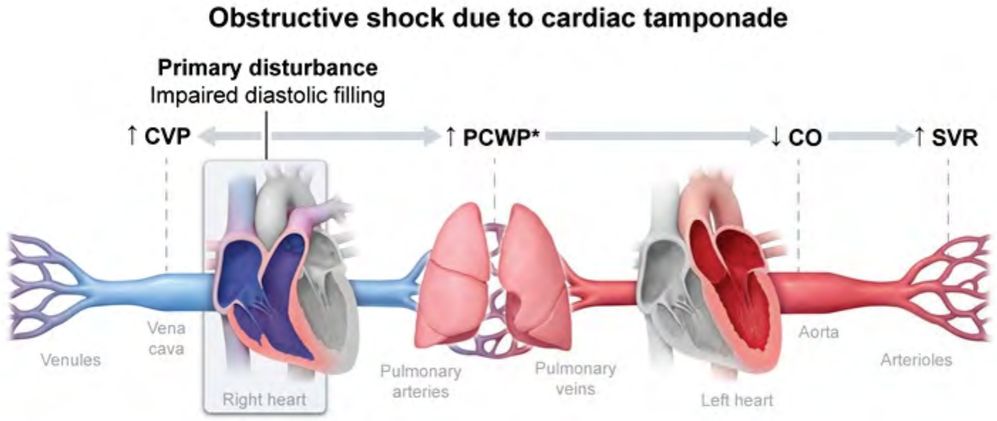

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Cardiac tamponade involves increased pericardial pressure that restricts diastolic filling of the right-sided heart

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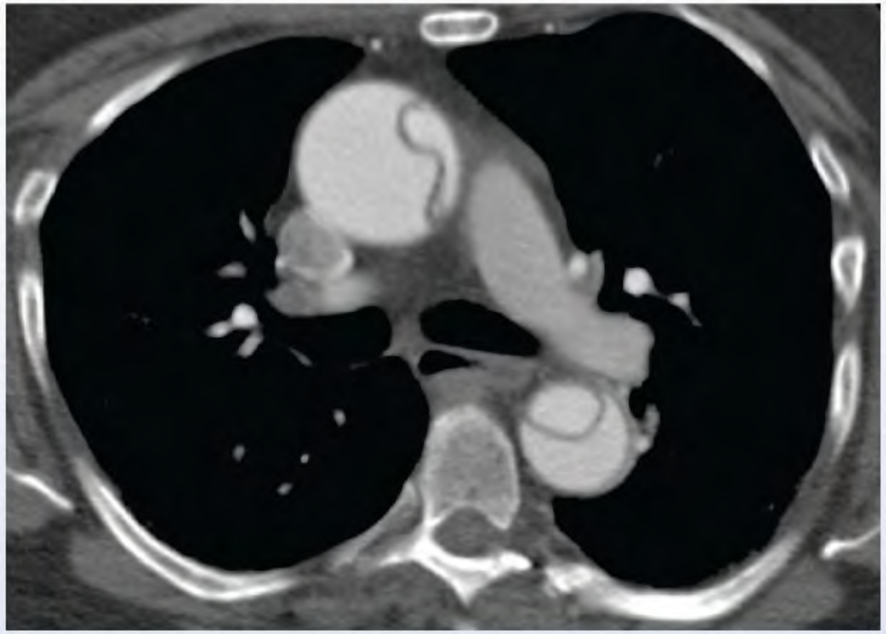
A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within normal limits. Contrast-enhanced computed tomography of his chest is shown below.



Which of the following is the single most important risk factor for this patient's current condition?

- A. Diabetes mellitus
- B. High cholesterol
- C. Hypertension
- D. Limited physical activity
- E. Smoking

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A 56-year-old man comes to the emergency department with acute, severe chest pain. His electrocardiogram is within

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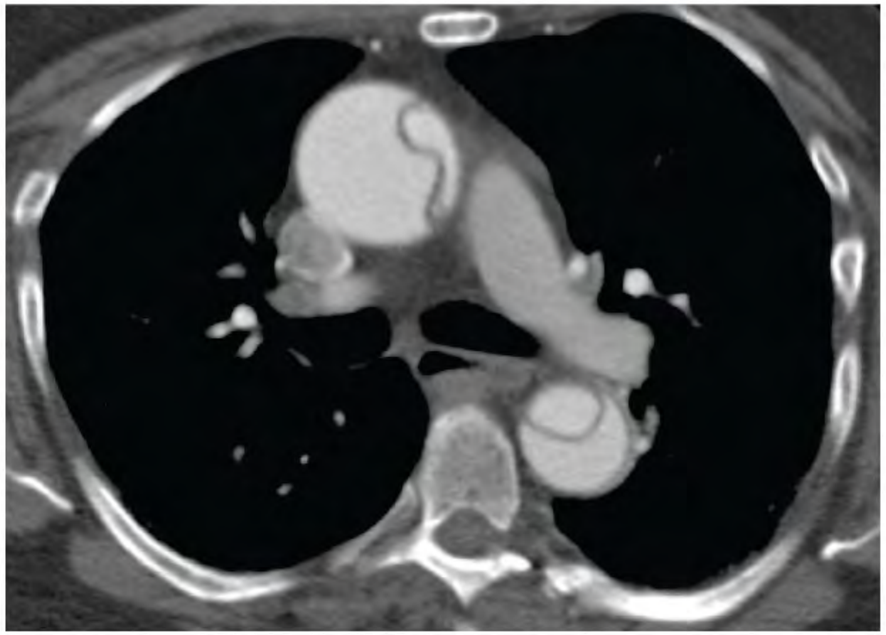

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E. Smoking

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Which of the following is the single most important risk factor for this patient's current condition?

- A. Diabetes mellitus
- B. High cholesterol
- C. Hypertension
- D. Limited physical activity
- E. Smoking
- F. Spirochete infection

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Which of the following is the single most important risk factor for this patient's current condition?

- A. Diabetes mellitus (1%)
- ✗ B. High cholesterol (6%)
- ✓ C. Hypertension (64%)
- D. Limited physical activity (2%)
- E. Smoking (15%)
- F. Spirochete infection (9%)

Incorrect
Correct answer

64%
Answered correctly

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F. Spirochete infection (9%)

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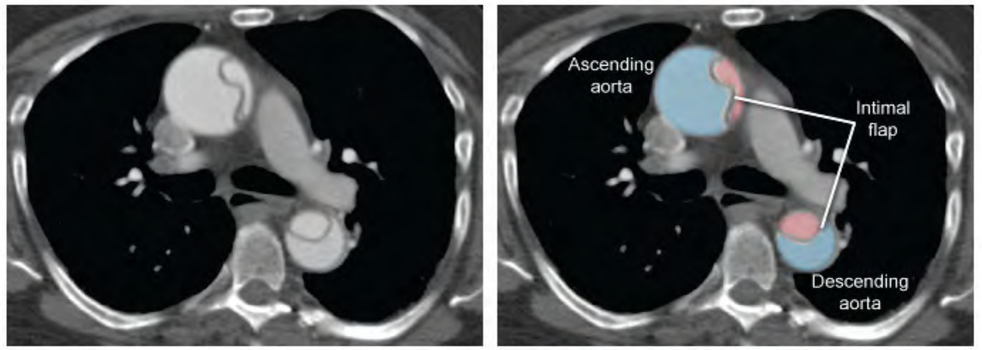
Type A aortic dissection (CT aortography)



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Hyper **dissection.** Cystic medial degeneration, which may be seen in connective tissue diseases such as Marfan

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This patient's chest CT scan reveals a widened, ascending thoracic aorta with a nonenhancing septum dividing the lumen of the ascending and descending aorta. The "septum" is actually the tunica intima of the aorta, which has been torn from the remainder of the aortic wall. A tear in the tunica intima is thought to be the primary event in the process leading to aortic dissection. As more and more blood is forced through the tear, an intramural hematoma begins to develop between the intimal flap and medial wall, creating a false lumen. The dissection can extend both proximally toward the heart and distally, sometimes extending all the way to the iliac and femoral arteries. **Hypertension** is the single most important risk factor for the development of intimal tears leading to **aortic dissection.** Cystic medial degeneration, which may be seen in connective tissue diseases such as Marfan syndrome, also predisposes patients (especially younger ones) to aortic dissection.

(Choice A) Diabetes mellitus is an important risk factor for the development of atherosclerosis and hypertension. It can increase the relative risk of aortic dissection by promoting hypertension, which is the single most important risk factor for aortic dissection.

(Choice B) High cholesterol is a risk factor for atherosclerosis. Atherosclerosis predisposes more to **aortic aneurysm** formation (especially abdominal aortic aneurysms) than to aortic dissection.

(Choice D) A sedentary lifestyle is a risk factor for cardiovascular disease (eg, stroke, myocardial infarction) but does not appear to be directly linked to aortic dissection.

(Choice E) Smoking contributes significantly to the risk of atherosclerosis and thromboangiitis obliterans (Buerger disease). Thromboangiitis obliterans is a vasculitis of medium and small arteries, principally the tibial and radial arteries.

(Choice F) Tertiary syphilis can result in spirochetal endarteritis that affects the vasa vasorum within the adventitia of the ascending thoracic aorta. This weakens the aortic wall and predisposes infected patients to ascending aortic aneurysms. Aortic dissection is only rarely caused by syphilitic aneurysms.

Educational objective:

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Educational objective:
Hypertension is the single most important risk factor for the development of intimal tears leading to aortic dissection. Hypertension, smoking, diabetes mellitus, and hypercholesterolemia are all major risk factors for atherosclerosis, which predisposes more to aortic aneurysm formation than aortic dissection.

Pathology	Cardiovascular System	Aortic dissection
Subject	System	Topic

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A 67-year-old man has recurrent witnessed episodes of loss of consciousness while shaving. The episodes are characterized by a feeling of faintness followed by loss of consciousness, and each episode resolves after less than a minute. Blood pressure is 130/80 mm Hg and pulse is 80/min and regular; there are no significant changes in blood pressure or heart rate with supine and standing positions. Cardiac auscultation is normal with no murmurs or extra sounds. There are no neck bruits and the lungs are clear to auscultation. Which of the following factors is most likely contributing to this patient's symptomatic episodes?

- A. Activation of atrioventricular nodal reentrant circuit
- B. Decreased systemic vascular resistance
- C. Increased pulmonary vascular resistance
- D. Intermittent complete heart block
- E. Polymorphic ventricular tachycardia

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- A. Activation of atrioventricular nodal reentrant circuit (16%)
- ✓ B. Decreased systemic vascular resistance (49%)
- ✗ C. Increased pulmonary vascular resistance (2%)
- D. Intermittent complete heart block (27%)
- E. Polymorphic ventricular tachycardia (3%)

Incorrect
Correct answer
B

49%
Answered correctly

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Explanation

Clues to etiology of syncope		
Reflex syncope	Vasovagal	• Trigger (eg, emotional stress, prolonged standing) • Prodrome (eg, nausea, sweating, warmth)
	Situational	• Occurs with micturition, defecation, swallowing, or coughing

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Clues to etiology of syncope		
Reflex syncope	**Vasovagal**	• Trigger (eg, emotional stress, prolonged standing) • Prodrome (eg, nausea, sweating, warmth)
	Situational	• Occurs with micturition, defecation, swallowing, or coughing
	Carotid hypersensitivity	• Tactile stimulation of carotid sinus while standing • Advanced age, carotid atherosclerosis
Orthostatic syncope	**Medications**	• Vasodilators (eg, alpha-1 blockers, antihypertensives) • Inotropic/chronotropic blockade (eg, beta blockers)
	Hypovolemia	• History consistent with volume loss
	Autonomic dysfunction	• Advanced age • Predisposing disease (eg, DM, Parkinson)
Cardiac syncope	**LV outflow obstruction**	• Syncope with exertion • Systolic ejection murmur
	Ventricular tachycardia	• No warning symptoms • Cardiomyopathy or ischemic HD (monomorphic) • QT-interval prolongation (polymorphic)
	Conduction impairment*	• Preceding fatigue or light-headedness • ECG abnormalities (eg, sinus pauses, dropped QRS complexes)

*Sick sinus syndrome or advanced atrioventricular block.

DM = diabetes mellitus; **HD** = heart disease; **LV** = left ventricular.

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This patient with recurrent episodes of **syncope while shaving** most likely has **carotid sinus hypersensitivity** (CSH). When carotid sinus baroreceptors detect increased blood pressure, the brainstem responds by increasing parasympathetic tone (slows the heart rate) and reducing sympathetic tone (induces vasodilation). In some individuals, especially **elderly men**, the carotid sinus baroreceptors become **overly sensitive to tactile stimulation**, triggering an **exaggerated vasovagal response** with marked peripheral vasodilation (ie, **decreased systemic vascular resistance**). This can cause a transient reduction in cerebral perfusion that manifests as presyncope (eg, faintness, lightheadedness) or syncope. As with other etiologies of syncope, cerebral perfusion quickly returns and symptoms resolve within 1-2 minutes.

In addition to shaving, rubbing of a shirt collar while dressing or turning the head is another common cause of syncope due to CSH.

(Choices A and E) Cardiac tachyarrhythmias are a serious cause of syncope due to reduced cardiac output and loss of cerebral perfusion. Spontaneous resolution of the arrhythmia within 1-2 minutes allows for return of cerebral perfusion (if spontaneous resolution does not occur, the arrhythmia may cause sudden death rather than syncope). Atrioventricular nodal reentrant tachycardia rarely causes syncope as it usually affects young patients who can tolerate rapid heart rates. Polymorphic ventricular tachycardia (torsade de pointes) typically results from delayed ventricular repolarization (ie, congenital or medication-induced QT interval prolongation) and can cause syncope in patients of any age. Syncope due to tachyarrhythmia usually occurs suddenly with no warning symptoms (eg, faintness, lightheadedness) other than sometimes palpitations.

(Choice C) Acute pulmonary embolism (PE) obstructs pulmonary blood flow and increases pulmonary vascular resistance. Some cases of acute PE present with syncope, but acute PE is unlikely to explain recurrent episodes of syncope. In addition, chronic pulmonary hypertension typically presents with exertional symptoms.

(Choice D) The exaggerated vagal response in reflex syncope causes transient sinus bradycardia, PR interval prolongation, and atrioventricular (AV) node conduction block, whereas syncope due to age-related degeneration of the AV node usually involves sustained and advanced AV block (eg, third-degree, Mobitz type II second-degree). This patient's recurrent episodes during shaving make reflex syncope due to CSH more likely.

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Educational objective:
Carotid sinus hypersensitivity is a common cause of syncope associated with tactile stimulation of the carotid sinus (eg, shaving). The syncope results from an exaggerated vagal response stimulated by the carotid baroreceptors, which leads to slowed heart rate and marked peripheral vasodilation with a resulting transient loss of cerebral perfusion.

References

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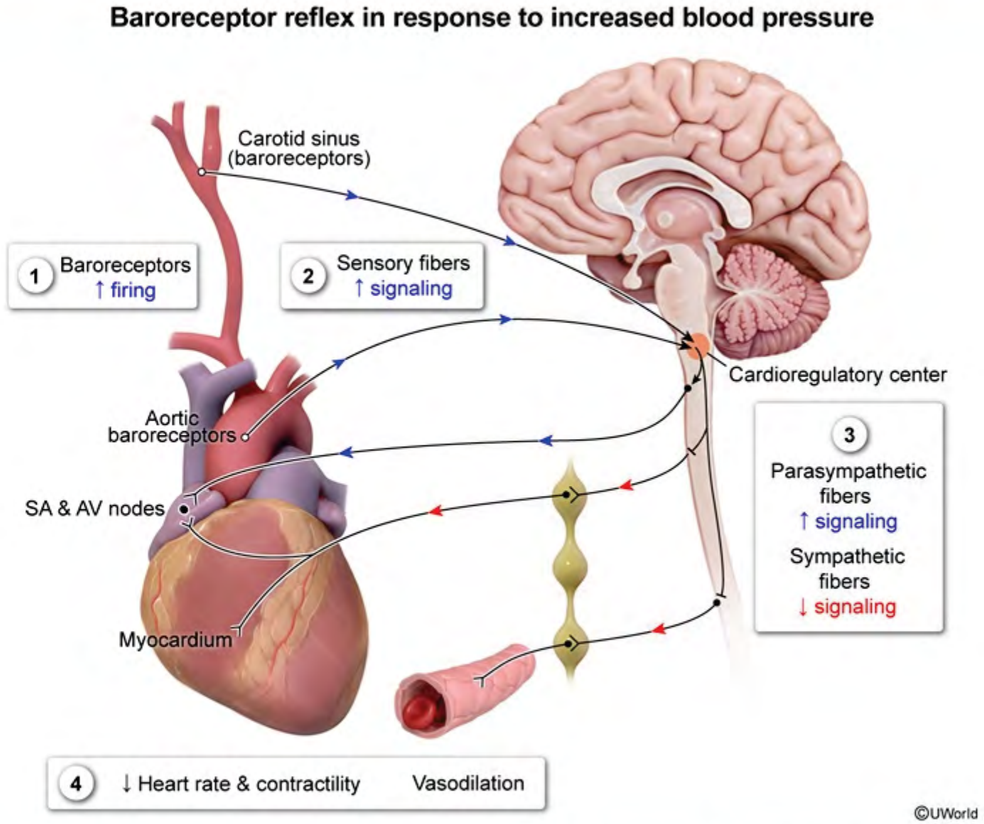

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A 68-year-old man comes to the office due to thigh and leg pain that worsens with exertion. He is unable to walk through the local mall with his wife without discomfort. Past medical history is significant for hypertension and diabetes mellitus. The patient smokes 2 packs of cigarettes a day and consumes alcohol occasionally. Physical examination of the extremities shows weak dorsalis pedis pulses in both feet. Further evaluation confirms moderate peripheral arterial disease involving both lower extremities. Which of the following drugs would best provide symptomatic improvement due to direct dilation of arteries and inhibition of platelet aggregation?

- A. Abciximab
- B. Argatroban
- C. Aspirin
- D. Cilostazol
- E. Heparin
- F. Tissue plasminogen activator
- G. Warfarin

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- A. Abciximab (8%)
- ✗ B. Argatroban (9%)
- C. Aspirin (36%)
- ✓ D. Cilostazol (39%)
- E. Heparin (2%)
- F. Tissue plasminogen activator (2%)
- G. Warfarin (1%)

Incorrect
Correct answer
D

39%
Answered correctly

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Explanation

Several molecules, including thrombin, adenosine diphosphate, and thromboxane A2, activate platelets by acting on cell surface receptors. However, interference with post-receptor signaling can alter platelet function. In particular, cyclic adenosine monophosphate (cAMP) activates a family of enzymes known as the cAMP-dependent protein kinases, or protein kinase A, and leads to inhibition of platelet aggregation. Agents that increase intra-

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Explanation

Several molecules, including thrombin, adenosine diphosphate, and thromboxane A2, activate platelets by acting on cell surface receptors. However, interference with post-receptor signaling can alter platelet function. In particular, cyclic adenosine monophosphate (cAMP) activates a family of enzymes known as the cAMP-dependent protein kinases, or protein kinase A, and leads to inhibition of platelet aggregation. Agents that increase intra-platelet cAMP levels decrease platelet aggregation by preventing platelet shape change and granule release.

Cilostazol reduces **platelet activation** by inhibiting platelet phosphodiesterase, the enzyme responsible for the breakdown of cAMP. It is also a direct arterial **vasodilator**. The net effect is a decrease in claudication **symptoms** and an increase in pain-free walking distances in patients with peripheral arterial disease (PAD). Patients with PAD should be initiated on a graded exercise program, which has also been shown to improve symptoms.

(Choices A, E, and F) Abciximab is a monoclonal antibody that inhibits platelet aggregation by targeting the platelet IIb/IIIa receptor. Heparin is an anticoagulant that potentiates antithrombin III, resulting in the inactivation of thrombin and other factors. Tissue plasminogen activator (alteplase) converts plasminogen to plasmin and causes fibrinolysis. These agents are used in the acute management of cardiovascular events but are not useful for long-term management.

(Choice B) Argatroban is a direct thrombin inhibitor primarily used in the treatment of heparin-induced thrombocytopenia.

(Choice C) Aspirin is an irreversible cyclooxygenase 1 & 2 inhibitor that has been shown to reduce the risk of cardiovascular events in high-risk patients. Patients with clinically significant PAD should take antiplatelet agents (aspirin or clopidogrel) to reduce the risk of cardiovascular events (secondary prevention), but antiplatelet agents do not improve PAD symptoms.

(Choice G) Warfarin is an oral anticoagulant that is a competitive inhibitor of vitamin K epoxide reductase and reduces production of vitamin K-dependent coagulation factors (II, VII, IX, X, protein C, protein S). It is used in the

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(Choice G) Warfarin is an oral anticoagulant that is a competitive inhibitor of vitamin K epoxide reductase and reduces production of vitamin K-dependent coagulation factors (II, VII, IX, X, protein C, protein S). It is used in the long-term treatment and prevention of thromboembolic events but does not reduce claudication in symptomatic PAD.

Educational objective:
Symptomatic management of peripheral artery disease (PAD) includes a graded exercise program and cilostazol. Cilostazol is a phosphodiesterase inhibitor that inhibits platelet aggregation and acts as a direct arterial vasodilator. Patients with PAD should also receive an antiplatelet agent (aspirin or clopidogrel) for secondary prevention of coronary heart disease and stroke.

References

- Safety and efficacy of cilostazol in the management of intermittent claudication.
- Management of lower extremity peripheral arterial disease

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A 12-day-old girl is evaluated in the neonatal intensive care unit (NICU) due to a murmur. The patient was born at 25 weeks gestation and was admitted to the NICU for respiratory support. She was born to a 21-year-old primigravida whose pregnancy was complicated by preeclampsia. Cardiovascular examination reveals a continuous murmur at the left sternal border and bounding femoral and palmar pulses. Echocardiography confirms the diagnosis. Compared to a newborn with a normal cardiovascular system, this patient most likely has which of the following changes in cardiac parameters?

	Left ventricular output	Systemic vascular resistance	Left atrial filling
○ A.	↓	↓	↓
○ B.	↓	↓	↑
○ C.	↑	↓	↑
○ D.	↑	↑	↑
○ E.	↑	↑	↓

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	Left ventricular output	Systemic vascular resistance	Left atrial filling	
✗ A.	↓	↓	↓	(17%)
B.	↓	↓	↑	(15%)
✓ C.	↑	↓	↑	(37%)
D.	↑	↑	↑	(17%)
E.	↑	↑	↓	(11%)

Incorrect
Correct answer
C

37%
Answered correctly

03 secs
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Explanation

Left-to-right cardiac shunts and volume overload

Shunt **proximal to AV valves** causes **right-sided volume overload**

• Excess pulmonary venous return is shunted to RA

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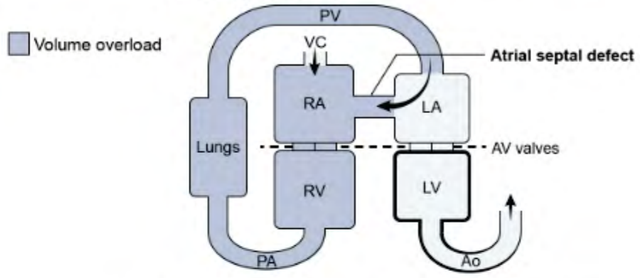
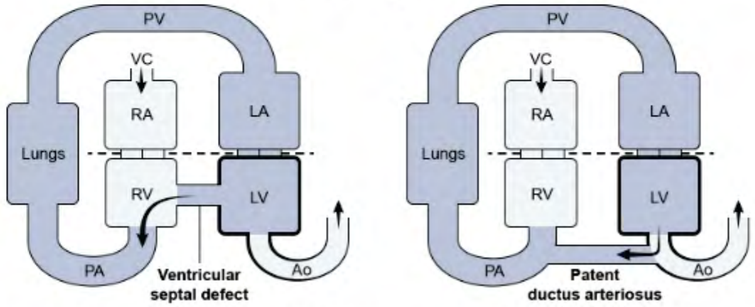
Left-to-right cardiac shunts and volume overload

Shunt **proximal to AV valves** causes **right-sided volume overload**
- Excess pulmonary venous return is shunted to RA
- RV cannot overload LV



Shunt **distal to AV valves** causes **left-sided volume overload**
- Excess pulmonary venous return must flow through LV
- LV can overload itself when given low-resistance pathway



Ao = aorta; **AV** = atrioventricular; **LA** = left atrium; **LV** = left ventricle; **PA** = pulmonary artery; **PV** = pulmonary vein; **RA** = right atrium; **RV** = right ventricle; **VC** = vena cava.

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Shunt **proximal to AV valves** causes **right-sided volume overload**
- Excess pulmonary venous return is shunted to RA

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This extremely premature newborn's continuous murmur and bounding peripheral pulses are consistent with a **patent ductus arteriosus** (PDA). A PDA allows left ventricular (LV) output to flow from the aorta directly into the pulmonary artery; because aortic pressure is higher than pulmonary arterial pressure during both systole and diastole, **left-to-right shunting** occurs throughout the cardiac cycle and creates the classic **continuous murmur**.

The low-resistance pathway into the pulmonary circulation effectively **decreases systemic vascular resistance** (SVR) compared to the normal circulatory system. Pulmonary overcirculation occurs with **increased blood return to the left atrium** and left ventricle (ie, left-sided volume overload). The left ventricle compensates for the increased preload (increased blood return) and reduced afterload (reduced SVR) by **increasing stroke volume and cardiac output**, which leads to increased pulse pressure (large difference between diastolic and systolic blood pressures) and bounding pulses.

(Choices A, B, D, and E) A PDA initially manifests with an overall decrease in SVR due to shunting of aortic blood into the low-resistance pulmonary circulation. Over time, left-sided volume overload caused by a large PDA can lead to LV failure and reduced cardiac output, which may induce a reflexive increase in SVR in an effort to maintain organ and tissue perfusion. However, bounding pulses (as seen in this patient) are not expected once LV failure develops and cardiac output drops.

Educational objective:
A patent ductus arteriosus allows blood from the aorta to flow directly into the pulmonary artery, which effectively decreases systemic vascular resistance. Pulmonary overcirculation occurs with increased blood return to the left atrium and left ventricle, and the left ventricle compensates by increasing stroke volume and cardiac output.

Physiology	Cardiovascular System	Patent ductus arteriosus
Subject	System	Topic

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Ventricular septal defect
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Patent ductus arteriosus
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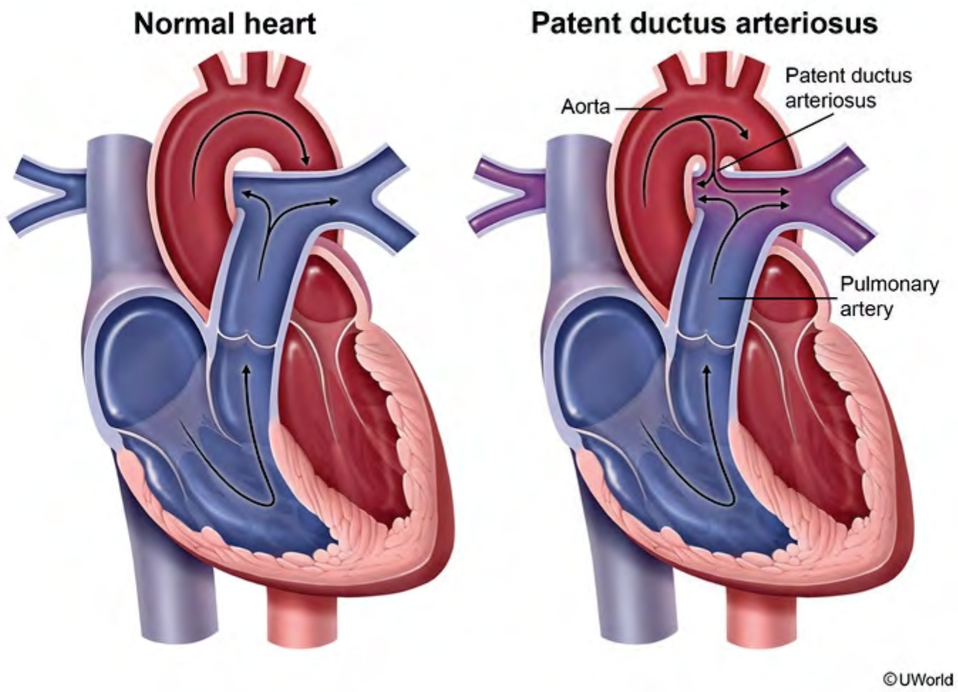

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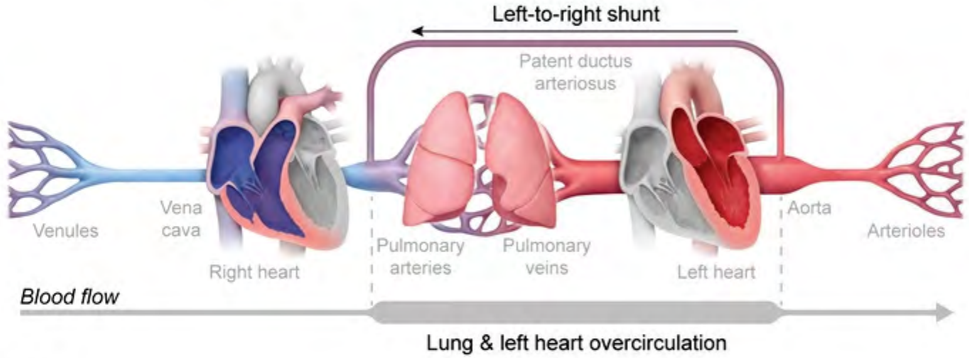
Hemodynamics of patent ductus arteriosus



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A 38-year-old woman suddenly collapses at home after experiencing severe chest pain and dies before reaching a hospital. Postmortem examination reveals an occluding thrombus overlying a ruptured atherosclerotic plaque on the left anterior descending artery. The patient is also found to have thickened mitral valve leaflets with multiple small vegetations on both valvular surfaces and fibrinoid necrosis of arterioles. Other findings include glomerular capillary basement membrane thickening with wire-loop changes. This patient was most likely suffering from which of the following conditions?

- A. Bacterial endocarditis
- B. Churg-Strauss syndrome
- C. Granulomatosis with polyangiitis
- D. Systemic lupus erythematosus
- E. Thromboangiitis obliterans

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- ✗ A. Bacterial endocarditis (8%)
- B. Churg-Strauss syndrome (4%)
- C. Granulomatosis with polyangiitis (8%)
- ✓ D. Systemic lupus erythematosus (74%)
- E. Thromboangiitis obliterans (3%)

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Correct answer
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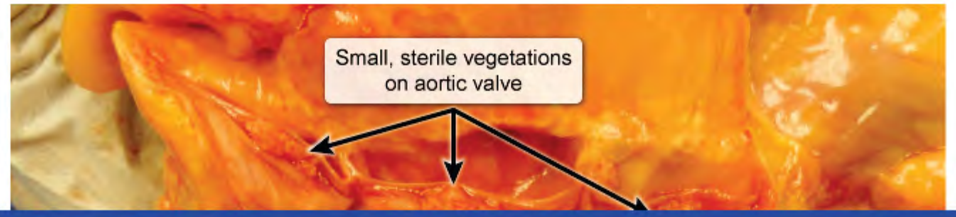
Explanation

Noninfective endocarditis



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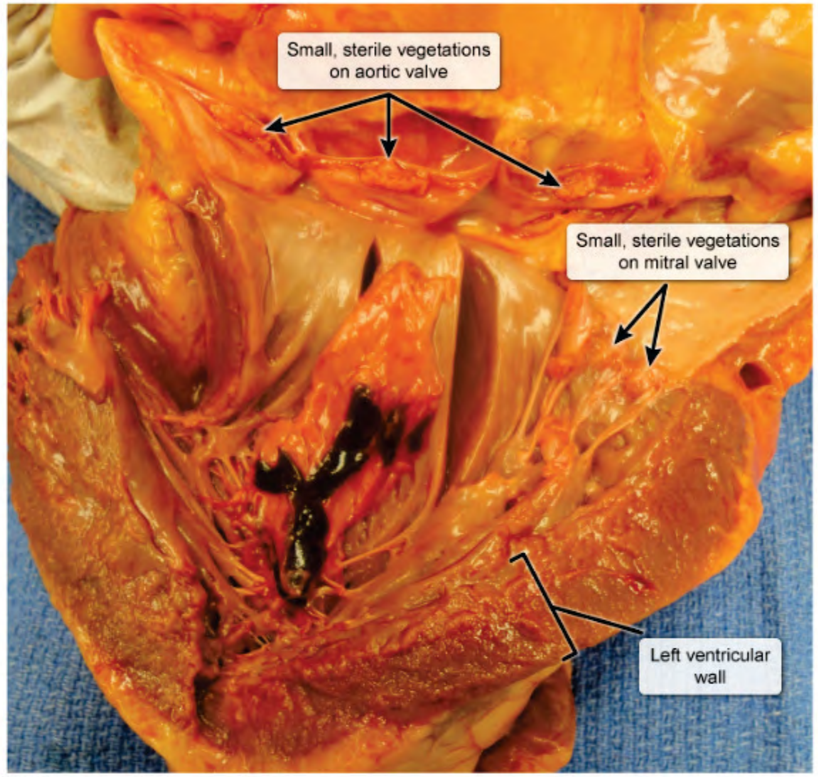
A 38-year-old woman suddenly collapses at home after experiencing severe chest pain and dies before reaching a hospital. Autopsy reveals a vegetation on the left atrial surface of the mitral valve and small vegetations on the aortic valve. Histologic examination of the capillaries in the kidneys reveals immune complex deposition. Which of the following is the most likely diagnosis?

Exhibit Display

Noninfective endocarditis



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This patient's postmortem findings are consistent with systemic lupus erythematosus (SLE), an autoimmune disease that affects women of childbearing age and results in immune complex-mediated tissue injury in various organs.

Cardiovascular manifestations of SLE include **accelerated atherosclerosis** with increased risk of myocardial infarction. Small arteries and arterioles can also show evidence of vasculitis with fibrinoid necrosis in any tissue. Other cardiac features include pericarditis and less commonly **verrucous (Libman-Sacks) endocarditis**, characterized by thickened valve leaflets (typically mitral and/or aortic) with multiple small vegetations on **both surfaces**. The vegetations are composed of **sterile** platelet thrombi intertwined with strands of fibrin, immune complexes, and mononuclear cells. These vegetations are easily dislodged and can result in systemic embolization.

Renal involvement in SLE most often manifests as **diffuse proliferative glomerulonephritis**, which is characterized by proliferative and necrotizing lesions with crescent formation during active disease. Light microscopy also classically shows diffuse thickening of the glomerular capillary walls with "wire-loop" structures due to subendothelial immune complex deposition.

(Choice A) Bacterial endocarditis is characterized by large, potentially destructive vegetations on the valve cusps that contain fibrin, inflammatory cells, and bacteria (eg, staphylococci, streptococci). Renal involvement can occur with septic embolization or development of immune complex-mediated glomerulonephritis with hypercellular infiltrate and immune deposits in the glomerular capillary wall.

(Choice B) Churg-Strauss syndrome (eosinophilic granulomatosis with polyangiitis) is a small-vessel necrotizing vasculitis characterized by allergic rhinitis, asthma, and peripheral eosinophilia. Although renal involvement is not typically prominent, patients can develop necrotizing crescentic glomerulonephritis.

(Choice C) Granulomatosis with polyangiitis (Wegener granulomatosis) is a small-vessel necrotizing vasculitis characterized by upper respiratory (eg, sinusitis, otitis media) and lower respiratory (eg, dyspnea, hemoptysis) symptoms as well as renal disease. Renal involvement typically manifests as focal necrotizing, crescentic glomerulonephritis.

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(Choice E) Thromboangiitis obliterans (Buerger disease) is a small- to medium-vessel vasculitis that typically occurs with cigarette smoking and causes vascular insufficiency in the extremities (eg, gangrene, autoamputation of digits). It is characterized histologically by intraluminal thrombus and prominent inflammatory cell infiltrate with relative sparing of the vessel wall.

Educational objective:
Cardiovascular manifestations of lupus include accelerated atherosclerosis, small-vessel necrotizing vasculitis, pericarditis, and Libman-Sacks endocarditis (small, sterile vegetations on both sides of the valve). Renal involvement classically manifests as diffuse proliferative glomerulonephritis, which is characterized by diffuse thickening of the glomerular capillary walls with "wire-loop" structures on light microscopy.

References

- The classification of glomerulonephritis in systemic lupus erythematosus revisited.
- Diagnostic histopathology of major systemic and pulmonary vasculitis syndromes.

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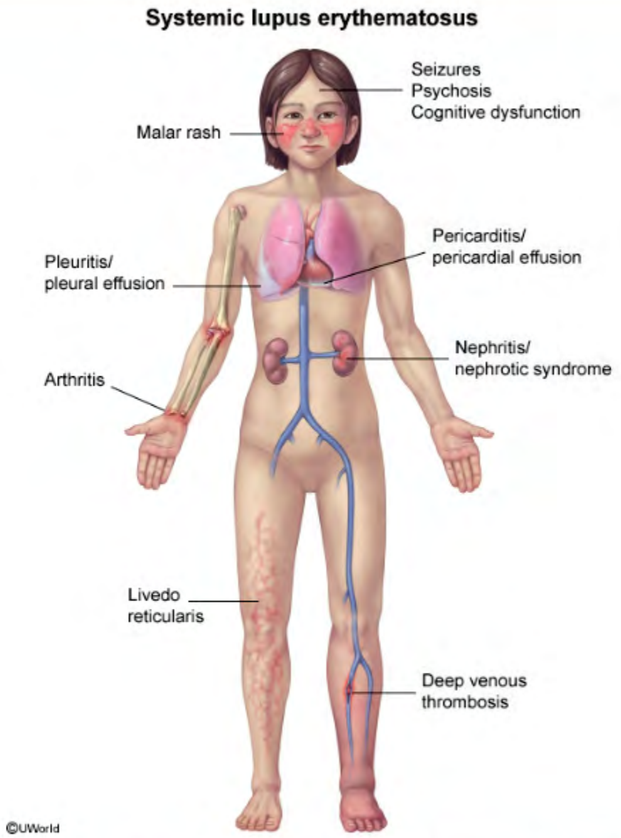

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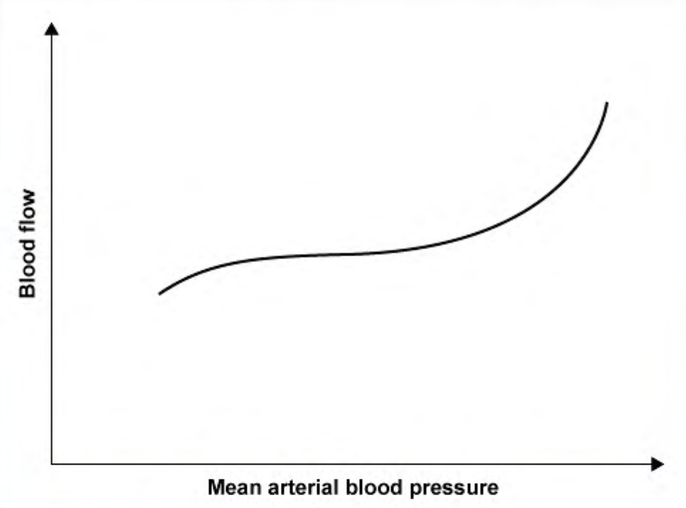
Researchers are observing how coronary blood flow changes in response to progressive increases in the mean arterial blood pressure (see graph below).



The mostly horizontal portion of the slope indicates a region where coronary blood flow is relatively insensitive to blood pressure changes. Within this zone of autoregulation, the metabolic demands of the myocardium are the main determinant of coronary blood flow. Which of the following endogenous factors is most responsible for controlling coronary blood flow within this range?

- A. Acetylcholine
- B. Angiotensin II
- C. Histamine
- D. Nitric oxide
- E. Norepinephrine

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Researchers are observing how coronary blood flow changes in response to progressive increases in the mean arteria

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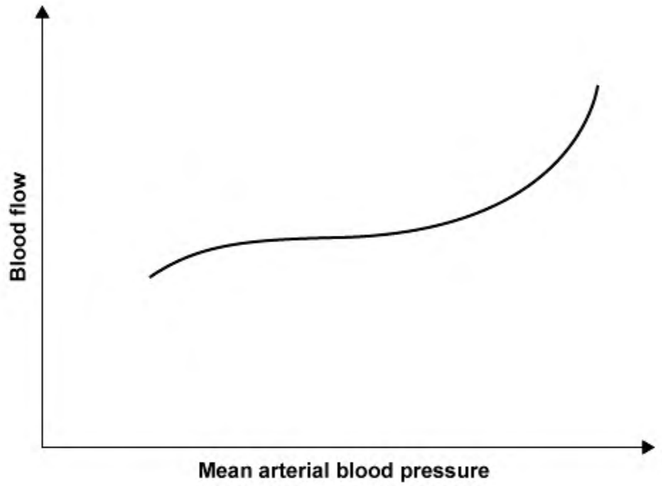

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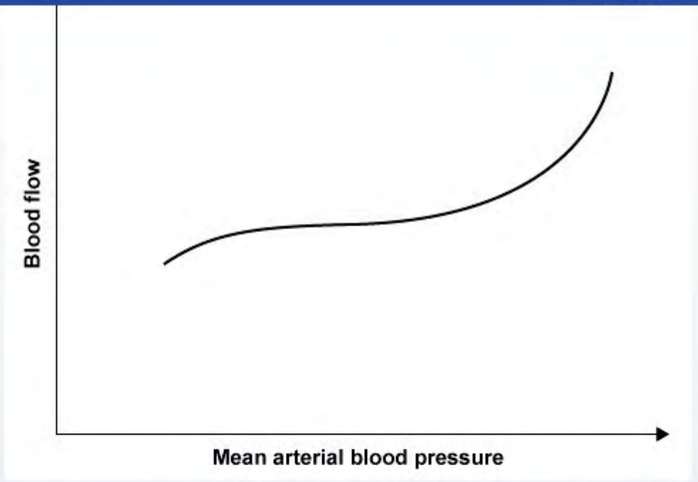

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- B. Angiotensin II
- C. Histamine
- D. Nitric oxide
- E. Norepinephrine
- F. Serotonin

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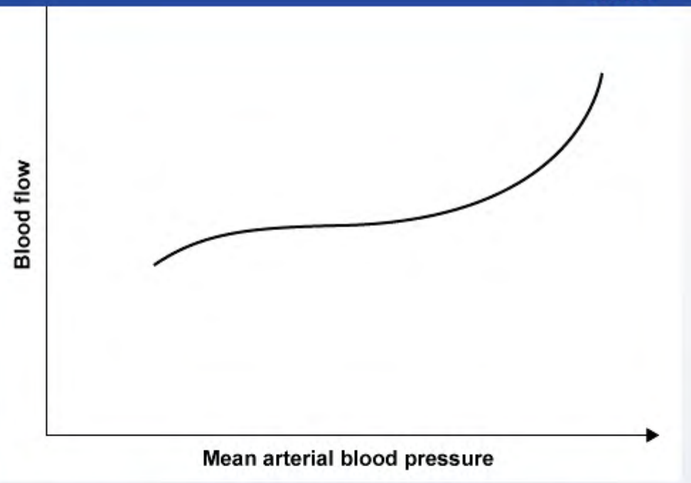

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- A. Acetylcholine (8%)
- ✗ B. Angiotensin II (5%)
- C. Histamine (0%)
- ✓ D. Nitric oxide (75%)
- E. Norepinephrine (8%)
- F. Serotonin (0%)

Incorrect
Correct answer
D

75%
Answered correctly

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D. Nitric oxide (75%)

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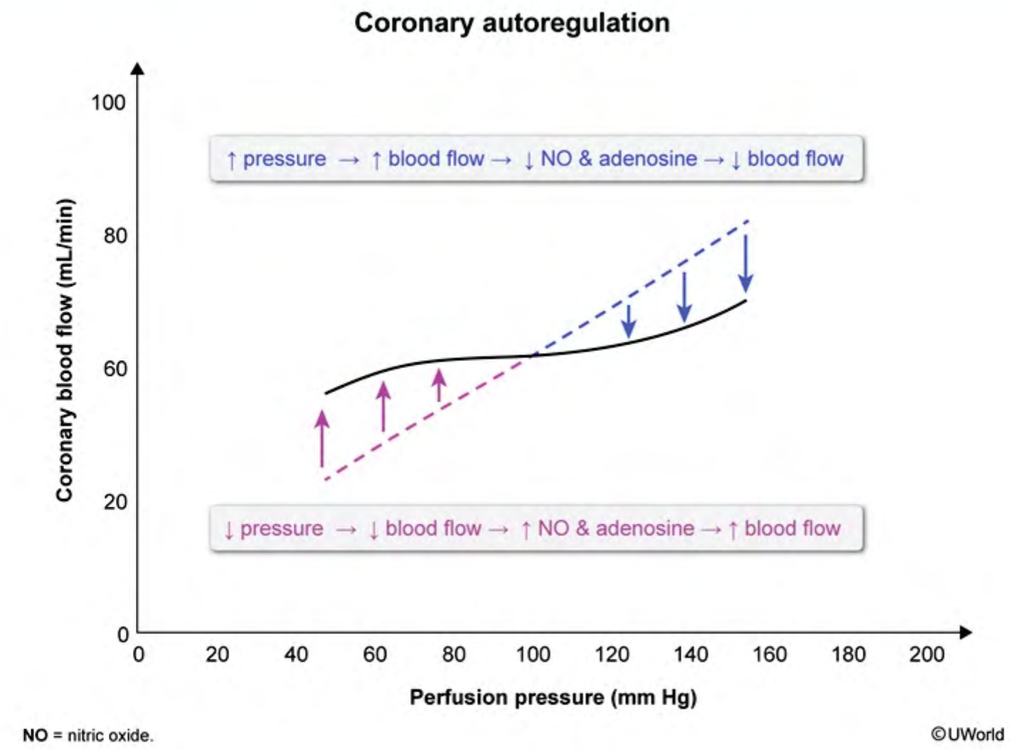

NO = nitric oxide.

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Perfusion pressure (mm Hg)

NO = nitric oxide.

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The myocardium has the highest resting oxygen requirement of any tissue in the body, extracting >70% of available oxygen from the blood at baseline (compared to ~30% in most other tissues). As such, a significant **increase in myocardial oxygen demand** can only be met through a corresponding **increase in coronary blood flow**.

Coronary autoregulation is a process that **maintains myocardial blood flow** in settings of decreased coronary perfusion pressure (eg, hemorrhage, sepsis). Within the region of autoregulation (eg, 60-140 mm Hg), changes in coronary blood flow are driven primarily by myocardial oxygen demand. This is accomplished mostly by automated alterations in vascular resistance via the release of locally acting mediators, namely **adenosine** (released from cardiomyocytes as ATP is broken down for energy) and **nitric oxide** (synthesized by endothelial cells in response to chemical mediators and mechanical stress).

When myocardial perfusion pressure decreases, there is decreased oxygen delivery to the myocardium and less ATP is regenerated via oxidative metabolism, resulting in increased levels of adenosine. Myocardial hypoxia also triggers increased synthesis of nitric oxide. Increased levels of both adenosine and nitric oxide act on vascular smooth muscle to stimulate **vasodilation**, allowing for a relative increase in coronary blood flow at the new, lower perfusion pressure (ie, coronary blood flow is maintained). A similar process occurs when coronary perfusion pressure increases; decreased levels of adenosine and nitric oxide increase vascular resistance, causing coronary blood flow to remain relatively unchanged at the new, higher perfusion pressure.

(Choices A and E) Acetylcholine causes coronary vasodilation (via stimulation of endothelial nitric oxide release), and norepinephrine causes coronary vasoconstriction (via stimulation of alpha-1 receptors on vascular smooth muscle). However, compared to locally released mediators, autonomic nervous system stimulation has a relatively small effect on coronary blood flow.

(Choice B) Angiotensin II stimulates vasoconstriction of coronary arteries, but it is not involved in coronary autoregulation.

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to chemical mediators and mechanical stress).

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(Choice B) Angiotensin II stimulates vasoconstriction of coronary arteries, but it is not involved in coronary autoregulation.

(Choice C) Histamine has variable vasoconstrictive and vasodilatory effects on coronary arteries depending on the type of histamine receptor triggered. However, histamine does not play a significant role in coronary autoregulation.

(Choice F) Serotonin primarily functions in the gastrointestinal tract and CNS. It may cause vasoconstriction of coronary arteries, evidenced by an association between $5HT_1$-receptor agonists (eg, sumatriptan) and coronary vasospasm. However, it is not significantly involved in coronary autoregulation.

Educational objective:
Coronary autoregulation allows coronary blood flow to be primarily driven by myocardial oxygen demand over a wide range of perfusion pressures (60-140 mm Hg). It is mostly accomplished by alterations in vascular resistance via release of adenosine and nitric oxide in response to myocardial hypoxia.

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A 78-year-old man dies of advanced esophageal cancer. The man's family donates his body for teaching purposes and an autopsy is performed. Gross examination of the heart reveals left ventricular chamber size and wall thickness within normal limits. No significant atherosclerosis is seen in the coronary arteries. On microscopic examination, myocardial cells demonstrate prominent yellow-brown intracytoplasmic granules as shown in the exhibit. Which of the following most likely accounts for the observed microscopic changes?

- A. Exogenous pigment endocytosis
- B. Glucose polymerization
- C. Iron overload
- D. Lipid peroxidation
- E. Protein accumulation
- F. Tyrosine oxidation

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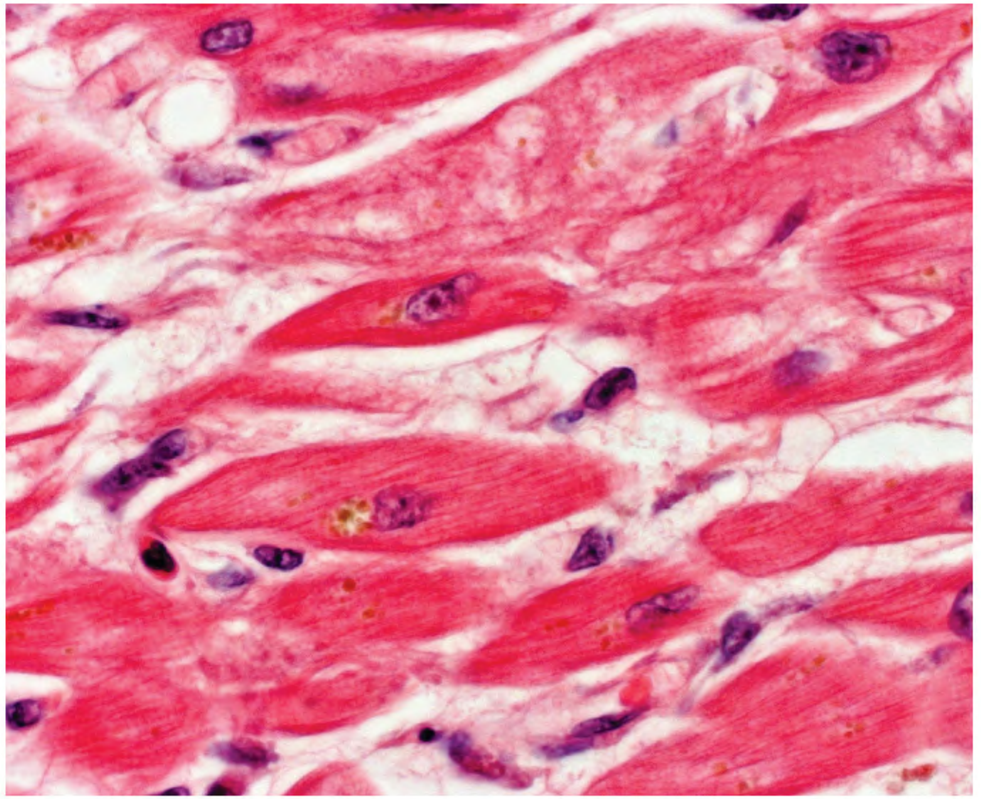

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- A. Exogenous pigment endocytosis (5%)
- B. Glucose polymerization (0%)
- ✗ C. Iron overload (18%)
- ✓ D. Lipid peroxidation (64%)
- E. Protein accumulation (9%)
- F. Tyrosine oxidation (1%)

Incorrect
Correct answer
D

64%
Answered correctly

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Explanation

The yellow-brown pigment contained within intracytoplasmic granules (ie, **residual bodies**) in this man's myocardium represents **lipofuscin**. Lipofuscin is composed of **lysosomal breakdown products** including **lipid polymers** and protein-complex **phospholipids** that result from free radical injury and **lipid peroxidation**. The insoluble granules are considered a **wear-and-tear product** that **accumulates with age**, and they are commonly seen in low-turnover cells (eg, heart, liver, central nervous system) of the elderly.

Malnourishment, as may have been present in this man with advanced esophageal cancer, may contribute to

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Malnourishment, as may have been present in this man with advanced esophageal cancer, may contribute to increased lipofuscin production.

(Choice A) A common example of exogenous pigment endocytosis occurs when urban-residing individuals inhale carbon or coal dust that is taken up by macrophages within the lung parenchyma, resulting in prominent black pigment (ie, anthracosis).

(Choice B) Glycogen is a glucose polymerization product and typically appears as clear vacuoles within the cytoplasm. These clear vacuoles are seen in abundance in patients with glycogen storage disease.

(Choice C) Iron-containing brown pigment called hemosiderin is often seen in areas of localized iron overload caused by bleeding into tissues. Decompensated heart failure causes leakage of edema and erythrocytes into the lung parenchyma, and hemosiderin-laden macrophages can be seen.

(Choice E) Hyaline is a form of protein accumulation that can be intracellular or extracellular. It typically appears as a glassy, homogeneous pink deposit, as seen with hyaline arteriosclerosis of the kidney.

(Choice F) Melanin is an oxidation product of tyrosine metabolism and typically appears as pigmented granules dark brown to black in color. Melanoma demonstrates high melanin content.

Educational objective:
Lipofuscin is a yellow-brown pigment composed of lysosomal breakdown products of lipid polymers and protein-complex phospholipids. It is considered a wear-and-tear product and marker of aging because it accumulates over time, and residual bodies containing lipofuscin are prevalent in low-turnover cells (eg, heart, liver, central nervous system) of the elderly.

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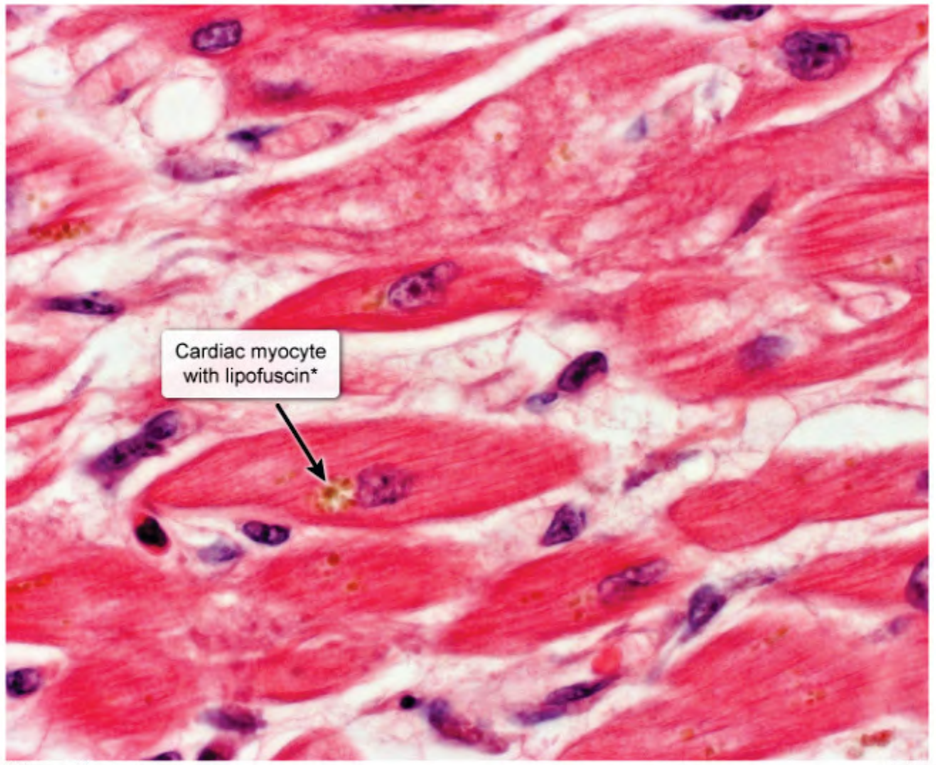
Exhibit Display

Lipofuscin



*"Wear and tear" pigment

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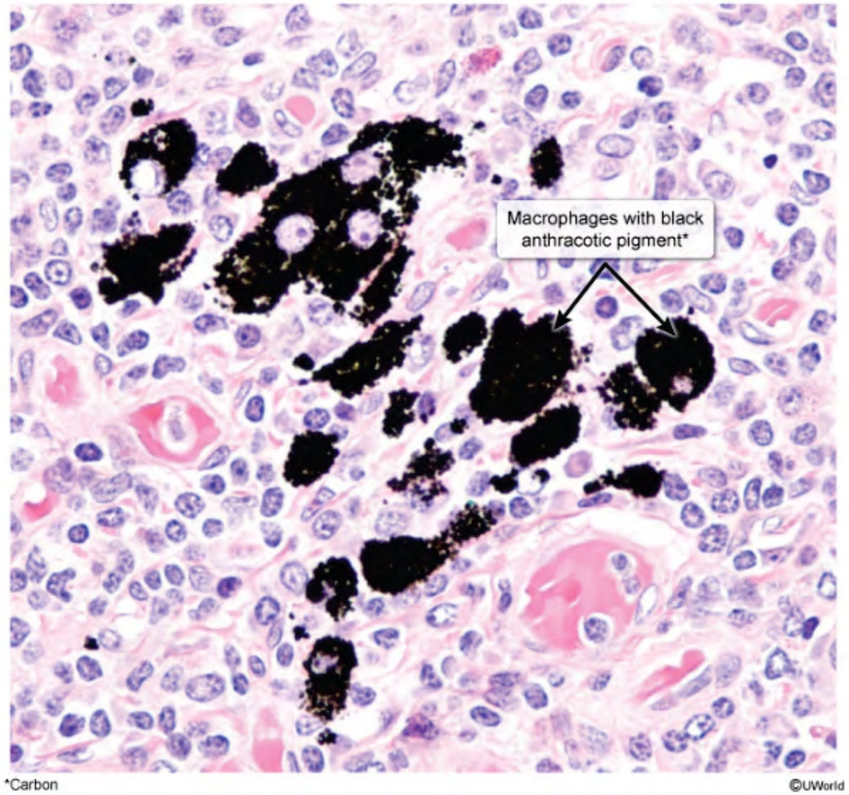
Anthracotic pigment



*Carbon

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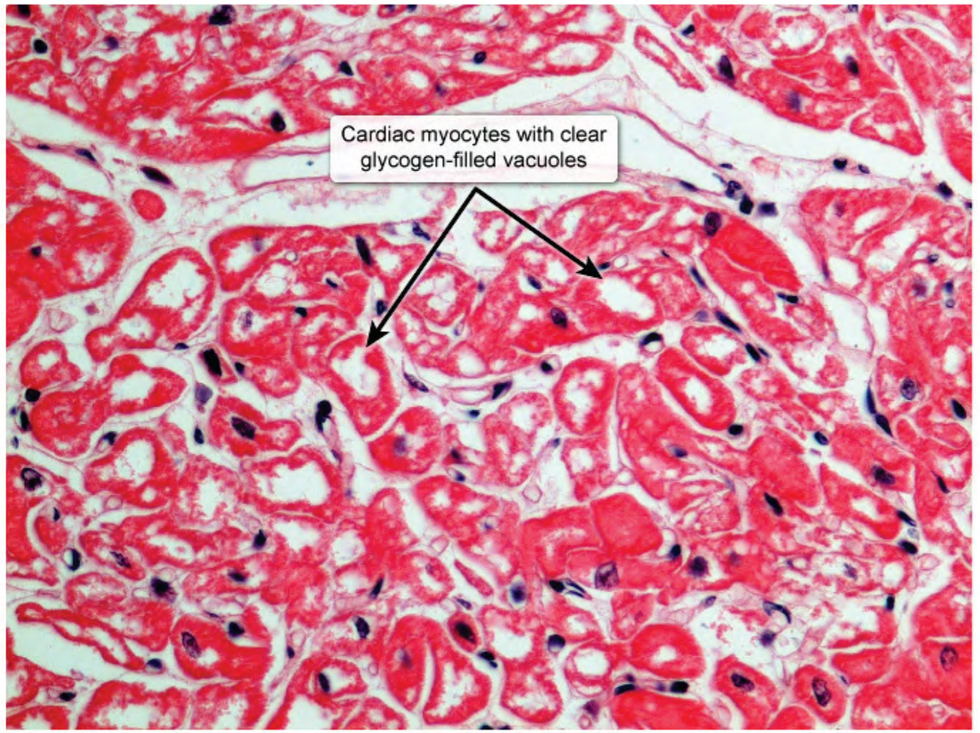
Glycogen storage disorder



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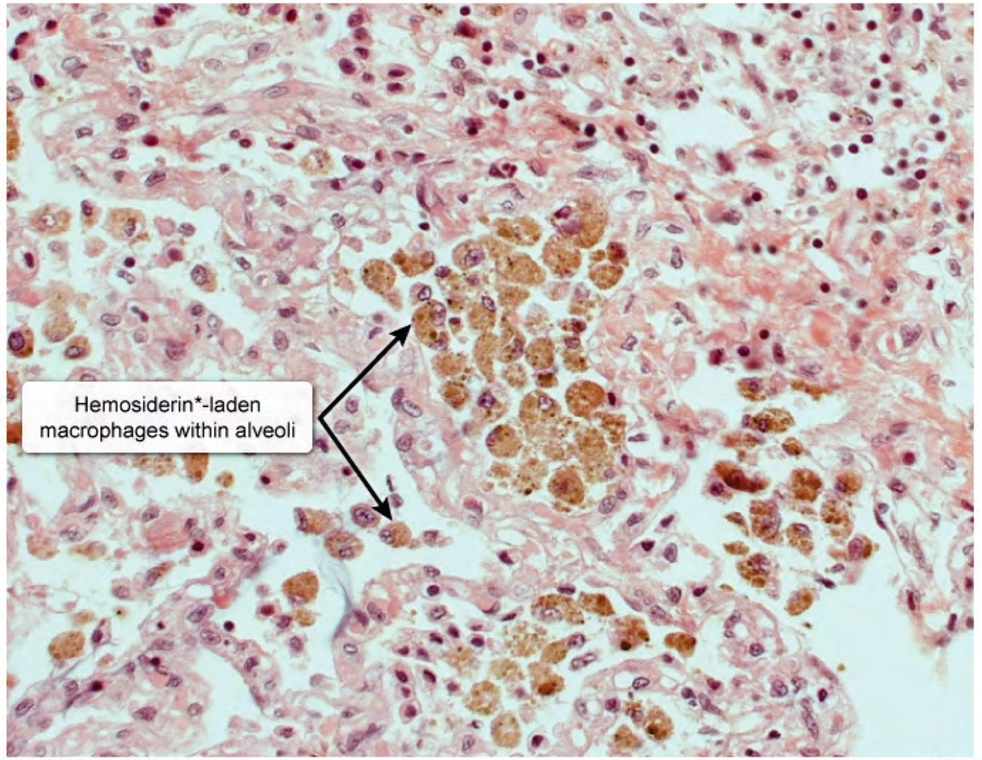

*Iron-containing pigment

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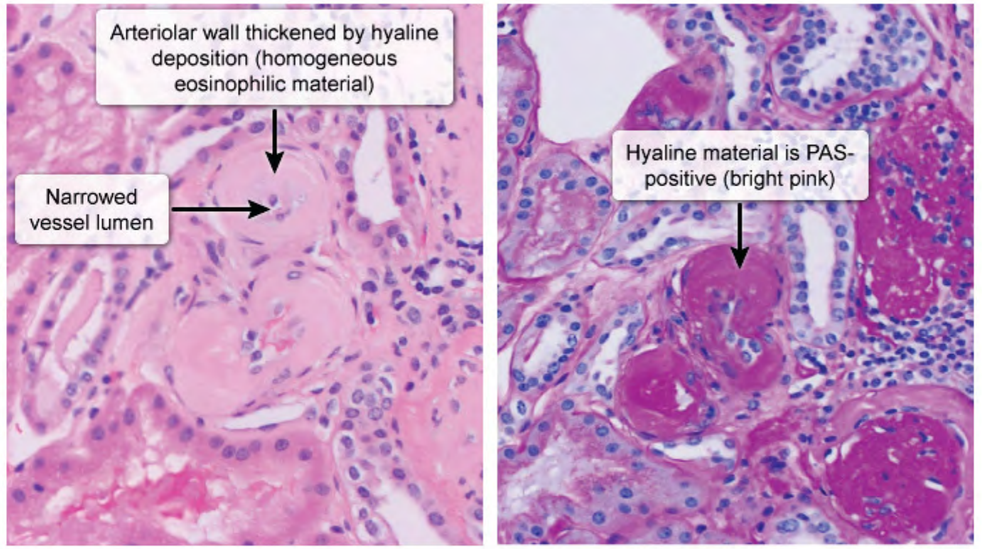
Hyaline arteriolosclerosis



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H&E stain

Periodic acid-Schiff (PAS) stain

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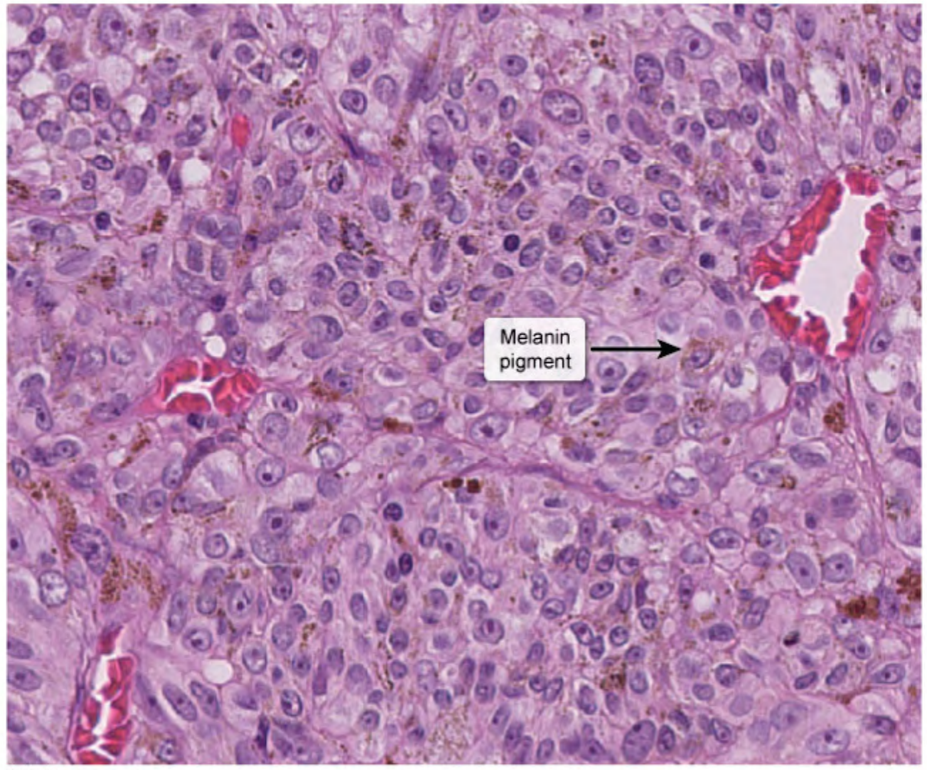
Melanin



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A 36-year-old man comes to the office after he was found to have an abnormal lipid panel during employee wellness testing at his company. He has no prior medical problems and takes no medications. The patient is a software technician and has a sedentary lifestyle. He eats mostly fast foods, rarely exercises, and drinks 2-3 cans of beer daily. His BMI is 31 kg/m^2. Physical examination is unremarkable. Results of laboratory studies performed in the office are as follows:

Total cholesterol	290 mg/dL
High-density lipoprotein	45 mg/dL
Low-density lipoprotein	110 mg/dL
Triglycerides	675 mg/dL

Lifestyle modification with a balanced diet, regular exercise, and reduced alcohol intake is advised. He is also started on fenofibrate therapy. This medication is most likely to help the patient by which of the following mechanisms?

- A. Blocking intestinal cholesterol absorption
- B. Decreasing hepatic cholesterol synthesis
- C. Increasing fecal loss of cholesterol derivatives
- D. Inhibiting LDL receptor degradation
- E. Reducing hepatic VLDL production

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Lifestyle modification with a balanced diet, regular exercise, and reduced alcohol intake is advised. He is also started on fenofibrate therapy. This medication is most likely to help the patient by which of the following mechanisms?

- A. Blocking intestinal cholesterol absorption (14%)
- ✗ B. Decreasing hepatic cholesterol synthesis (8%)
- C. Increasing fecal loss of cholesterol derivatives (16%)
- D. Inhibiting LDL receptor degradation (15%)
- ✓ E. Reducing hepatic VLDL production (45%)

Incorrect
Correct answer
E

45%
Answered correctly

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Time Spent

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C. Increasing fecal loss of cholesterol derivatives (16%)

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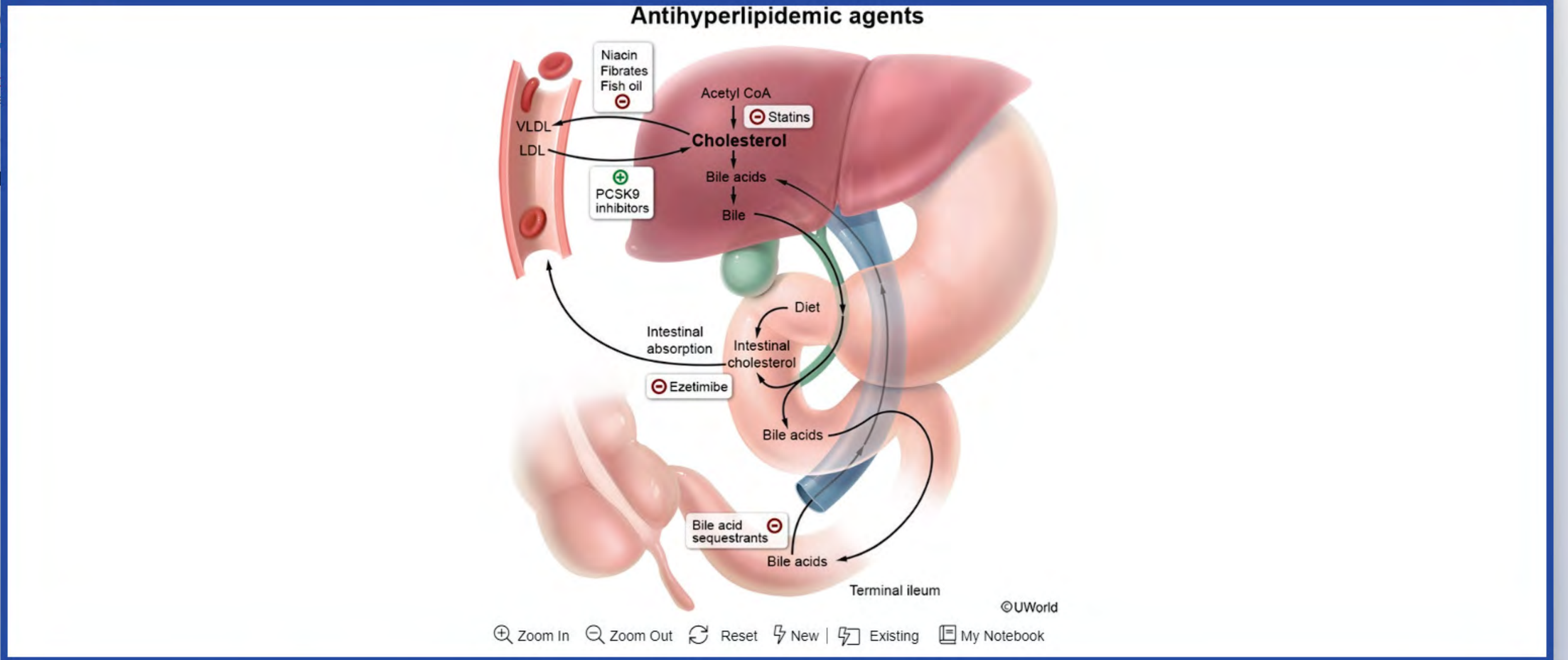

Ezetimibe

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Terminal ileum

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This patient has a moderately elevated (>500 mg/dL) triglyceride level, which is associated with an increased risk of cardiovascular disease. Lifestyle modifications (increased aerobic exercise, decreased alcohol and total caloric intake) can decrease triglycerides, but moderate (or worse) hypertriglyceridemia usually requires pharmacologic therapy.

Lipoprotein lipase (LPL) hydrolyzes triglycerides in chylomicrons and VLDL to release free fatty acids, which can be used for energy or converted back to triglycerides for storage in adipose tissue. It also facilitates the transfer of triglycerides from these lipoproteins to HDL. **Fibrates** (eg, gemfibrozil, fenofibrate) activate **peroxisome proliferator-activated receptor alpha** (PPAR-α), which leads to **decreased hepatic VLDL** production and increased LPL activity. They are able to decrease triglyceride levels by 25%-50% and increase HDL by 5%-20%. Fish oil supplements containing high concentrations of **omega-3 fatty acids** also decrease **VLDL production**, and inhibit synthesis of apolipoprotein B as well. These supplements lower triglycerides and can be used as an alternate treatment for patients with moderate hypertriglyceridemia.

(Choices A, C, and D) Ezetimibe selectively blocks intestinal cholesterol absorption. Bile acid-binding resins (eg, cholestyramine) increase fecal loss of cholesterol derivatives by binding bile acids in the intestine and disrupting enterohepatic bile acid circulation. Proprotein convertase subtilisin kexin 9 (PCSK9) inhibitors are monoclonal antibodies that reduce LDL receptor degradation in the liver. These drugs are used primarily in patients with elevated cholesterol levels but have minimal effect on triglyceride levels.

(Choice B) Statins (eg, atorvastatin) decrease hepatic cholesterol synthesis by inhibiting HMG-CoA reductase. Statins have a modest effect on triglyceride levels and are recommended for patients with mild to moderate hypertriglyceridemia.

Educational objective:
Fibrates lower triglyceride levels by activating peroxisome proliferator-activated receptor alpha, which leads to decreased hepatic VLDL production and increased lipoprotein lipase activity. Fish oil supplements containing high

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Educational objective:
Fibrates lower triglyceride levels by activating peroxisome proliferator-activated receptor alpha, which leads to decreased hepatic VLDL production and increased lipoprotein lipase activity. Fish oil supplements containing high concentrations of omega-3 fatty acids lower triglycerides by decreasing production of VLDL and apolipoprotein B.

References

- Fenofibrate: a review of its use in dyslipidaemia.

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A 53-year-old man comes to the emergency department with shortness of breath and chest tightness. The patient was playing in a poker tournament when his symptoms first began. He has a history of hypertension and is not compliant with his medications. His last medical follow-up was a year ago. Blood pressure is 195/115 mm Hg and pulse is 90/min and regular. Lung examination reveals bibasilar crackles. Nitroglycerin infusion is started and results in significant symptomatic improvement. Repeat blood pressure is 165/90 mm Hg. Which of the following intracellular events is most likely responsible for the beneficial effects of this patient's treatment?

- A. Actin phosphorylation
- B. Calcium release from sarcoplasmic reticulum
- C. Enhanced cyclic mononucleotide degradation
- D. Inositol triphosphate accumulation
- E. Myosin dephosphorylation
- F. Tyrosine kinase activation

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- A. Actin phosphorylation (3%)
- ✕ B. Calcium release from sarcoplasmic reticulum (8%)
- C. Enhanced cyclic mononucleotide degradation (22%)
- D. Inositol triphosphate accumulation (11%)
- ✓ E. Myosin dephosphorylation (49%)
- F. Tyrosine kinase activation (4%)

Incorrect
Correct answer
E

49%
Answered correctly

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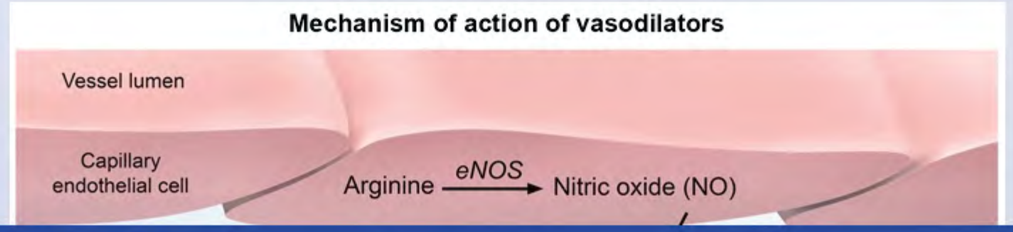
Explanation



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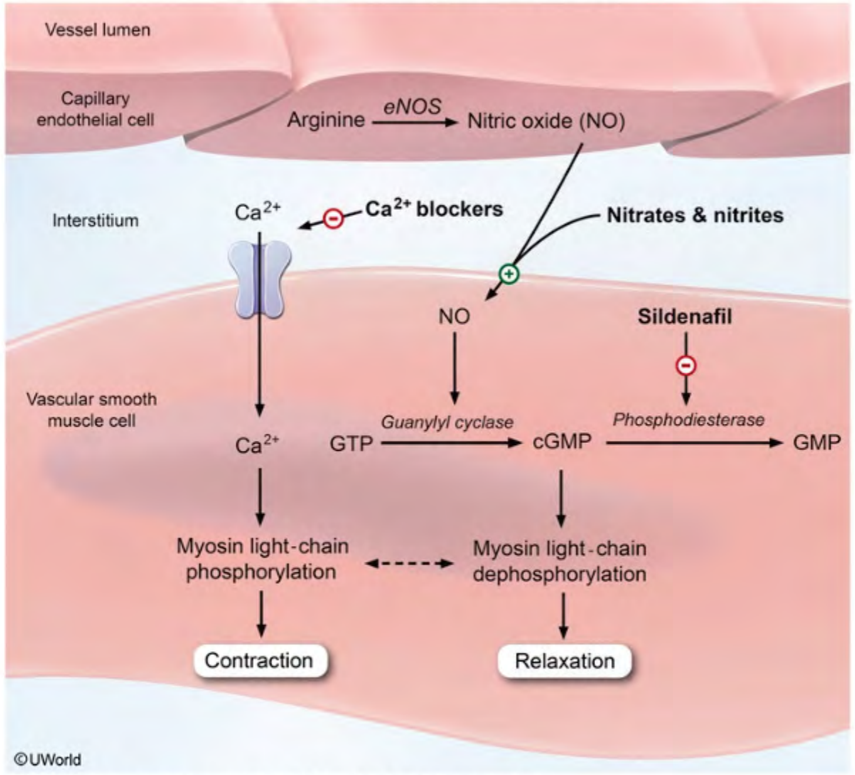
Mechanism of action of vasodilators



cGMP = cyclic guanosine monophosphate; **eNOS** = endothelial nitric oxide synthase; **GMP** = guanosine monophosphate; **GTP** = guanosine 5′-triphosphate.

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endothelial cell
Arginine
Nitric oxide (NO)

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cGMP = cyclic guanosine monophosphate; **eNOS** = endothelial nitric oxide synthase; **GMP** = guanosine monophosphate; **GTP** = guanosine 5′-triphosphate.

This patient's clinical presentation is consistent with acute pulmonary edema due to severely elevated blood pressure (**hypertensive emergency**). In such cases, intravenous vasodilators (nitroglycerine, sodium nitroprusside) are often used to improve the acute heart failure by reducing preload and afterload.

Nitrates are metabolized within vascular smooth muscle cells to nitric oxide, which activates guanylate cyclase and promotes the conversion of guanosine triphosphate (GTP) to cyclic guanosine monophosphate (**cGMP**). Increased levels of cGMP lead to **decreased intracellular calcium** (reduces the activity of myosin light-chain kinase) and activation of myosin light chain phosphatase. This promotes **myosin light-chain dephosphorylation** and vascular smooth muscle **relaxation**.

(Choice A) Actin phosphorylation has no role in smooth or skeletal muscle relaxation. In smooth muscle, a stimulus to contract leads to increased intracellular calcium, which leads to myosin phosphorylation. Once myosin is phosphorylated, it is able to bind directly to actin and cause muscle contraction.

(Choice B) Calcium release from sarcoplasmic reticulum would lead to increased contraction of the cardiac myocytes. It is not involved in the mechanism of action of nitrates on vascular smooth muscle cells.

(Choice C) Nitrates do not cause an increase in cyclic mononucleotide degradation. On the contrary, they activate guanylate cyclase and promote conversion of GTP to cGMP, which ultimately leads to smooth muscle relaxation.

(Choice D) Inositol triphosphate binds to its receptor on the endoplasmic reticulum and leads to the release of Ca^{2+} into the cytoplasm. In vascular smooth muscle cells, this increased concentration of cytoplasmic Ca^{2+} results in increased smooth muscle contraction.

(Choice F) Tyrosine kinases play a role in a wide variety of intracellular processes and pathways. The effects of insulin and insulin-like growth factor occur via activation of receptor tyrosine kinases, which in turn cause phosphorylation of tyrosine residues on intracellular proteins important for signal transduction.

Educational objective:

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Educational objective:
Nitrates (via conversion to nitric oxide) activate guanylate cyclase and increase intracellular levels of cyclic guanosine monophosphate (cGMP). Increased levels of cGMP lead to myosin light-chain dephosphorylation, resulting in vascular smooth muscle relaxation.

References

- Identification of the enzymatic mechanism of nitroglycerin bioactivation.

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A 53-year-old man comes to the physician for a follow-up visit after an acute myocardial infarction. His medications include metoprolol and low-dose aspirin. He used to smoke 2 packs of cigarettes daily but quit after his myocardial infarction. The patient's father has hypertension and his mother has type 2 diabetes mellitus. He currently weighs 100 kg (220 lb) and is 178 cm (70 in) tall. Examination shows an obese male with no other abnormalities. His total serum cholesterol level is 155 mg/dL, with an HDL level of 27 mg/dL and a triglyceride level of 92 mg/dL. Which of the following lipid-lowering agents would be most effective for preventing future cardiovascular events in this patient?

- A. Absorption inhibitor
- B. Cationic exchange resin
- C. Enzyme inhibitor
- D. Essential fatty acids
- E. Pharmacologic vitamin
- F. Transcription factor ligand

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A 53-year-old man comes to the physician for a follow-up visit after an acute myocardial infarction. His medications include metoprolol and low-dose aspirin. He used to smoke 2 packs of cigarettes daily but quit after his myocardial infarction. The patient's father has hypertension and his mother has type 2 diabetes mellitus. He currently weighs 100 kg (220 lb) and is 178 cm (70 in) tall. Examination shows an obese male with no other abnormalities. His total serum cholesterol level is 155 mg/dL, with an HDL level of 27 mg/dL and a triglyceride level of 92 mg/dL. Which of the following lipid-lowering agents would be most effective for preventing future cardiovascular events in this patient?

- ✗ A. Absorption inhibitor (4%)
- B. Cationic exchange resin (2%)
- ✓ C. Enzyme inhibitor (55%)
- D. Essential fatty acids (5%)
- E. Pharmacologic vitamin (28%)
- F. Transcription factor ligand (4%)

Incorrect
Correct answer
C

55%
Answered correctly

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Explanation

Lipid-lowering therapy			
	LDL cholesterol	HDL cholesterol	Serum triglycerides

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Explanation

Lipid-lowering therapy			
	LDL cholesterol	**HDL cholesterol**	**Serum triglycerides**
Statins (atorvastatin, rosuvastatin, simvastatin)	↓↓	↑	↓
Fibrates (fenofibrate, gemfibrozil)	↓	↑	↓↓
Bile acid sequestrants (cholestyramine, colesevelam, colestipol)	↓	-	↑
Niacin	↓	↑↑	↓
Ezetimibe	↓	-	↓
Omega-3 fatty acids	-	↑	↓
Exercise & weight loss	↓	↑	↓↓
HDL = high-density lipoprotein; **LDL** = low-density lipoprotein.			

Patients with **low high-density lipoprotein (HDL) levels** (men <40 mg/dL, women <50 mg/dL) are at **increased risk for cardiovascular disease**. HDL is involved in reverse cholesterol transport; it helps to remove cholesterol from peripheral tissues and transports it to the liver for metabolism. This is accomplished through 2 different pathways. In the direct pathway, HDL delivers cholesterol esters directly to the liver via a scavenger receptor (SCARB1) on the hepatocyte cell membrane. In the indirect pathway, the cholesterol in HDL is transferred to low-

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Nonpharmacologic measures such as exercise, weight loss, and smoking cessation help to raise HDL levels and have significant cardiovascular benefits. In contrast, use of medications to raise HDL levels does **not** improve cardiovascular outcomes. Pharmacologic treatment of patients with low HDL levels should focus on lowering LDL cholesterol with **HMG-CoA reductase inhibitors** (statins), as these are the most effective lipid-lowering drugs for preventing cardiovascular events. Statins are indicated for secondary prevention in all patients with known atherosclerotic cardiovascular disease, **regardless** of baseline lipid levels.

(Choice A) Ezetimibe inhibits cholesterol absorption from the gastrointestinal tract, but evidence for reduction in cardiovascular events is mixed, and benefits over statin monotherapy are likely minimal. It may be considered for patients who cannot take statins due to significant hepatotoxicity or myopathy, or who have very high LDL levels despite statin therapy.

(Choice B) Cationic exchange resins (eg, cholestyramine) decrease LDL cholesterol by interfering with the enterohepatic circulation of bile acids. Bile acid binding resins can significantly increase triglyceride levels in patients with pre-existing hypertriglyceridemia. In addition, cationic exchange resins have not been proven to prevent cardiac events.

(Choice D) Omega-3 essential fatty acids are sometimes used for hyperlipidemia treatment. They cause a decrease in serum triglycerides and a slight increase in HDL concentration but do not appear to significantly reduce cardiovascular events.

(Choice E) Niacin (vitamin B3) given in pharmacologic doses is the most effective agent for raising HDL levels, but

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atherosclerotic cardiovascular disease, **regardless** of baseline lipid levels.

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(Choice B) Cationic exchange resins (eg, cholestyramine) decrease LDL cholesterol by interfering with the enterohepatic circulation of bile acids. Bile acid binding resins can significantly increase triglyceride levels in patients with pre-existing hypertriglyceridemia. In addition, cationic exchange resins have not been proven to prevent cardiac events.

(Choice D) Omega-3 essential fatty acids are sometimes used for hyperlipidemia treatment. They cause a decrease in serum triglycerides and a slight increase in HDL concentration but do not appear to significantly reduce cardiovascular events.

(Choice E) Niacin (vitamin B_3) given in pharmacologic doses is the most effective agent for raising HDL levels, but does not reduce the risk of cardiovascular events and is associated with adverse effects (eg, flushing, hyperglycemia). It is primarily used for patients who have failed other lipid-lowering drugs.

(Choice F) Fibrates activate peroxisomal proliferator-activated receptor alpha, a transcription factor that increases lipoprotein lipase activity. Fibrates decrease triglyceride levels and raise HDL levels. However, fibrates are inferior to statins for reducing cardiovascular events and are primarily used to prevent pancreatitis in patients with very high triglyceride levels.

Educational objective:
Although low HDL concentration is associated with increased cardiovascular risk, the use of medications to raise HDL levels does not improve cardiovascular outcomes. HMG-CoA reductase inhibitors (statins) lower total cholesterol and LDL levels. Statins are the most effective lipid-lowering drugs for primary and secondary prevention of cardiovascular events, regardless of baseline lipid levels.

References

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A 68-year-old man comes to the office to establish care. He has type 2 diabetes mellitus complicated by chronic kidney disease and peripheral sensory neuropathy. Medical history is notable for hypertension and coronary artery disease. The patient works for an agricultural supply company and spends most of his day walking and standing on a warehouse floor. Vital signs are normal. BMI is 34 kg/m^2. Examination of the lower extremities shows decreased sensation to monofilament testing below the knees bilaterally, with 2+ pitting edema, varicosities, and extensive skin discoloration, as shown below.

Which of the following is the most likely cause of the discoloration in this patient's skin?

- A. Arteriolar calcium phosphate deposition
- B. Cutaneous fungal infection

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Which

A. Arterial calcium phosphate deposition
B. Cutaneous fungal infection

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Which of the following is the most likely cause of the discoloration in this patient's skin?

- A. Arteriolar calcium phosphate deposition
- B. Cutaneous fungal infection
- C. Glycosaminoglycan accumulation
- D. Hemosiderin deposition
- E. Increased melanin production

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Which of the following is the most likely cause of the discoloration in this patient's skin?

- A. Arteriolar calcium phosphate deposition (9%)
- ✗ B. Cutaneous fungal infection (3%)
- C. Glycosaminoglycan accumulation (25%)
- ✓ D. Hemosiderin deposition (57%)
- E. Increased melanin production (3%)

Incorrect
Correct answer
D

57%
Answered correctly

35 secs
Time Spent

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✓ D. Hemosiderin deposition (57%)

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pigmentation

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This patient has varicose veins and edema in the lower extremities consistent with **chronic venous insufficiency**. Venous insufficiency is a multifactorial process; contributing factors include obstruction of venous return (eg, pregnancy, **obesity**), increased orthostatic pressure (eg, **prolonged standing**), decreased muscular compression (eg, sedentary lifestyle), and damaged venous valves (eg, due to deep vein thrombosis).

Over time, patients with venous insufficiency can develop extravasation of fluid, plasma proteins, and red cells, leading to chronic inflammation and **stasis dermatitis (SD)**. SD is characterized by erythema, induration, fibrosis, and deposition of **hemosiderin** (from breakdown of extravasated RBCs) manifesting as **reddish-brown discoloration**, as in this patient. Symptoms are typically bilateral and usually worse at or above the ankles. Complications of SD include poor wound healing and **ulceration**.

(Choice A) Deposition of calcium phosphate salts leads to calcific uremic arteriolopathy (calciphylaxis). This usually occurs in patients with end-stage renal disease receiving hemodialysis and presents with extremely painful nodules, plaques, and ulcers.

(Choice B) Tinea versicolor is a superficial fungal infection that causes primarily macular, rather than regional, pigment changes. It is typically found on the trunk and upper extremities rather than the ankles.

(Choice C) Dermal deposition of glycosaminoglycans is seen in patients with untreated hypothyroidism (ie, myxedema) and Graves disease (ie, thyroid dermopathy). It can manifest as induration and thickening of the skin over the shins.

(Choice E) Chronic radiation dermatitis is characterized by focal fibrosis and increased production of melanin. It is usually seen following external beam radiation therapy for cancer, and the findings are typically most notable in the treatment field.

Educational objective:
Stasis dermatitis is characterized by chronic erythema, fibrosis, and reddish-brown discoloration due to deposition of hemosiderin (from breakdown of extravasated red cells). Symptoms are typically bilateral, and usually worse at or above the ankles. Complications include poor wound healing, weeping, and ulceration.

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Calciphylaxis

*Skin ischemia and necrosis due to subcutaneous vascular calcification and thrombosis. ©UWorld

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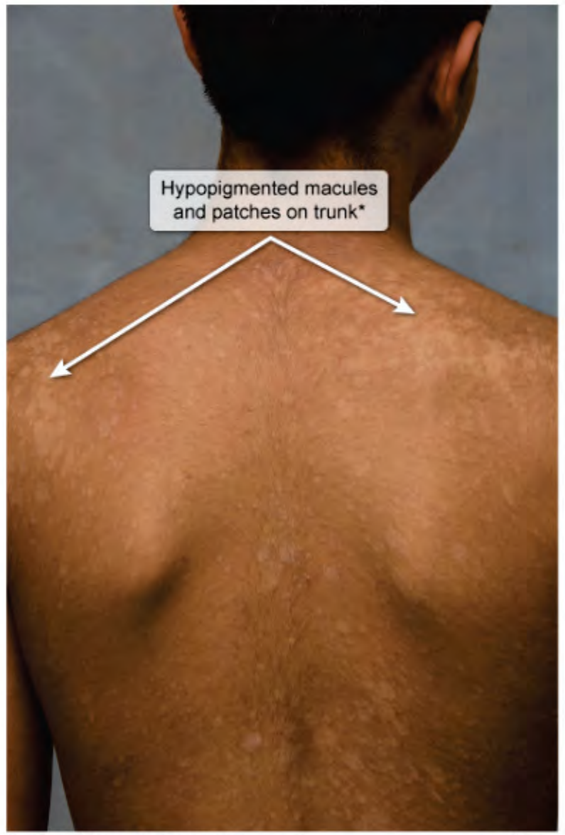
Tinea versicolor



*Due to *Malassezia* fungal infection.

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A 65-year-old man comes to the emergency department due to substernal chest discomfort and intermittent palpitations that began this morning. The patient has a history of coronary artery disease and stable angina that is managed medically. He takes high-dose metoprolol, atorvastatin, and low-dose aspirin. The patient reports that he has been traveling for the past week and ran out of his medications 2 days ago. Temperature is 37 C (98.6 F), blood pressure is 148/82 mm Hg, and pulse is 112/min. ECG reveals sinus tachycardia with a 1-mm ST segment depression in the lateral leads. Which of the following is most likely responsible for this patient's presentation?

- A. Increased levels of circulating catecholamines
- B. Predominant activation of alpha-adrenergic receptors
- C. Reduced activity of cyclic AMP–mediated messaging
- D. Reduced cellular sensitivity to circulating catecholamines
- E. Upregulation of beta-adrenergic receptors

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- ✗ A. Increased levels of circulating catecholamines (14%)
- B. Predominant activation of alpha-adrenergic receptors (4%)
- C. Reduced activity of cyclic AMP–mediated messaging (6%)
- D. Reduced cellular sensitivity to circulating catecholamines (1%)
- ✓ E. Upregulation of beta-adrenergic receptors (72%)

Incorrect
Correct answer
E

72%
Answered correctly

03 secs
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Explanation

This patient with elevated blood pressure, tachycardia, palpitations, and anginal pain following recent discontinuation of metoprolol is most likely experiencing **beta blocker withdrawal syndrome**. With changes in environmental stimulus, the feedback mechanisms in cells adjust the density of cell surface membrane receptors to regulate sensitivity to the stimulus. Prolonged beta-adrenergic blockade, for example, stimulates an increase in surface membrane expression of beta-adrenergic receptors, a process called **upregulation**. When beta-adrenergic blockade is abruptly withdrawn, the increased density of beta-adrenergic receptors creates an amplified response to circulating catecholamines (ie, **increased sensitivity**).

Because metoprolol is cardioselective and primarily blocks beta-1 receptors, abrupt cessation stimulates beta-1

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Because metoprolol is cardioselective and primarily blocks beta-1 receptors, abrupt cessation stimulates beta-1 receptor–mediated **increased heart rate** and **cardiac contractility**. There is also **increased blood pressure** due to increased cardiac output. These changes create **increased oxygen demand** that may cause ischemia (evidenced by ST depression on ECG) and trigger **angina** in patients with underlying coronary artery disease.

(Choice A) Beta blocker withdrawal symptoms result from an increase in the density of beta-adrenergic receptors, leading to an amplified response to circulating catecholamines. The level of circulating catecholamines is unchanged from baseline.

(Choice B) Alpha-adrenergic receptors are predominantly activated in the setting of beta blockade. On cessation of beta blocker therapy, there is redistribution of catecholamines to beta-adrenergic receptors.

(Choice C) Beta-adrenergic receptors function via cyclic AMP second messaging. The increased stimulation of beta-adrenergic receptors following beta blocker cessation leads to increased cyclic AMP activity.

(Choice D) Downregulation of surface membrane receptors occurs when cells are exposed to frequent agonist activity. For example, prolonged use of an alpha-1 agonist nasal spray (eg, phenylephrine) causes downregulation of alpha-1 receptors and reduced sensitivity to endogenous catecholamines. This is opposite to what is occurring in this patient.

Educational objective:
When exposed to a prolonged environmental stimulus, cells can adjust their sensitivity to the stimulus by upregulating or downregulating surface membrane receptors. Prolonged beta blockade leads to upregulation of beta-adrenergic receptors and increased sensitivity to circulating catecholamines, causing an enhanced beta-adrenergic response on abrupt beta blocker cessation (ie, beta blocker withdrawal syndrome).

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Because metoprolol is cardioselective and primarily blocks beta-1 receptors, abrupt cessation stimulates beta-1 receptor–mediated **increased heart rate** and **cardiac contractility**. There is also **increased blood pressure** due to increased cardiac output. These changes create **increased oxygen demand** that may cause ischemia (evidenced by ST depression on ECG) and trigger **angina** in patients with underlying coronary artery disease.

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References

- Abrupt withdrawal of beta-blocking agents in patients with arterial hypertension. Effect on blood pressure, heart rate and plasma catecholamines and prolactin

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- Abrupt withdrawal of beta-blocking agents in patients with arterial hypertension. Effect on blood pressure, heart rate and plasma catecholamines and prolactin

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Receptor stimulated	Change in second messenger	Primary effects
Alpha-1	↑ IP_3	• Peripheral vasoconstriction • Urethral constriction • Pupillary dilation
Alpha-2	↓ cAMP	• CNS sympatholytic • ↓ insulin release & intestinal motility
Beta-1	↑ cAMP	• ↑ Cardiac contractility & heart rate • ↑ Renin release by JG cells of kidney
Beta-2	↑ cAMP	• Peripheral vasodilation • Bronchodilation
Muscarinic-2	↓ cAMP	• ↓ Cardiac contractility & heart rate
Muscarinic-3	↑ IP_3	• Bronchoconstriction • ↑ insulin release & intestinal motility • Bladder contraction • Pupillary constriction • Peripheral vasodilation*

*Via release of nitric oxide.
cAMP = cyclic AMP; **IP_3** = inositol 1,4,5-triphosphate; **JG** = juxtaglomerular.

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A 65-year-old man is brought to the emergency department due to central chest pain for the past 2 hours. The patient describes the pain as "very severe" and "going back all the way to my spine." He has never had similar pain. Medical history is significant for hypertension; the patient has been nonadherent with prescribed medical therapy and follow-up visits. He has a 20-pack-year smoking history. Blood pressure is 195/101 mm Hg, and pulse is 93/min and regular. Chest examination reveals clear lungs. ECG shows sinus rhythm and voltage criteria for left ventricular hypertrophy. Portable chest x-ray reveals a widened mediastinum. The patient is immediately treated with intravenous esmolol. Which of the following is the most likely purpose of this patient's current therapy?

- A. Decreasing left ventricular contraction velocity
- B. Decreasing myocardial oxygen demand
- C. Decreasing peripheral vascular resistance
- D. Increasing left ventricular filling pressure
- E. Increasing left ventricular stroke volume

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- ✓ A. Decreasing left ventricular contraction velocity (43%)
- ✗ B. Decreasing myocardial oxygen demand (42%)
- C. Decreasing peripheral vascular resistance (9%)
- D. Increasing left ventricular filling pressure (1%)
- E. Increasing left ventricular stroke volume (3%)

Incorrect
Correct answer
A

43%
Answered correctly

03 secs
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Explanation

This patient's severe chest pain **radiating to the back** and a **widened mediastinum** on chest x-ray are consistent with **acute aortic dissection**. Uncontrolled **hypertension**, suggested by the patient's nonadherence with medical therapy and ECG evidence of left ventricular (LV) hypertrophy, is the strongest risk factor for aortic dissection.

With each LV contraction, the ejected stroke volume rapidly increases aortic pressure and generates shear stress on the aortic wall. Medical therapy for aortic dissection is aimed at **reducing aortic wall shear stress** to limit extension of the dissection. This is accomplished via **anti-impulse therapy** to decrease the rate of change in aortic blood pressure per time (ie, reduce dP/dt).

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Esmolol is a selective **beta-1 receptor blocker** that is given by intravenous infusion. It is first-line therapy for acute aortic dissection as it has a short half-life (allowing for easy dose titration) and reduces shear stress in 2 ways:

- Negative inotropy **decreases LV contraction velocity**. This decreases the rate of blood ejection and reduces stroke volume **(Choice E)** to decrease the rise in blood pressure with each contraction (ie, decreased rate and magnitude of blood pressure change).
- Negative chronotropy **decreases heart rate**, subjecting the aortic wall to fewer LV contractions per minute (ie, decreased number of blood pressure changes).

Once shear stress has been minimized with adequate reduction in LV contractility and heart rate, decreasing peripheral vascular resistance (PVR) to further reduce blood pressure is also beneficial. Because esmolol has little effect on PVR **(Choice C)**, vasodilators (eg, nitroprusside, nicardipine) may be added for this purpose. Vasodilators should never be given prior to a negative chronotropic agent because vasodilation causes reflex tachycardia that increases aortic wall shear stress.

(Choice B) Reduced LV contractility and heart rate decrease myocardial oxygen demand. Although this is the major goal of medical therapy for treating myocardial ischemia, reducing aortic wall shear stress is much more important for treating aortic dissection.

(Choice D) Reduced LV contractility and heart rate may slightly increase LV filling pressure (due to less blood being ejected with each contraction and increased LV filling time), but this effect is of minimal consequence in

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Educational objective:
The primary goal of medical therapy for acute aortic dissection is to reduce aortic wall shear stress by minimizing the change in aortic blood pressure per time (dP/dt). Esmolol, a selective beta-1 receptor blocker, decreases left ventricular contraction velocity and heart rate to reduce dP/dt.

References

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A 6-week-old infant is brought to the office due to difficulty feeding. The patient was born at 39 weeks gestation via spontaneous vaginal delivery. He is breastfeeding for 5 minutes per side every 3 hours. The patient has profuse sweating and difficulty breathing with feeds. Weight is at the 5th percentile. On examination, the patient is tachypneic. Cardiac examination shows a holosystolic murmur at the left lower sternal border; a palpable thrill is present at the left sternal border. Compared to a healthy infant, this patient likely has which of the following intracardiac pressure changes?

	Right atrium	Right ventricle	Left atrium	Left ventricle
◯ A.	Decreased	Increased	Unchanged	Unchanged
◯ B.	Increased	Decreased	Increased	Increased
◯ C.	Increased	Increased	Decreased	Decreased
◯ D.	Unchanged	Decreased	Increased	Increased
◯ E.	Unchanged	Increased	Increased	Increased

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	Right atrium	Right ventricle	Left atrium	Left ventricle	
A.	Decreased	Increased	Unchanged	Unchanged	(10%)
✗ B.	Increased	Decreased	Increased	Increased	(4%)
C.	Increased	Increased	Decreased	Decreased	(40%)
D.	Unchanged	Decreased	Increased	Increased	(6%)
✓ E.	Unchanged	Increased	Increased	Increased	(38%)

Incorrect
Correct answer
E

38%
Answered correctly

03 secs
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Explanation

Moderate or large ventricular septal defect	
Timeline	**Pathophysiologic changes**
In utero	• RV & LV pressures equal • No significant hemodynamic consequences

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Moderate or large ventricular septal defect	
Timeline	**Pathophysiologic changes**
In utero	• RV & LV pressures equal • No significant hemodynamic consequences
Postnatal transition	• ↓ PVR, ↑ SVR • Left-to-right shunting through VSD
Infancy	• RV volume overload/dilation • Pulmonary overcirculation • LA & LV volume overload/dilation
Late childhood	• Pulmonary arterial vascular thickening (↑ PVR) • Right-to-left shunting through VSD (ie, Eisenmenger syndrome)

LA = left atrium; **LV** = left ventricle; **PVR** = pulmonary vascular resistance; **RV** = right ventricle; **SVR** = systemic vascular resistance; **VSD** = ventricular septal defect.

This patient's **holosystolic murmur** along the lower left sternal border and a palpable thrill are consistent with a **ventricular septal defect** (VSD), an abnormal communication between the right ventricle (RV) and the left ventricle (LV). Although small VSDs are typically asymptomatic, large VSDs often present in early infancy with signs of **heart failure** (eg, poor feeding, sweating/tachypnea with feeds) and **failure to thrive** due to left-sided volume overload with inability to maintain adequate cardiac output.

In utero, vascular resistance against left- and right-sided cardiac outflow is nearly equal. At birth, once the placenta is no longer responsible for oxygenation, pulmonary vascular resistance drops and systemic vascular resistance increases.

In patients with VSD, this transition causes shunting of blood from the LV to the RV due to increased left-sided

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	• LA & LV volume overload/dilation
Late childhood	• Pulmonary arterial vascular thickening (↑ PVR) • Right-to-left shunting through VSD (ie, Eisenmenger syndrome)

LA = left atrium; **LV** = left ventricle; **PVR** = pulmonary vascular resistance; **RV** = right ventricle; **SVR** = systemic vascular resistance; **VSD** = ventricular septal defect.

This patient's **holosystolic murmur** along the lower left sternal border and a palpable thrill are consistent with a **ventricular septal defect** (VSD), an abnormal communication between the right ventricle (RV) and the left ventricle (LV). Although small VSDs are typically asymptomatic, large VSDs often present in early infancy with signs of **heart failure** (eg, poor feeding, sweating/tachypnea with feeds) and **failure to thrive** due to left-sided volume overload with inability to maintain adequate cardiac output.

In utero, vascular resistance against left- and right-sided cardiac outflow is nearly equal. At birth, once the placenta is no longer responsible for oxygenation, pulmonary vascular resistance drops and systemic vascular resistance increases.

In patients with VSD, this transition causes shunting of blood from the LV to the RV due to increased left-sided cardiac pressure relative to right-sided pressure. The unrestricted blood flow to the RV causes **increased pressure** in the **RV (Choices B and D)** and allows a large volume of blood from the LV to reenter the pulmonary circulation; this high volume of recirculating blood leads to left-sided volume overload with **increased pressure** in the **LV** and **left atrium (Choices A and C)**. Right atrial pressure is unchanged.

Educational objective:
Ventricular septal defect causes left-to-right shunting with increased right ventricular pressure and, if large, left-sided volume overload with increased left ventricular and left atrial pressures. A holosystolic murmur is characteristic, and failure to thrive and signs of heart failure (eg, poor feeding, diaphoresis) can occur.

References

- Ventricular septal defect

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- Pulmonary arterial vascular thickening

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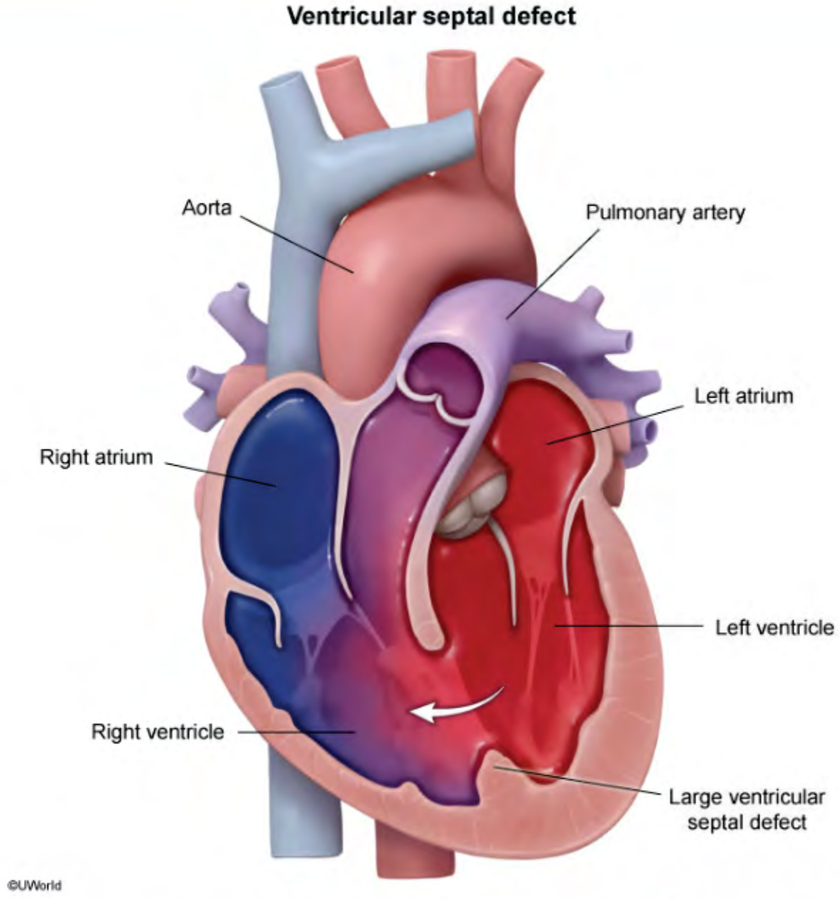

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	• LA & LV volume overload/dilation
	• Pulmonary arterial vascular thickening

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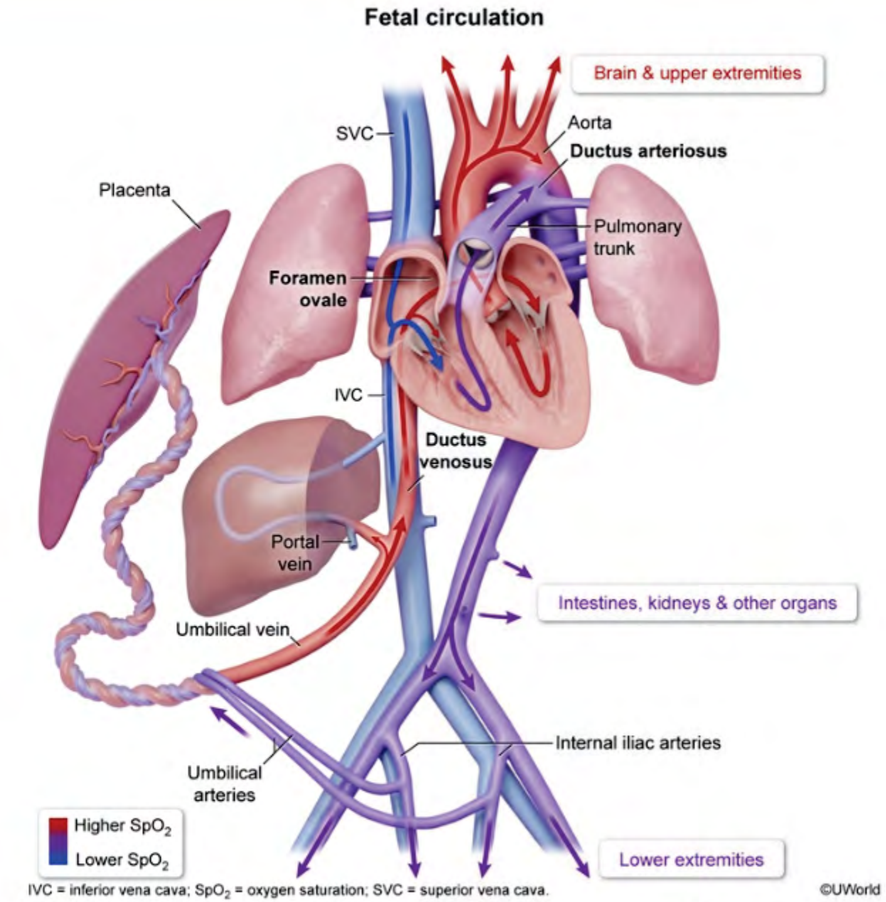

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References

- Ventricular septal defect

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A newborn is transferred to the neonatal intensive care unit 4 hours after delivery due to cyanosis. The infant was born via a spontaneous vaginal delivery at 41 weeks gestation to a 22-year-old gravida 2 para 2 woman. The pregnancy and delivery were uncomplicated. Three hours after delivery, circumoral cyanosis was noted after the infant was breastfed. Pulse oximetry readings were 98% in the right hand and 83% in the left foot. On physical examination, the patient has subcostal retractions and grunting. Cardiac examination reveals a 2/6 systolic murmur at the left lower sternal border. Femoral pulses are strong bilaterally. Which of the following is the most likely cause of this infant's cyanosis?

- A. Left-to-right shunt via atrial septal defect
- B. Left-to-right shunt via patent ductus arteriosus
- C. Left-to-right shunt via ventricular septal defect
- D. Right-to-left shunt via atrial septal defect
- E. Right-to-left shunt via patent ductus arteriosus
- F. Right-to-left shunt via ventricular septal defect

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- ✗ A. Left-to-right shunt via atrial septal defect (1%)
- B. Left-to-right shunt via patent ductus arteriosus (11%)
- C. Left-to-right shunt via ventricular septal defect (14%)
- D. Right-to-left shunt via atrial septal defect (2%)
- ✓ E. Right-to-left shunt via patent ductus arteriosus (36%)
- F. Right-to-left shunt via ventricular septal defect (33%)

Incorrect
Correct answer
E

36%
Answered correctly

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Explanation

Persistent pulmonary hypertension of the newborn	
Pathogenesis	• Abnormal persistence of elevated fetal pulmonary vascular resistance • Right-to-left shunting across ductus arteriosus

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Explanation

Persistent pulmonary hypertension of the newborn	
Pathogenesis	• Abnormal persistence of elevated fetal pulmonary vascular resistance • Right-to-left shunting across ductus arteriosus
Risk factors	• Lung hypoplasia (eg, congenital diaphragmatic hernia) • Meconium aspiration syndrome • Infection (eg, neonatal pneumonia)
Examination	• ↓ Postductal relative to preductal oxygen saturation • Respiratory distress & cyanosis • Prominent S2
Treatment	• Oxygenation & ventilation • Inhaled nitric oxide (pulmonary vasodilator)

This infant has **central cyanosis**, a condition characterized by blue discoloration of the mucous membranes and/or skin (not confined to the distal extremities). Central cyanosis is caused by circulation of deoxygenated hemoglobin because of a pulmonary, cardiac, or hematologic condition.

In this case, the neonate also has a **postductal** (left foot) oxygen saturation level that is significantly **lower** than the **preductal saturation** (right hand) measurement. This **differential cyanosis** (or differential hypoxemia) suggests that some deoxygenated blood from the pulmonary artery is flowing to the aorta and mixing with oxygenated blood from the left ventricle. In other words, this patient has an extracardiac **right-to-left shunt** across a patent ductus arteriosus (PDA), a condition typically associated with **persistent pulmonary hypertension of the**

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This infant has **central cyanosis**, a condition characterized by blue discoloration of the mucous membranes and/or skin (not confined to the distal extremities). Central cyanosis is caused by circulation of deoxygenated hemoglobin because of a pulmonary, cardiac, or hematologic condition.

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PPHN results from failure of pulmonary vascular resistance (PVR) to decline following birth, and is more likely given this patient's strong femoral pulses (as opposed to the diminished pulses seen in left-sided outflow tract obstruction). In PPHN, suprasystemic pulmonary arterial pressure drives deoxygenated blood through the PDA to the aorta. Tricuspid regurgitation, which can occur in PPHN due to elevated PVR leading to tricuspid valve insufficiency, is the likely cause of this neonate's murmur. Patients also typically have respiratory distress (eg, grunting, retractions) within hours of birth due to an underlying pulmonary pathology (eg, pulmonary hypoplasia, meconium aspiration).

(Choices A, B, and C) Left-to-right shunts through septal defects and PDAs are characterized by oxygenated blood crossing back into the right-sided circulation. Therefore, cyanosis does not occur.

(Choices D and F) Examples of intracardiac right-to-left shunts include a patent foramen ovale in PPHN and a ventricular septal defect in tetralogy of Fallot. Because the mixing of deoxygenated and oxygenated blood occurs within the heart in these cases, preductal and postductal saturations would be *equally* low.

Educational objective:
Differential cyanosis (ie, postductal < preductal oxygen saturation) in a neonate suggests right-to-left shunting across a patent ductus arteriosus, such as in persistent pulmonary hypertension of the newborn (PPHN). Patients with PPHN typically have an underlying pulmonary disorder with respiratory distress and strong femoral pulses.

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This infant has **central cyanosis**, a condition characterized by blue discoloration of the mucous membranes and/or skin (not confined to the distal extremities). Central cyanosis is caused by circulation of deoxygenated hemog

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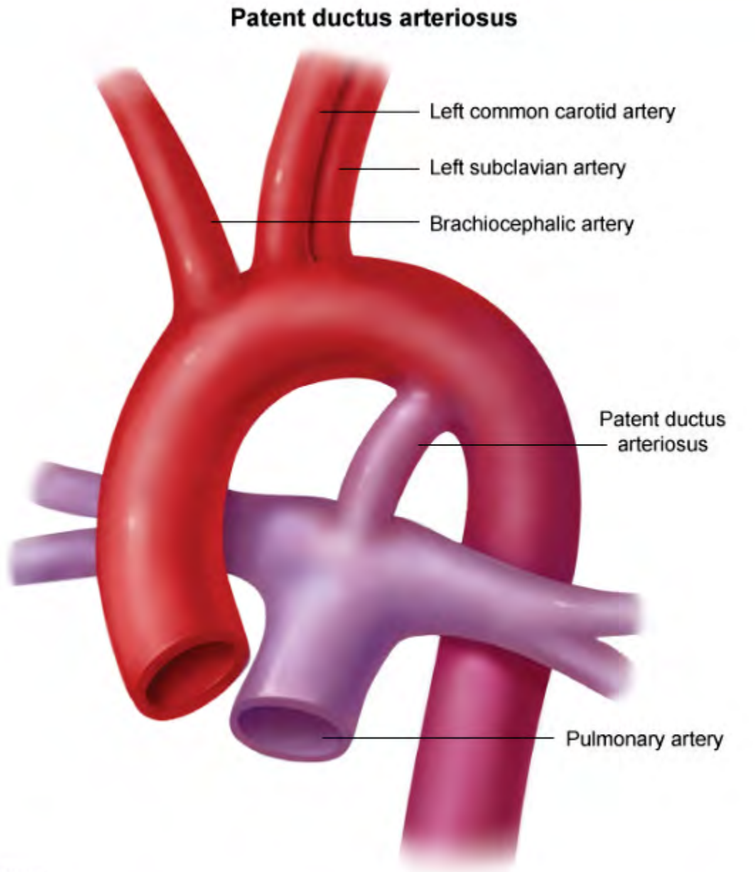

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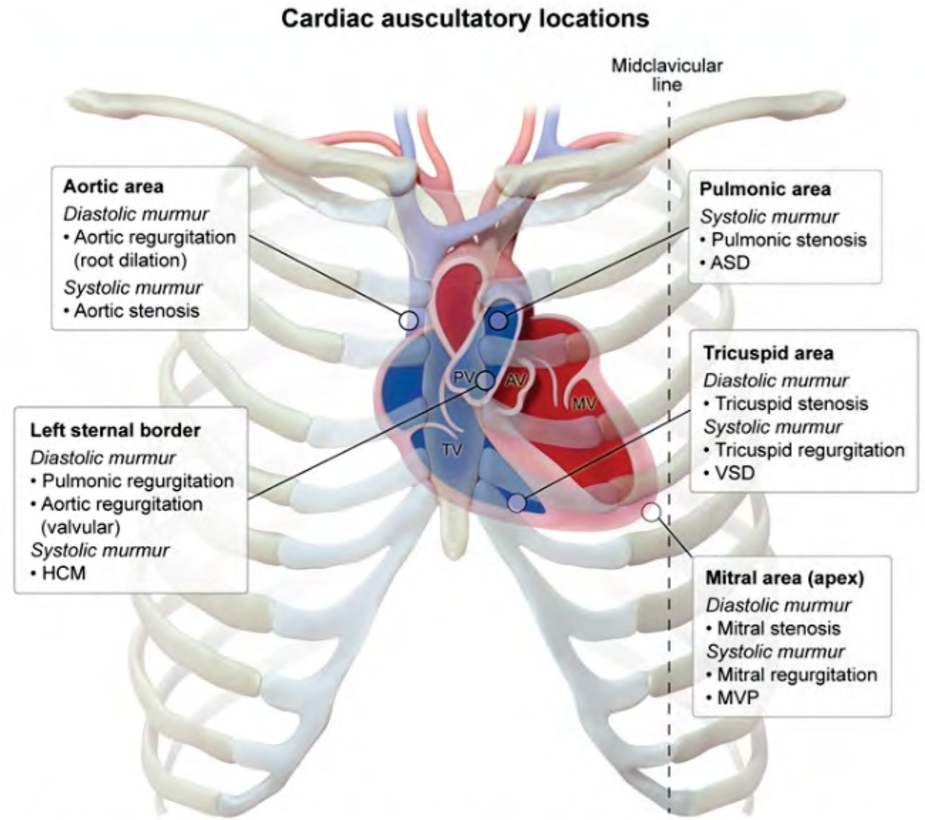

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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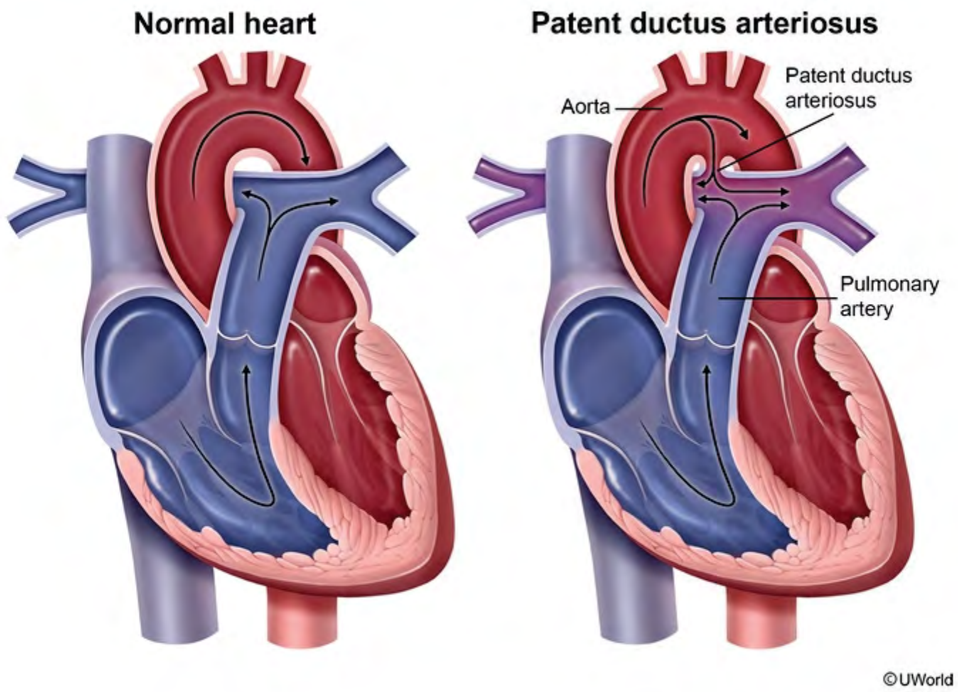

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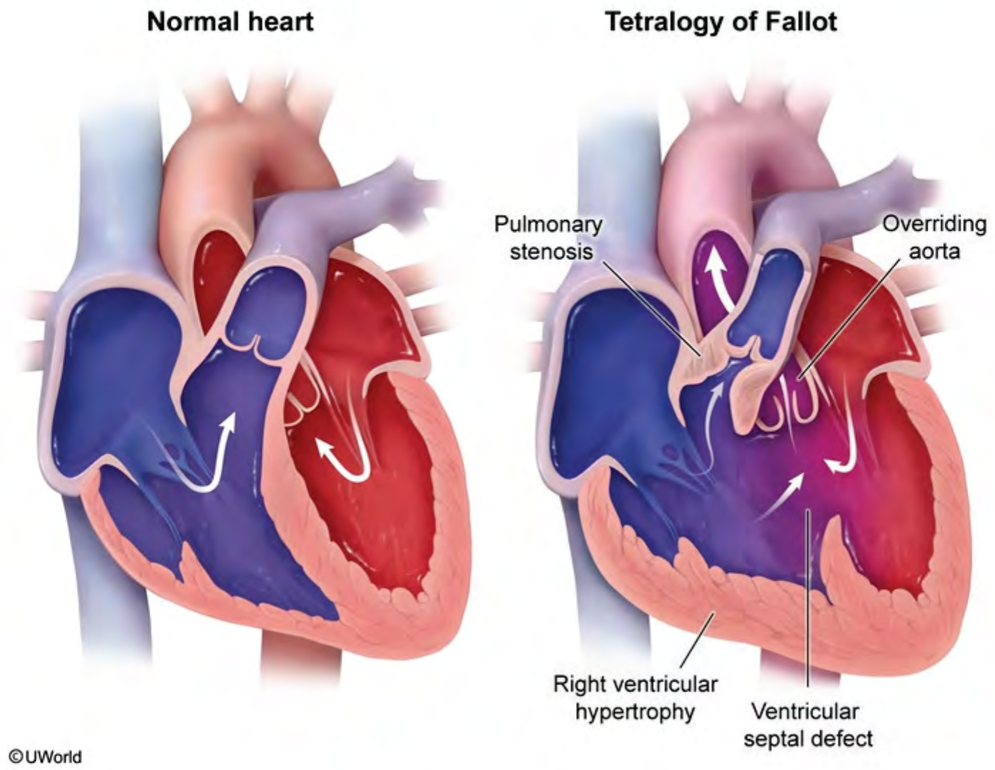

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A 72-year-old man comes to the office due to palpitations, fatigue, and dyspnea for the past 2 weeks. The patient experienced an episode of atrial fibrillation 4 years ago during hospitalization for pneumonia; the episode spontaneously resolved 2 days later. Since then, he has had 3 episodes of symptomatic atrial fibrillation that self-terminated within a week. Other medical history includes hypertension, type 2 diabetes mellitus, and chronic heart failure. Blood pressure is 130/84 mm Hg and pulse is 110/min. The lungs are clear to auscultation. Cardiac examination demonstrates tachycardia with irregularly irregular heartbeats. ECG confirms atrial fibrillation. The patient's ventricular rate is controlled with beta blocker therapy, but the arrhythmia fails to resolve as in previous episodes. Which of the following factors is most likely involved in the progression of this patient's arrhythmia?

- A. Abnormal atrioventricular nodal pathway
- B. Atrial structural remodeling
- C. Cardiac autonomic neuropathy
- D. Conduction system fibrosis
- E. Enhanced sinoatrial node automaticity

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- ✗ A. Abnormal atrioventricular nodal pathway (22%)
- ✓ B. Atrial structural remodeling (54%)
- C. Cardiac autonomic neuropathy (4%)
- D. Conduction system fibrosis (9%)
- E. Enhanced sinoatrial node automaticity (9%)

Incorrect
Correct answer
B

54%
Answered correctly

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Explanation

Atrial fibrillation	
Pathogenesis	• Atrial remodeling due to: ◦ Age-related myocardial changes ◦ Atrial enlargement from heart disease (eg, HTN, MS) • Foci of rapid electrical activity: ◦ Commonly originate in pulmonary veins

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Explanation

Atrial fibrillation	

Pathogenesis	• Atrial remodeling due to:
	  ◦ Age-related myocardial changes
	  ◦ Atrial enlargement from heart disease (eg, HTN, MS)
	• Foci of rapid electrical activity:
	  ◦ Commonly originate in pulmonary veins
	  ◦ Alcohol intake & ↑ sympathetic drive may contribute

Clinical presentation	• Asymptomatic or palpitations ± dyspnea, fatigue • Irregularly irregular pulse • May present with thromboembolic event (eg, stroke)
Diagnosis	• ECG shows variable R-R intervals & no P waves • Continuous ECG often needed for paroxysmal disease
HTN = hypertension; **MS** = mitral stenosis.	

This patient's **progression** from paroxysmal **atrial fibrillation** (resolving spontaneously within 7 days) to persistent atrial fibrillation (lasting >7 days) is most attributable to **atrial remodeling**. The development of atrial fibrillation is primarily driven by both **structural** and **electrical conduction** changes in the atrial myocardium that predispose to the initiation and maintenance of electrical reentrant circuits and/or ectopic foci:

- The structural component of atrial remodeling likely involves comorbidities that lead to chronic **atrial stretching and dilation**. These include hypertension, heart failure, and mitral valve disease. Physiologic age-related atrial dilation also contributes.
- The electrical conduction component likely involves age-related changes as well as previous exposure to atrial fibrillation. During atrial fibrillation, the conduction system undergoes changes that **facilitate propagation** of the arrhythmia; over time it becomes more difficult to reverse (ie, atrial fibrillation begets atrial fibrillation).

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primarily driven by both **structural** and **electrical conduction** changes in the atrial myocardium that predispose to the initiation and maintenance of electrical reentrant circuits and/or ectopic foci:

- The structural component of atrial remodeling likely involves comorbidities that lead to chronic **atrial stretching and dilation**. These include hypertension, heart failure, and mitral valve disease. Physiologic age-related atrial dilation also contributes.
- The electrical conduction component likely involves age-related changes as well as previous exposure to atrial fibrillation. During atrial fibrillation, the conduction system undergoes changes that **facilitate propagation** of the arrhythmia; over time it becomes more difficult to reverse (ie, atrial fibrillation begets atrial fibrillation).

As atrial remodeling progresses, the atria become increasingly susceptible to electrical triggers (eg, ectopic foci in pulmonary veins) that initiate fibrillation and more likely to maintain persistently disorganized electrical activity.

(Choice A) An abnormal pathway in the atrioventricular node is the typical cause of atrioventricular nodal reentrant tachycardia. This arrhythmia most commonly affects young patients with an otherwise normal heart.

(Choice C) Cardiac autonomic neuropathy, which can develop in patients with Parkinson disease or poorly controlled diabetes mellitus, typically leads to an impaired sympathetic cardiac response. This often results in exercise intolerance and orthostasis but does not significantly contribute to cardiac arrhythmia.

(Choice D) Conduction system fibrosis (often age-related) is a primary contributor to bradyarrhythmias, including sinus bradycardia (eg, sick sinus syndrome) and atrioventricular block.

(Choice E) Enhanced sinoatrial node automaticity causes sinus tachycardia (eg, physiologic with exercise). During atrial fibrillation, electrical signals in the sinoatrial node are suppressed by widespread disorganized electrical activity throughout the atria.

Educational objective:
Atrial remodeling is the major pathogenic contributor to atrial fibrillation. Age and comorbidities that cause atrial dilation (eg, hypertension, heart failure, mitral valve disease) cause atrial structural changes that predispose to atrial fibrillation development. In addition, age and previous atrial fibrillation cause atrial conduction system changes that further propagate atrial fibrillation.

References

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primarily driven by both **structural** and **electrical conduction** changes in the atrial myocardium that predispose to the initiation and maintenance of electrical reentrant circuits and/or ectopic foci:

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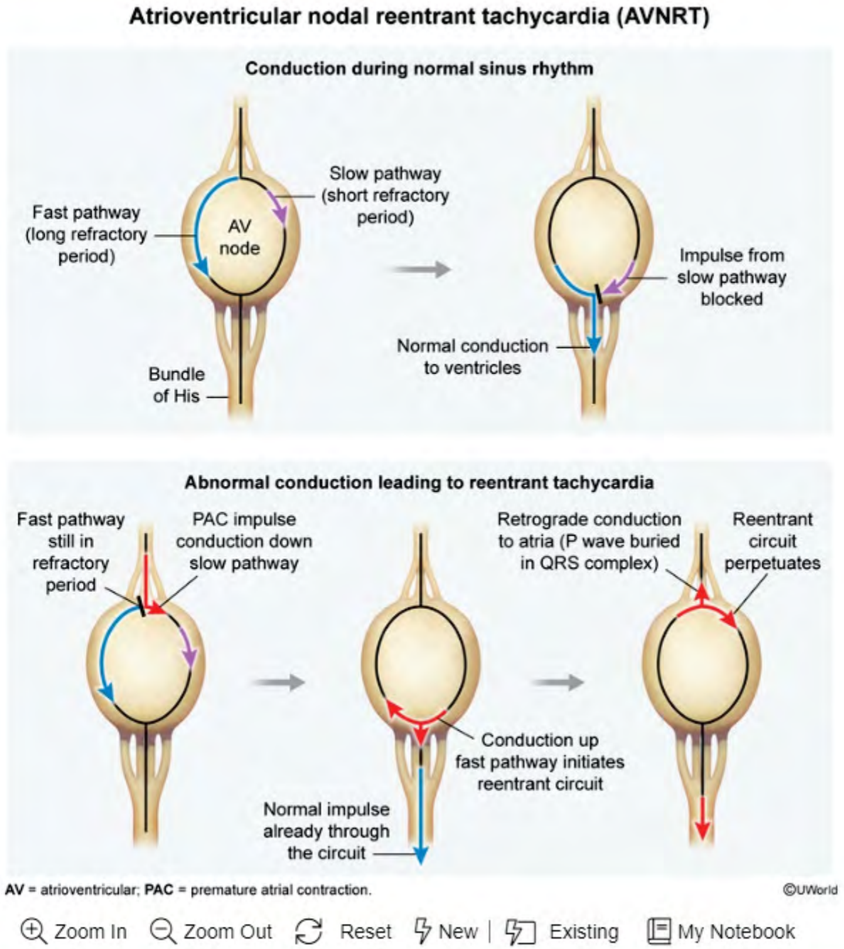

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A 68-year-old man with a history of permanent atrial fibrillation comes to the office for follow-up. He has been having symptoms due to ineffective ventricular rate control despite aggressive medical therapy. On examination, the patient's heart rate is 125/min and irregular. ECG shows atrial fibrillation with rapid ventricular response. A catheter-based radiofrequency ablation of the atrioventricular node with placement of a permanent ventricular pacemaker is planned. The ablation tip should be positioned at which of the following locations?

- A. Between the tricuspid valve and the inferior vena cava opening
- B. Interatrial septum near the opening of the coronary sinus
- C. Left atrium near the opening of the pulmonary veins
- D. The posterior border of the fossa ovalis
- E. Upper part of crista terminalis near the superior vena cava opening

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- ✗ A. Between the tricuspid valve and the inferior vena cava opening (17%)
- ✓ B. Interatrial septum near the opening of the coronary sinus (45%)
- C. Left atrium near the opening of the pulmonary veins (20%)
- D. The posterior border of the fossa ovalis (7%)
- E. Upper part of crista terminalis near the superior vena cava opening (9%)

Incorrect
Correct answer
B

45%
Answered correctly

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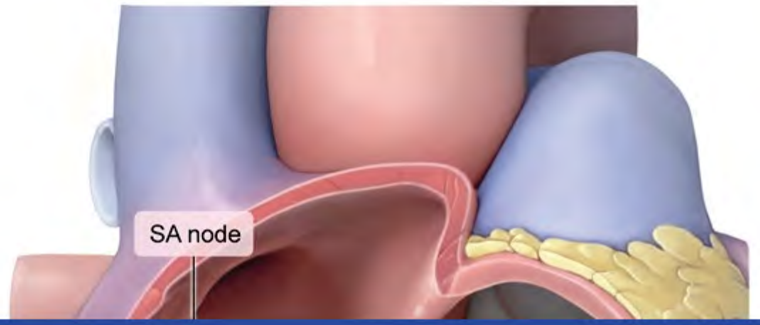
Explanation

Right atrial anatomy



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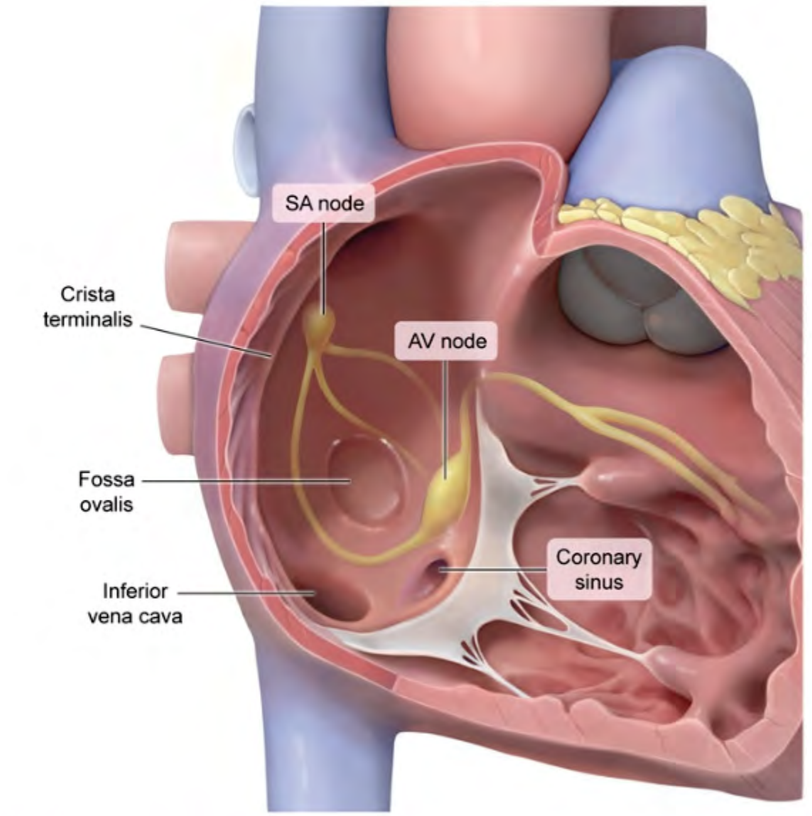
Right atrial anatomy



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The **atrioventricular (AV) node** controls the rate at which atrial impulses are conducted to the ventricles. In patients with atrial fibrillation, continuous stimulation of the AV node can sometimes lead to a **rapid ventricular rate**, which can cause hemodynamic instability and (if persistent) tachycardia-induced cardiomyopathy. In order to prevent these complications, patients with a rapid ventricular response sometimes undergo **radiofrequency ablation** of the AV node. The AV node is located on the endocardial surface of the right atrium, **near** the insertion of the septal leaflet of the **tricuspid valve** and the orifice of the **coronary sinus**.

(Choices A and D) The isthmus between the inferior vena cava and the tricuspid annulus is the site of radiofrequency ablation for atrial flutter. The fossa ovalis is the name given to the foramen ovale (fetal opening between the right and left atria) once it is closed. However, neither option refers to the location of the AV node.

(Choice C) The pulmonary vein ostia are often the origination site of the aberrant electrical activity that triggers atrial fibrillation, and catheter ablation of these trigger sites (pulmonary vein isolation) can be used to prevent recurrent atrial fibrillation. However, this patient has already developed permanent atrial fibrillation and is undergoing an ablation procedure involving the AV node.

(Choice E) The sinoatrial node is located in the upper anterior right atrium at the opening of the superior vena cava.

Educational objective:
The atrioventricular node is located on the endocardial surface of the right atrium, near the insertion of the septal leaflet of the tricuspid valve and the orifice of the coronary sinus.

References

- Anatomy and electrophysiology of the human AV node.
- The anatomy of the cardiac conduction system.

Anatomy	Cardiovascular System	Av node
Subject	System	Topic

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A 32-year-old man is brought to the emergency department with a several-day history of high-grade fever, dyspnea, and fatigue. The patient's temperature is 39.4 C (102.9 F), blood pressure is 122/70 mm Hg, and pulse is 102/min and regular. Physical examination reveals a new holosystolic murmur with a blowing quality that is best heard over the cardiac apex. Skin examination shows nontender lesions on the palms. A representative lesion is shown in the image below:

Which of the following do this patient's skin lesions most likely represent?

- A. Areas of local vascular growth
- B. Foci of granulomatous inflammation
- C. Immune complex-mediated vascular lesions
- D. Microemboli to skin vessels
- E. Primary infection foci

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- B. Foci of granulomatous inflammation
- C. Immune complex-mediated vascular lesions
- D. Microemboli to skin vessels
- E. Primary infection foci

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heard over the cardiac apex. Skin examination shows nontender lesions on the palms. A representative lesion is shown in the image below:

Which of the following do this patient's skin lesions most likely represent?

- A. Areas of local vascular growth (1%)
- ✗ B. Foci of granulomatous inflammation (4%)
- C. Immune complex-mediated vascular lesions (34%)
- ✓ D. Microemboli to skin vessels (51%)
- E. Primary infection foci (6%)

Incorrect
Correct answer
D

51%
Answered correctly

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Time Spent

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Explanation

Vascular & immunologic manifestations of infective endocarditis	
Vascular phenomena	• Systemic emboli (cerebral, pulmonary, or splenic infarcts) • Mycotic aneurysm • Janeway lesions: macular, erythematous, **nontender** lesions on the palms & soles
Immunologic phenomena	• Osler nodes: **painful**, violaceous nodules seen on the fingertips & toes • Roth spots: edematous & hemorrhagic lesions of the retina

This patient's presentation (fever, dyspnea, and a new blowing holosystolic murmur best heard at the cardiac apex) is suggestive of mitral regurgitation due to **infective endocarditis** (IE). Patients with IE can develop several peripheral cutaneous or mucocutaneous stigmata of endocarditis including petechiae, splinter hemorrhages, Roth spots, Janeway lesions, and Osler nodes.

Janeway lesions are nontender, macular, and erythematous lesions typically located on the palms and soles. They are the result of septic embolization from valvular vegetations and are composed of bacteria, neutrophils (microabscesses), necrotic material, and subcutaneous hemorrhage.

(Choice A) A neoplastic proliferation of cells of vascular origin (eg, Kaposi sarcoma) cannot be ruled out based on the appearance of this patient's skin lesions alone. However, the overall clinical presentation is highly suggestive of bacterial endocarditis as the unifying diagnosis.

(Choice B) Granulomatous inflammation can produce cutaneous lesions in miliary tuberculosis, sarcoidosis, and leprosy. However, the lesions do not localize preferentially to the palms.

(Choice C) Osler nodes are tender, violaceous nodules typically located in the pulp of fingers and toes. The pathogenesis of Osler nodes involves immune-complex deposition in skin vessels.

(Choice E) The lesions on the palms could represent primary foci of infection as might occur following focal skin abrasions/puncture or from sporotrichosis. However, this patient's new holosystolic murmur is more suggestive of

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Immunologic phenomena	• Osler nodes: **painful**, violaceous nodules seen on the fingertips & toes • Roth spots: edematous & hemorrhagic lesions of the retina

This patient's presentation (fever, dyspnea, and a new blowing holosystolic murmur best heard at the cardiac apex) is suggestive of mitral regurgitation due to **infective endocarditis** (IE). Patients with IE can develop several peripheral cutaneous or mucocutaneous stigmata of endocarditis including petechiae, splinter hemorrhages, Roth spots, Janeway lesions, and Osler nodes.

Janeway lesions are nontender, macular, and erythematous lesions typically located on the palms and soles. They are the result of septic embolization from valvular vegetations and are composed of bacteria, neutrophils (microabscesses), necrotic material, and subcutaneous hemorrhage.

(Choice A) A neoplastic proliferation of cells of vascular origin (eg, Kaposi sarcoma) cannot be ruled out based on the appearance of this patient's skin lesions alone. However, the overall clinical presentation is highly suggestive of bacterial endocarditis as the unifying diagnosis.

(Choice B) Granulomatous inflammation can produce cutaneous lesions in miliary tuberculosis, sarcoidosis, and leprosy. However, the lesions do not localize preferentially to the palms.

(Choice C) Osler nodes are tender, violaceous nodules typically located in the pulp of fingers and toes. The pathogenesis of Osler nodes involves immune-complex deposition in skin vessels.

(Choice E) The lesions on the palms could represent primary foci of infection as might occur following focal skin abrasions/puncture or from sporotrichosis. However, this patient's new holosystolic murmur is more suggestive of mitral valve IE.

Educational objective:
Janeway lesions are nontender, macular, and erythematous lesions typically located on the palms and soles of patients with acute infective endocarditis and are the result of septic embolization from valvular vegetations.

Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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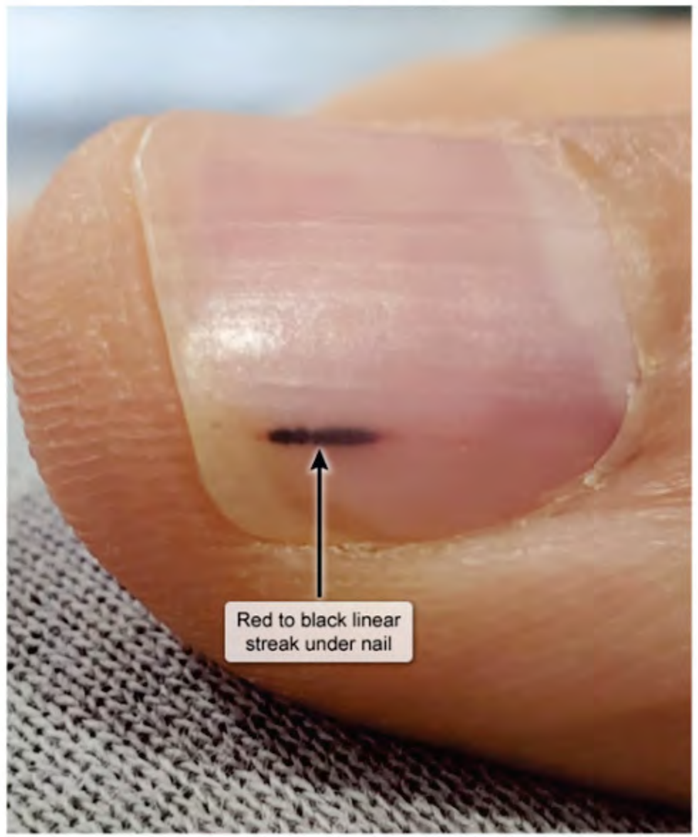
Splinter hemorrhage



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Osler node

*Manifestation of infective endocarditis.

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Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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Janeway lesion

*Manifestation of infective endocarditis.

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Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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Kaposi sarcoma

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Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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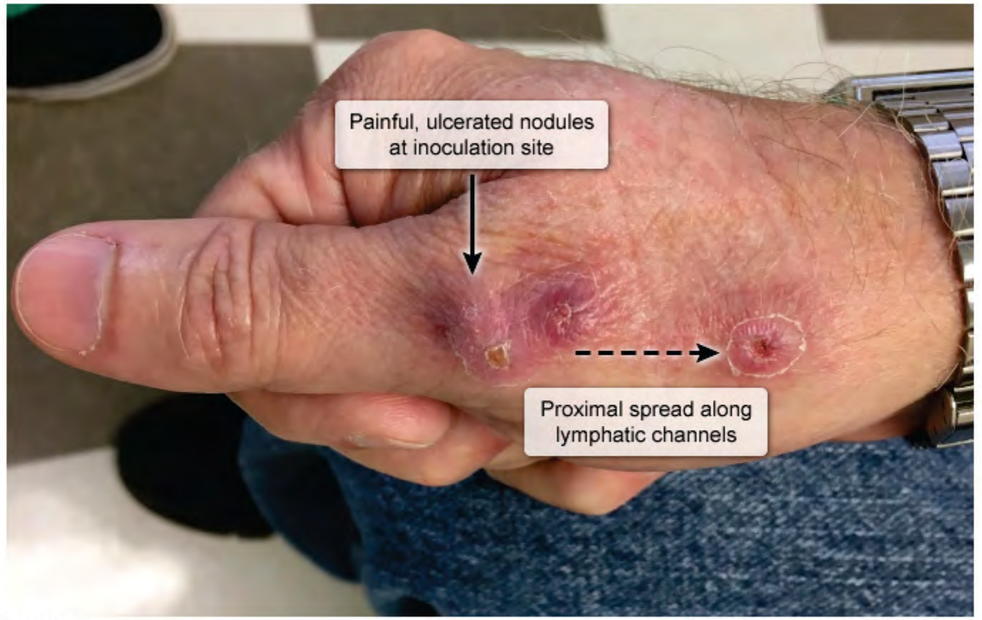
Sporotrichosis



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Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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A 45-year-old man comes to the emergency department due to shortness of breath and fatigue that has progressed over the last 2 weeks. The patient was diagnosed with nonischemic cardiomyopathy 2 years ago and has not been adherent with physician visits and his medical regimen. Blood pressure is 106/58 mm Hg, and pulse is 88/min and regular. The patient appears uncomfortable when lying flat. Jugular venous pressure is elevated. Bibasilar crackles are heard on lung auscultation and an S3 is present. There is 1+ bilateral lower extremity pitting edema. Milrinone infusion is initiated. Which of the following is the most likely response to this therapy?

- A. Angioedema
- B. Antiarrhythmic action
- C. Atrioventricular conduction block
- D. Decreased renal perfusion
- E. Increased right atrial pressure
- F. Vasodilation

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- ✗ A. Angioedema (2%)
- B. Antiarrhythmic action (9%)
- C. Atrioventricular conduction block (3%)
- D. Decreased renal perfusion (8%)
- E. Increased right atrial pressure (12%)
- ✓ F. Vasodilation (63%)

Incorrect
Correct answer
F

63%
Answered correctly

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Time Spent

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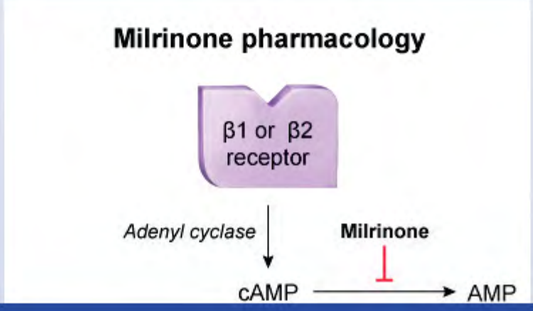
Explanation



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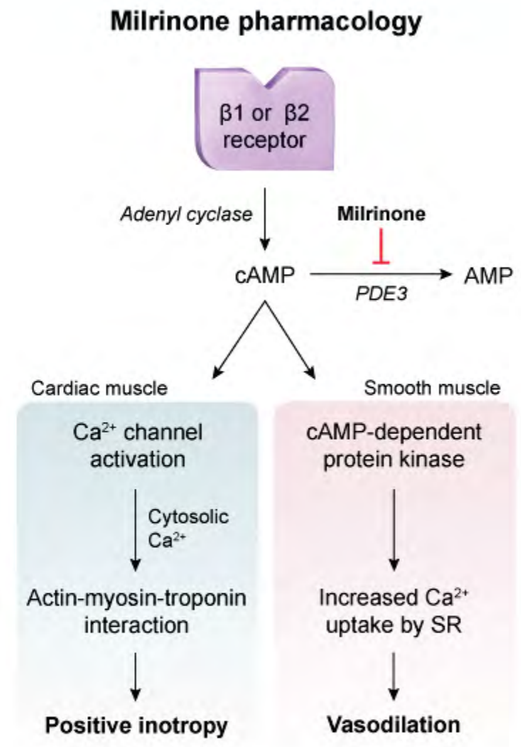

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cAMP = cyclic adenosine monophosphate;
PDE-3 = phosphodiesterase-3; **SR** = sarcoplasmic reticulum.
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Milrinone is a selective **phosphodiesterase (PDE)-3 enzyme inhibitor** that can be used in patients with refractory heart failure due to left ventricular systolic dysfunction. The PDE-3 enzyme is normally responsible for degrading cyclic adenosine monophosphate (cAMP) to AMP; the inhibition of cAMP degradation via milrinone has 2 positive effects on heart failure:

- In cardiomyocytes, intracellular calcium influx is increased, which increases cardiac contractility (**positive inotropy**) to improve stroke volume and cardiac output.
- In vascular smooth muscle, uptake of calcium by the sarcoplasmic reticulum is increased, which reduces calcium-myosin light chain kinase interaction to stimulate relaxation and **vasodilation**. Venous vasodilation **reduces preload** and arterial vasodilation **reduces afterload** to provide a cumulative reduction in cardiac work.

Although the vasodilatory action of milrinone can lead to hypotension, this effect is often compensated for by an increase in stroke volume that maintains blood pressure.

(Choice A) Angioedema refers to localized subcutaneous swelling that affects the submucosal tissues of the face, mouth, throat, and genitalia. It is typically seen as a side effect of ACE inhibitors due to inhibition of bradykinin degradation.

(Choice B) Milrinone does not have an antiarrhythmic effect on the myocardium. In fact, its positive inotropic effect may induce ventricular arrhythmias in some patients.

(Choice C) Atrioventricular conduction block can be caused by toxicity due to digoxin, nondihydropyridine calcium channel blockers, or beta blockers; however, PDE inhibitors have minimal chronotropic effect (positive or negative).

(Choice D) Renal perfusion is increased with milrinone due to an increase in cardiac output.

(Choice E) Right atrial pressure is decreased with milrinone due to venous dilation and a consequent reduction in cardiac venous return (preload).

Educational objective:
Milrinone is a phosphodiesterase-3 inhibitor that reduces the degradation of cyclic adenosine monophosphate to

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refractory heart failure due to left ventricular systolic dysfunction. The PDE-3 enzyme is normally responsible for degrading cyclic adenosine monophosphate (cAMP) to AMP; the inhibition of cAMP degradation via milrinone has 2 positive effects on heart failure:

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Educational objective:
Milrinone is a phosphodiesterase-3 inhibitor that reduces the degradation of cyclic adenosine monophosphate to provide 2 beneficial effects for treating systolic heart failure. Calcium influx into cardiomyocytes is increased, which increases cardiac contractility. In addition, calcium-myosin light chain kinase interaction is reduced, which causes vasodilation and reduces cardiac preload and afterload.

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A 64-year-old man comes to the office due to exertional chest pain over the last 6 months. He is a lifelong 1 pack per day cigarette smoker and has a history of type 2 diabetes mellitus and peripheral artery disease. The patient undergoes treadmill exercise stress testing and develops substernal chest pain on moderate exertion accompanied by ECG changes that resolve immediately upon rest. He refuses invasive cardiac testing. The patient is started on low-dose aspirin therapy for secondary prevention of cardiovascular disease but experiences shortness of breath and wheezing with the medication. Which of the following is the best alternate therapy for this patient?

- A. Apixaban
- B. Cilostazol
- C. Clopidogrel
- D. Enoxaparin
- E. Eptifibatide
- F. Naproxen
- G. Warfarin

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- ✗ A. Apixaban (2%)
- B. Cilostazol (6%)
- ✓ C. Clopidogrel (70%)
- D. Enoxaparin (5%)
- E. Eptifibatide (1%)
- F. Naproxen (5%)
- G. Warfarin (7%)

Incorrect
Correct answer
C

70%
Answered correctly

09 secs
Time Spent

2023
Version

Explanation

This patient's clinical history is consistent with stable angina (**angina pectoris**) due to underlying coronary heart disease (CHD). Patients with stable angina should be started on aspirin to decrease the risk of adverse cardiovascular events. Aspirin impairs prostaglandin synthesis by irreversibly inhibiting cyclooxygenase (COX). Inhibition of COX-1 in platelets prevents synthesis of thromboxane A2, a potent stimulator of platelet aggregation and vasoconstriction. This helps reduce the risk of occlusive thrombus formation and subsequent myocardial infarction.

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Explanation

This patient's clinical history is consistent with stable angina (**angina pectoris**) due to underlying coronary heart disease (CHD). Patients with stable angina should be started on aspirin to decrease the risk of adverse cardiovascular events. Aspirin impairs prostaglandin synthesis by irreversibly inhibiting cyclooxygenase (COX). Inhibition of COX-1 in platelets prevents synthesis of thromboxane A2, a potent stimulator of platelet aggregation and vasoconstriction. This helps reduce the risk of occlusive thrombus formation and subsequent myocardial infarction.

Some patients are unable to tolerate aspirin due to exacerbation of preexisting respiratory symptoms (eg, rhinitis, asthma) or development of allergic reactions (eg, urticaria, angioedema, anaphylaxis). In these patients, alternate antiplatelet agents should be used for prevention of cardiovascular events. **Clopidogrel** irreversibly **blocks** the $P2Y_{12}$ component of **ADP receptors** on the platelet surface and prevents platelet aggregation. Clopidogrel is as **effective as aspirin** for **prevention of cardiovascular events** and should be used in patients with aspirin allergy.

(Choices A and G) Apixaban is a direct factor Xa inhibitor that prevents platelet activation and fibrin clot formation. Warfarin inhibits an enzyme required for synthesis of active vitamin K, resulting in decreased synthesis of vitamin K-dependent clotting factors (II, VII, IX, and X). Both apixaban and warfarin are typically used for prevention and/or treatment of thromboembolic events, not coronary artery disease.

(Choice B) Cilostazol is a phosphodiesterase inhibitor that is occasionally used in patients with symptomatic peripheral vascular disease (ie, claudication).

(Choice D) Low molecular weight heparins (LMWHs; enoxaparin, dalteparin) are indirect thrombin inhibitors that bind with antithrombin and convert it from a slow to a rapid inactivator of thrombin and factor Xa. They are used in patients with acute coronary syndrome (unstable angina or myocardial infarction) but have no role in the management of patients with stable angina.

(Choice E) Eptifibatide is a platelet glycoprotein IIb/IIIa inhibitor that inhibits the final common pathway of platelet aggregation. Eptifibatide is occasionally used in some patients with acute coronary syndrome, but it is not used for patients with stable CHD.

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(Choice E) Eptifibatide is a platelet glycoprotein IIb/IIIa inhibitor that inhibits the final common pathway of platelet aggregation. Eptifibatide is occasionally used in some patients with acute coronary syndrome, but it is not used for patients with stable CHD.

(Choice F) Naproxen is a nonsteroidal anti-inflammatory drug (NSAID) that is used for pain management in patients with osteoarthritis. Use of both nonselective and selective COX-2 NSAIDs increases the risk of adverse cardiovascular events.

Educational objective:
Clopidogrel irreversibly blocks the $P2Y_{12}$ component of ADP receptors on the platelet surface and prevents platelet aggregation. Clopidogrel is as effective as aspirin in the prevention of cardiovascular events in patients with coronary heart disease.

References

- Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, myocardial

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A 78-year-old woman comes to the emergency department due to a 3-day history of intermittent but progressive substernal chest pain. Medical history includes hypertension and hyperlipidemia, but the patient has had poor adherence with her medication and outpatient follow-up. She has a 30 pack-year smoking history. On admission, ECG reveals normal sinus rhythm with ST-segment elevation and pathologic Q waves in leads I, aVL, and V2 to V5. The patient refuses percutaneous coronary intervention and is treated medically. On the fifth day of hospitalization, she experiences severe dyspnea, sweating, and hypotension. Physical examination shows raised jugular venous pressure, no heart murmurs, and clear lungs. She rapidly loses consciousness and becomes pulseless, and the subsequent resuscitation attempt is ineffective. Which of the following most likely contributed to this patient's death?

- A. Blood accumulation in the pericardial space
- B. Complete rupture of the papillary muscle
- C. Rapid reperfusion of the ischemic myocardium
- D. Rupture of the interventricular septum
- E. True aneurysm of the ventricular wall

Submit

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A 78-year-old woman comes to the emergency department due to a 3-day history of intermittent but progressive substernal chest pain. Medical history includes hypertension and hyperlipidemia, but the patient has had poor adherence with her medication and outpatient follow-up. She has a 30 pack-year smoking history. On admission, ECG reveals normal sinus rhythm with ST-segment elevation and pathologic Q waves in leads I, aVL, and V2 to V5. The patient refuses percutaneous coronary intervention and is treated medically. On the fifth day of hospitalization, she experiences severe dyspnea, sweating, and hypotension. Physical examination shows raised jugular venous pressure, no heart murmurs, and clear lungs. She rapidly loses consciousness and becomes pulseless, and the subsequent resuscitation attempt is ineffective. Which of the following most likely contributed to this patient's death?

- ✓ A. Blood accumulation in the pericardial space (52%)
- B. Complete rupture of the papillary muscle (15%)
- C. Rapid reperfusion of the ischemic myocardium (5%)
- D. Rupture of the interventricular septum (17%)
- E. True aneurysm of the ventricular wall (9%)

Correct

52% Answered correctly

03 secs Time Spent

2023 Version

Explanation

Mechanical complications of acute myocardial infarction

Complication	Time course	Clinical findings
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR)

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Mechanical complications of acute myocardial infarction		
Complication	**Time course**	**Clinical findings**
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR) • Hypotension/cardiogenic shock
Interventricular septum rupture	Acute or within 3-5 days	• Chest pain • New holosystolic murmur • Hypotension/cardiogenic shock • Step up in O_2 level from RA to RV
Free wall rupture**	Within 5 days or up to 2 weeks	• Chest pain • Distant heart sounds • Shock, rapid progression to cardiac arrest
Left ventricular aneurysm**	Up to several months	• Heart failure • Angina, ventricular arrhythmias
*Usually due to right coronary artery occlusion. **Usually due to left anterior descending artery occlusion. **MR** = mitral regurgitation; **RA** = right atrium; **RV** = right ventricle.		

This patient was initially admitted with chest pain and ST-segment elevation in the anterolateral ECG leads, findings consistent with an **ST-elevation myocardial infarction** (MI) of the left ventricle. Several days later, her abrupt-onset dyspnea and hypotension were most likely due to **left ventricular (LV) free wall rupture**, a potential mechanical complication of transmural MI.

LV free wall rupture usually occurs within 5 days but can occur up to 2 weeks following MI. During this time, the infarcted myocardial wall is substantially weakened by coagulative necrosis, leukocyte infiltration, and enzymatic lysis of myocardial connective tissue and may be unable to withstand the shear stress generated by the high-pressure left ventricle. **Delayed reperfusion** (or absent reperfusion as in this patient), increases myocardial

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mechanical complication of transmural MI.

LV free wall rupture usually occurs within 5 days but can occur up to 2 weeks following MI. During this time, the infarcted myocardial wall is substantially weakened by coagulative necrosis, leukocyte infiltration, and enzymatic lysis of myocardial connective tissue and may be unable to withstand the shear stress generated by the high-pressure left ventricle. **Delayed reperfusion** (or absent reperfusion as in this patient), increases myocardial damage, further weakening the LV wall and **increasing the risk of rupture**.

With LV rupture, blood abruptly fills the pericardial space, resulting in hemopericardium and **acute cardiac tamponade** with rapidly **increased intrapericardial pressure**. At onset, patients may experience dyspnea, diaphoresis, and sometimes chest discomfort. Diastolic filling of the relatively low-pressure right-sided heart chambers is abruptly restricted (causing jugular venous distension), and blood flow through the pulmonary circulation to the left side of the heart drops precipitously (the **lungs remain clear**). Cardiac output profoundly decreases, resulting in **hypotension** and obstructive shock that rapidly leads to cardiac arrest.

(Choices B and D) Papillary muscle rupture, causing acute mitral regurgitation, and interventricular septum rupture, causing a ventricular septal defect, can occur acutely or within 5 days of MI. Both defects typically cause a new holosystolic murmur and lead to LV failure with pulmonary edema. Cardiogenic shock with hypotension commonly develops, but lung crackles would be expected on examination.

(Choice C) Ischemic reperfusion injury can occur when blood flow is abruptly restored to infarcted myocardium, and it may rarely lead to cardiac arrest (via precipitation of ventricular fibrillation or profound myocardial contractile dysfunction). However, it is extremely unlikely in this patient who received no reperfusion therapy.

(Choice E) LV aneurysm formation is typically a delayed mechanical complication of MI, occurring after several weeks or months. Patients typically develop gradually progressive heart failure or angina.

Educational objective:
Left ventricular free wall rupture is a mechanical complication that can occur within 5 days or up to 2 weeks following transmural (ST-elevation) myocardial infarction. Patients usually develop acute cardiac tamponade with hypotension and obstructive shock that rapidly leads to cardiac arrest.

Pathophysiology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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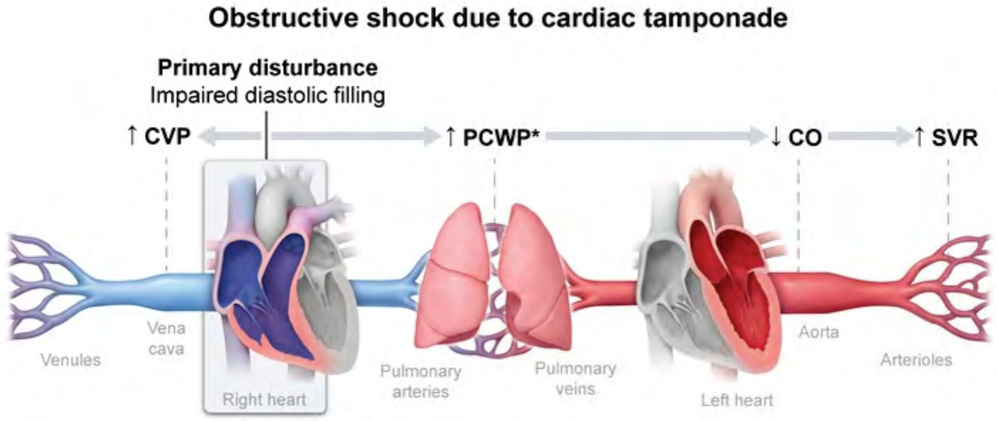

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Pathophysiology Cardiovascular System Myocardial infarction

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mechanical complication of transmural MI.

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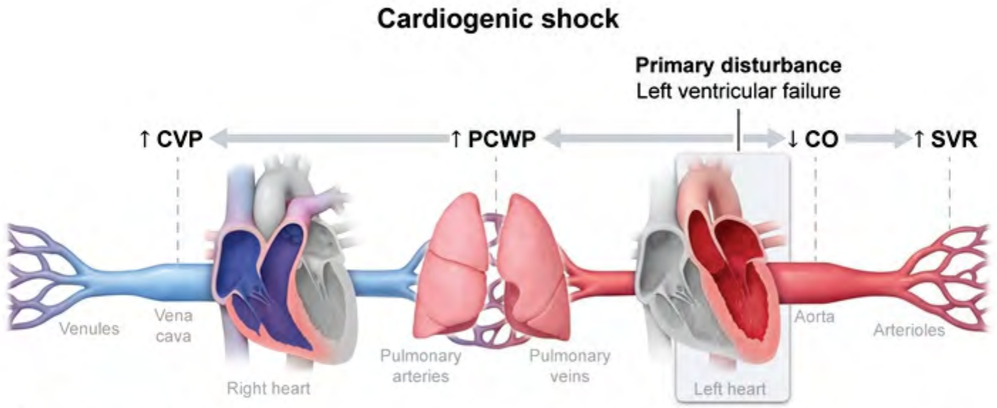

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Pathophysiology | Cardiovascular System | Myocardial infarction

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A previously healthy 15-year-old girl is brought to the emergency department due to respiratory distress and continuous cough after eating at a buffet in a local restaurant. Blood pressure is 80/40 mm Hg, pulse is 130/min, and respirations are 32/min. She is in marked respiratory distress but is able to speak. Diffuse wheezing and erythematous skin rash are present. Which of the following hemodynamic changes are most likely present in this patient?

	Central venous pressure	Pulmonary capillary wedge pressure	Systemic vascular resistance
A.	Decreased	Decreased	Decreased
B.	Decreased	Decreased	Increased
C.	Increased	Decreased	Increased
D.	Increased	Increased	Decreased
E.	Increased	Increased	Increased

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		Central venous pressure	Pulmonary capillary wedge pressure	Systemic vascular resistance	
✓	A.	Decreased	Decreased	Decreased	(65%)
	B.	Decreased	Decreased	Increased	(9%)
	C.	Increased	Decreased	Increased	(6%)
	D.	Increased	Increased	Decreased	(12%)
	E.	Increased	Increased	Increased	(5%)

Correct

65% Answered correctly

03 secs Time Spent

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Explanation

Hemodynamic measurements in shock				
Parameter	Hypovolemic shock	Cardiogenic shock	Obstructive shock	Distributive shock
CVP (right-sided	↓	↑	↑	↓

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Hemodynamic measurements in shock

Parameter	Hypovolemic shock	Cardiogenic shock	Obstructive shock	Distributive shock
CVP (right-sided preload)	↓	↑	↑	↓
PCWP (left-sided preload)	↓	↑	↓*	↓
Cardiac index (LV output)	↓	↓	↓	↑**
SVR (afterload)	↑	↑	↑	↓
SvO_2	↓	↓	↓	↑**

*In tamponade, left-sided preload is decreased, but measured PCWP is paradoxically increased due to external compression by pericardial fluid.

**Cardiac index & SvO_2 are usually decreased in neurogenic shock due to impaired sympathetic reflexes.

CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **SvO_2** = mixed venous oxygen saturation; **SVR** = systemic vascular resistance.

This patient with respiratory distress, wheezing, and erythematous skin rash following food exposure likely has anaphylaxis; her severe hypotension and tachycardia indicate **anaphylactic shock**.

The primary disturbance in distributive shock (eg, septic shock, anaphylactic shock) is profound **peripheral vasodilation** which, in anaphylactic shock, results from widespread IgE-mediated release of inflammatory mediators (eg, histamine). This has the following effects:

- Arteriolar vasodilation causes **decreased systemic vascular resistance** (SVR) and consequent

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CVP = central venous pressure; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; SvO_2 = mixed venous oxygen saturation; **SVR** = systemic vascular resistance.

This patient with respiratory distress, wheezing, and erythematous skin rash following food exposure likely has anaphylaxis; her severe hypotension and tachycardia indicate **anaphylactic shock**.

The primary disturbance in distributive shock (eg, septic shock, anaphylactic shock) is profound **peripheral vasodilation** which, in anaphylactic shock, results from widespread IgE-mediated release of inflammatory mediators (eg, histamine). This has the following effects:

- Arteriolar vasodilation causes **decreased systemic vascular resistance** (SVR) and consequent **hypotension**.
- Venular vasodilation causes **decreased central venous pressure** (CVP) and reduces venous return to the right atrium **(Choice D)**.
- The vasculature becomes leaky, resulting in loss of intravascular volume that further contributes to both hypotension and decreased CVP.

With reduced venous return to the right atrium, there is reduced right ventricular output and reduced blood delivery to the left atrium and left ventricle. This leads to **decreased pulmonary capillary wedge pressure** (PCWP), a reflection of left atrial pressure. Despite the reduced left ventricular preload, cardiac output is commonly normal or increased in early distributive shock due to both the large decrease in SVR and a baroreceptor reflex–mediated increase in heart rate (ie, tachycardia). The increased cardiac output leads to increased mixed venous oxygen saturation due to rapid blood transit through the peripheral capillaries and incomplete oxygen uptake by the tissues.

(Choice B) Hypovolemic shock involves massive intravascular volume depletion, leading to decreased CVP and PCWP. SVR is reflexively increased in response to reduced cardiac output and hypotension.

(Choice C) Obstructive shock most commonly involves an obstruction to blood flow between the right atrium and the pulmonary capillaries (eg, pulmonary embolism). CVP is increased, PCWP is decreased, and there is a reflexive increase in SVR.

(Choice E) Cardiogenic shock results from left ventricular failure. PCWP and CVP are increased due to backward

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- Venular vasodilation causes **decreased central venous pressure** (CVP) and reduces venous return to the right atrium **(Choice D)**.
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With reduced venous return to the right atrium, there is reduced right ventricular output and reduced blood delivery to the left atrium and left ventricle. This leads to **decreased pulmonary capillary wedge pressure** (PCWP), a reflection of left atrial pressure. Despite the reduced left ventricular preload, cardiac output is commonly normal or increased in early distributive shock due to both the large decrease in SVR and a baroreceptor reflex–mediated increase in heart rate (ie, tachycardia). The increased cardiac output leads to increased mixed venous oxygen saturation due to rapid blood transit through the peripheral capillaries and incomplete oxygen uptake by the tissues.

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(Choice C) Obstructive shock most commonly involves an obstruction to blood flow between the right atrium and the pulmonary capillaries (eg, pulmonary embolism). CVP is increased, PCWP is decreased, and there is a reflexive increase in SVR.

(Choice E) Cardiogenic shock results from left ventricular failure. PCWP and CVP are increased due to backward transmission of increased pressure, and there is a reflexive increase in SVR.

Educational objective:
Anaphylaxis can cause distributive shock due to widespread release of inflammatory mediators (eg, histamine) that leads to massive peripheral vasodilation. Arteriolar vasodilation decreases systemic vascular resistance to cause hypotension, and venular vasodilation decreases central venous pressure and pulmonary capillary wedge pressure. Cardiac output is often increased in early distributive shock due to reduced SVR and a baroreceptor reflex–mediated increase in heart rate.

References

- Distributive shock.

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*In tamponade, left-sided preload is decreased, but measured PCWP is paradoxically increased due to external compression by pericardial fluid.

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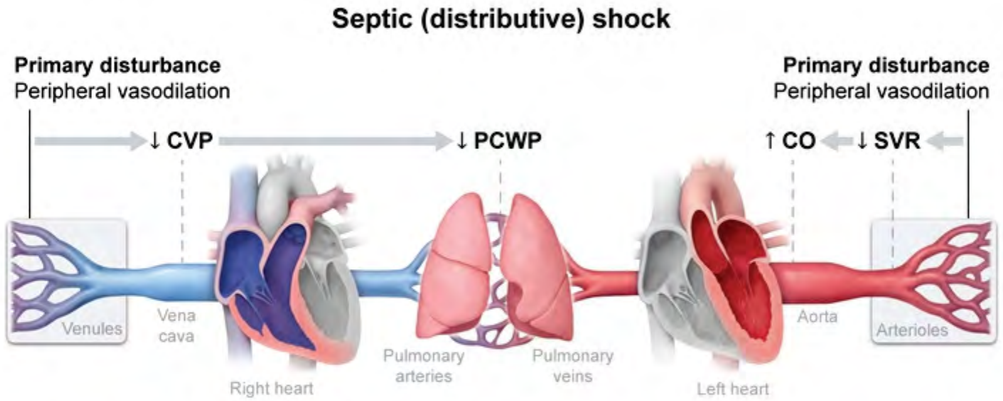

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venous oxygen saturation; **SVR** = systemic vascular resistance.

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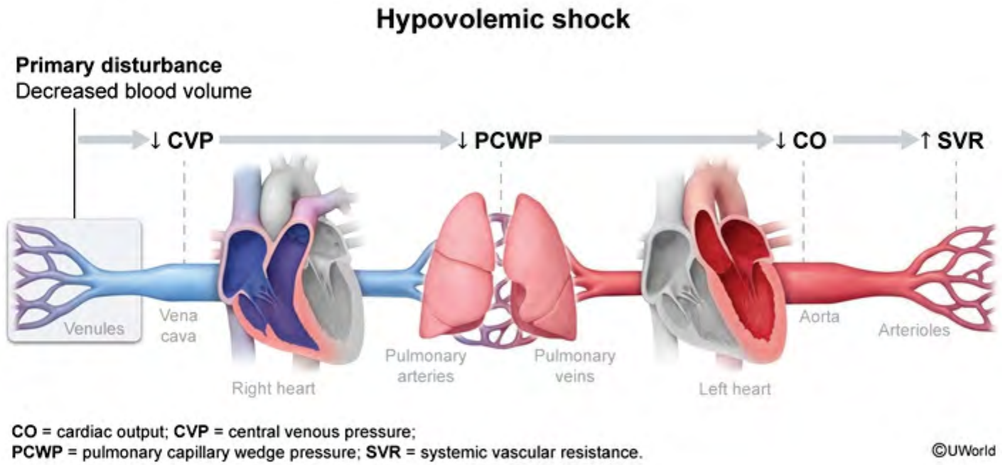

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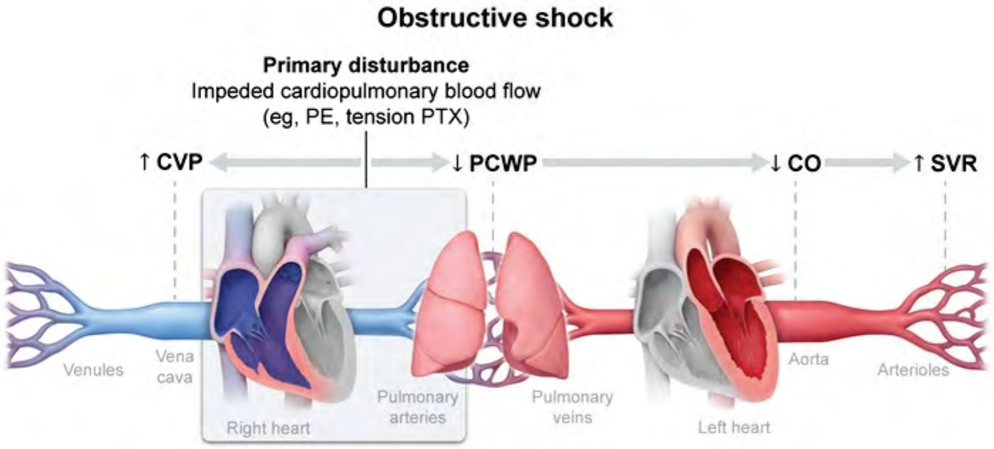

CO = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **PE** = pulmonary embolism; **PTX** = pneumothorax; **SVR** = systemic vascular resistance.

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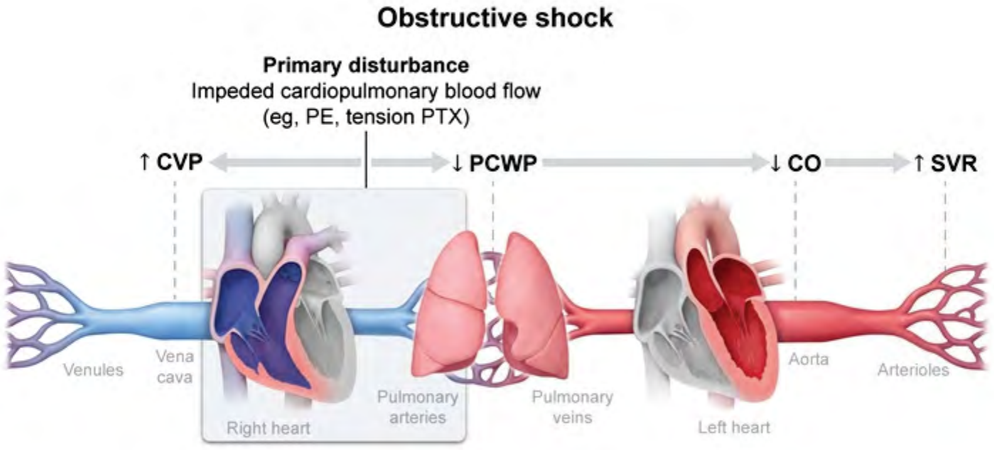

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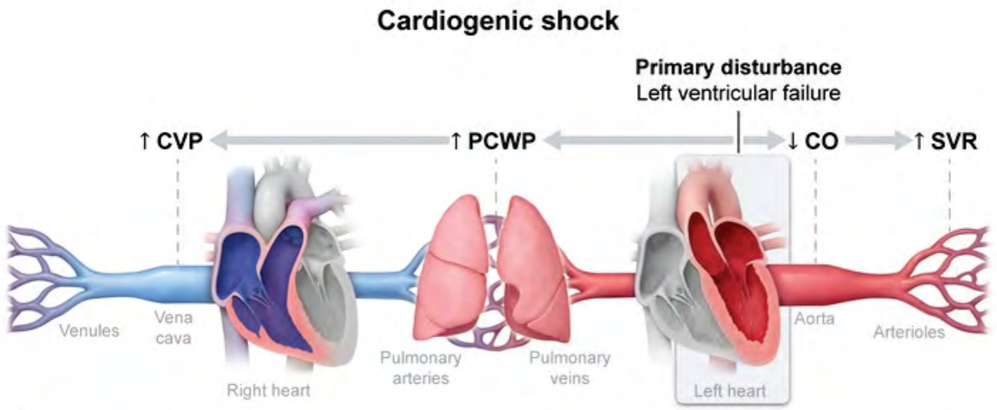

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A 54-year-old man comes to the physician for evaluation of exertional chest pain. He first noticed the pain a couple months ago when he was shoveling snow off his driveway and has since experienced several episodes while doing other strenuous tasks. He describes the pain as a heavy, crushing sensation and says it is relieved with rest. The patient undergoes coronary angiography and is found to have severe stenosis of the right coronary artery and left anterior descending and circumflex arteries. He is referred to a surgeon for coronary artery bypass grafting. During the procedure, a portion of his great saphenous vein is removed and grafted to one of the diseased coronary arteries to bypass its atherosclerotic narrowing. The vein used as a graft during this patient's procedure can be accessed at which of the following sites?

- A. At the midline of the popliteal fossa
- B. Just inferior to the anterior superior iliac spine
- C. Just inferolateral to the pubic tubercle
- D. Just superior to the inguinal ligament
- E. Over the lateral aspect of the foot

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- A. At the midline of the popliteal fossa (31%)
- ✗ B. Just inferior to the anterior superior iliac spine (7%)
- ✓ C. Just inferolateral to the pubic tubercle (39%)
- D. Just superior to the inguinal ligament (3%)
- E. Over the lateral aspect of the foot (17%)

Incorrect
Correct answer
C

39%
Answered correctly

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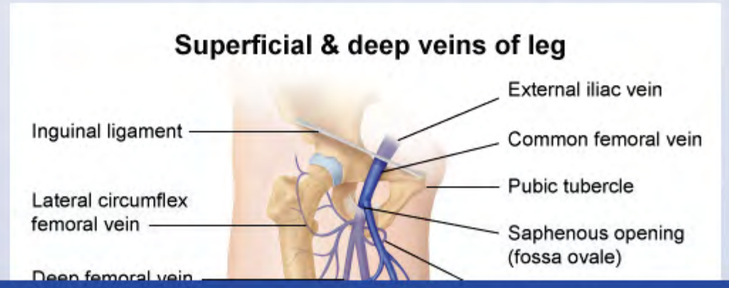
Explanation



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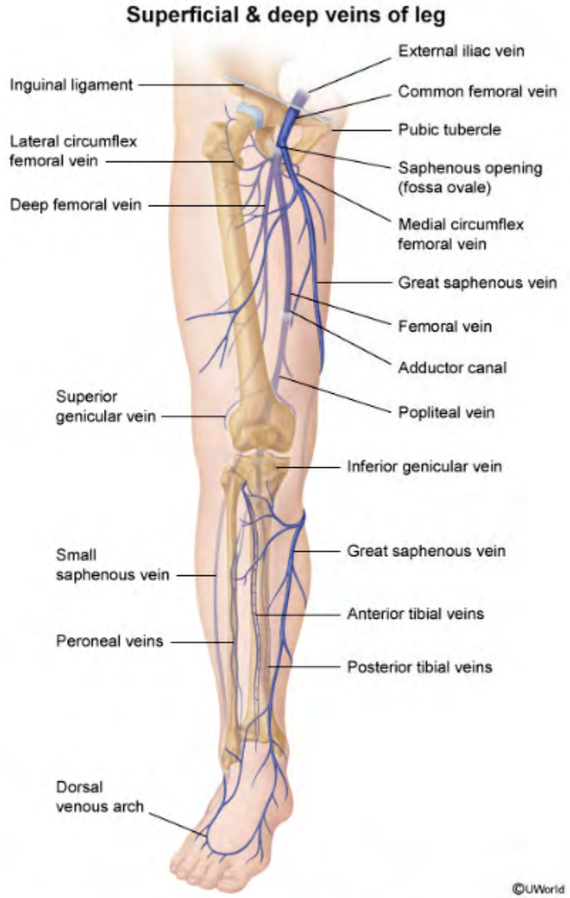

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femoral vein
Saphenous opening (fossa ovale)
Deep femoral vein

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When the left anterior descending artery (LAD) alone is occluded by an atherosclerotic plaque, the left internal mammary (thoracic) artery is the preferred vessel for bypass grafting due to superior patency rates. However, when multiple coronary arteries or vessels other than the LAD require revascularization, great saphenous vein grafts are routinely used.

The **great saphenous vein** is located superficially in the leg and is the longest vein in the body. It courses superiorly from the medial foot, anterior to the medial malleolus, and up the medial aspect of the leg and thigh. In the proximal anterior thigh, 3-4 centimeters inferolateral to the pubic tubercle, the great saphenous vein dives deep through the cribriform fascia of the saphenous opening to join the femoral vein. Surgeons access the great saphenous vein in the medial leg or, less commonly, near its point of termination in the femoral triangle of the upper thigh. The femoral triangle is bordered by the inguinal ligament superiorly, sartorius muscle laterally, and adductor longus muscle medially.

(Choice A) The popliteal artery and vein course centrally through the popliteal fossa together with the tibial nerve. Common medical problems that occur in the popliteal fossa include popliteal artery aneurysms, which account for the majority of peripheral artery aneurysms, and synovial (Baker's) cysts, which are commonly associated with arthritis.

(Choice B) No major vessels are located immediately inferior to the anterior superior iliac spine (ASIS). The ASIS serves as the superior attachment of the inguinal ligament, and a penetrating injury to the region below the ASIS could damage the lateral cutaneous nerve of the thigh.

(Choice D) The deep circumflex iliac vessels course parallel and just superior to the inguinal ligament. The superficial and inferior epigastric veins course above the midportion of the inguinal ligament.

(Choice E) The small saphenous vein can be found at the lateral aspect of the foot. This vein courses posteriorly to drain into the popliteal vein.

Educational objective:
The great saphenous vein is a superficial vein of the leg that originates on the medial side of the foot, courses anterior to the medial malleolus, and then travels up the medial aspect of the leg and thigh. It drains into the

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Educational objective:
The great saphenous vein is a superficial vein of the leg that originates on the medial side of the foot, courses anterior to the medial malleolus, and then travels up the medial aspect of the leg and thigh. It drains into the femoral vein within the region of the femoral triangle, a few centimeters inferolateral to the pubic tubercle.

Anatomy Cardiovascular System CABG

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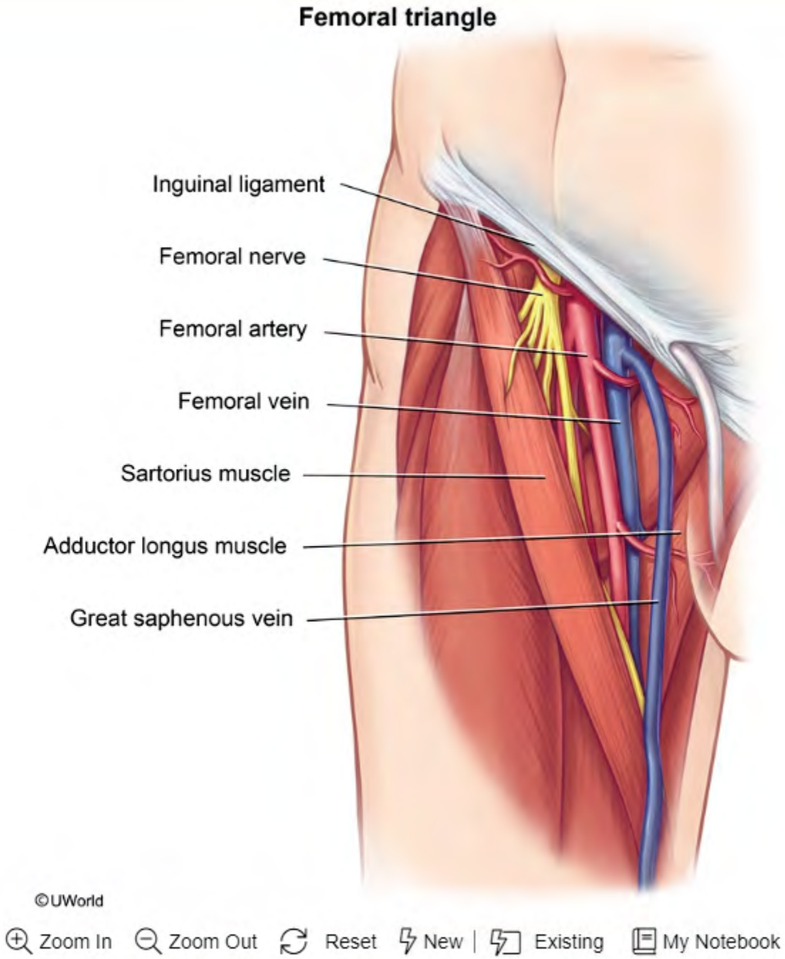

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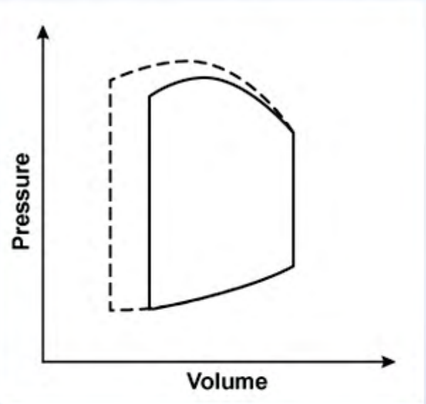
A 45-year-old man comes to the emergency department due to a 2-day history of dyspnea, orthopnea, and ankle swelling. This morning, he also had nausea, vomiting, and a headache. Blood pressure is elevated, and physical examination shows pulmonary crackles and bilateral pitting edema up to the knees. A sodium nitroprusside infusion is begun. Which of the following left ventricular pressure/volume relationships best represents the effects of this therapy?

*Solid line = before nitroprusside; dashed line = during nitroprusside infusion.

A.



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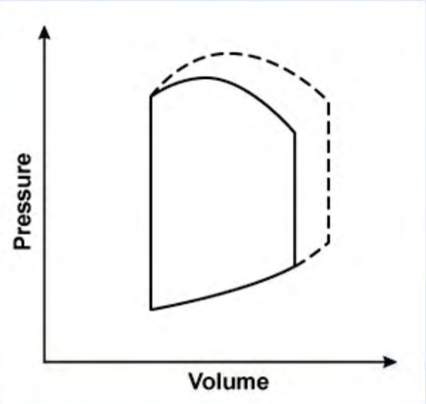

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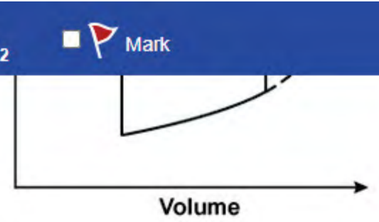

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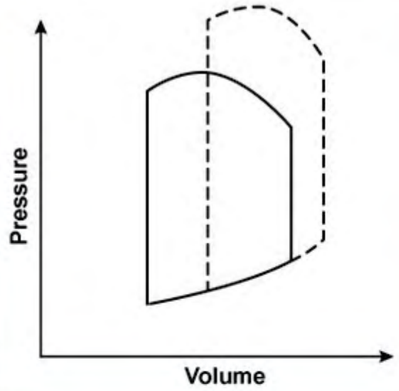

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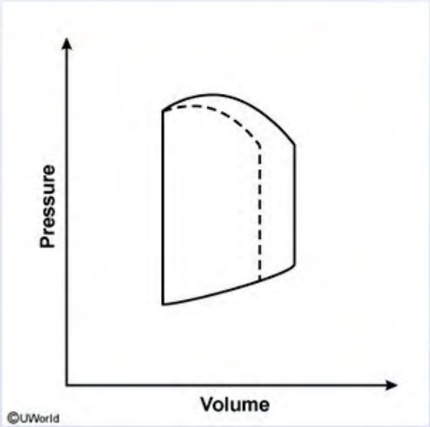

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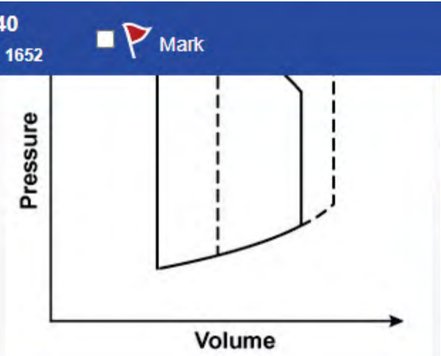

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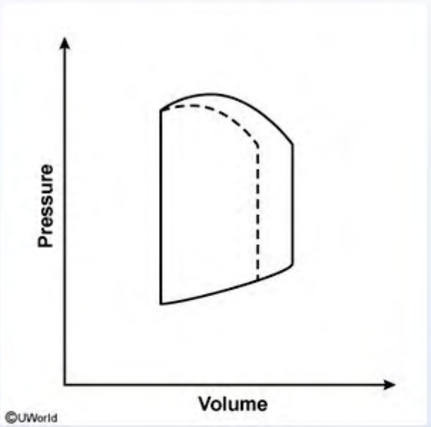

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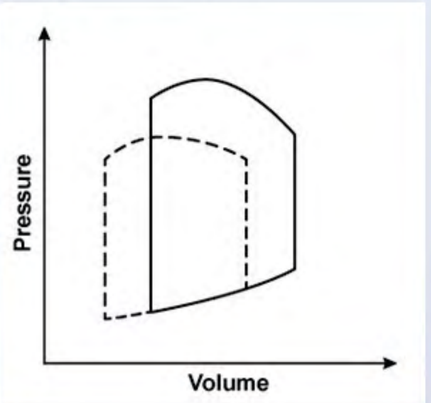

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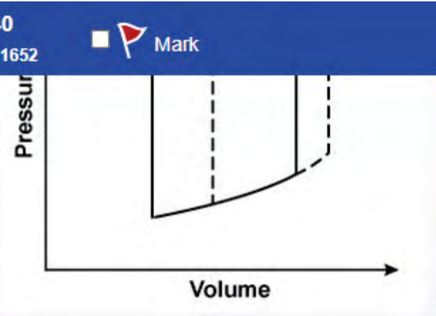
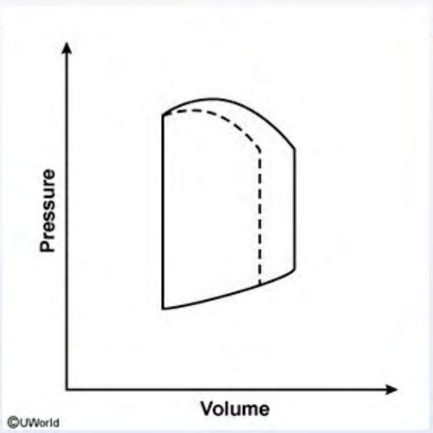
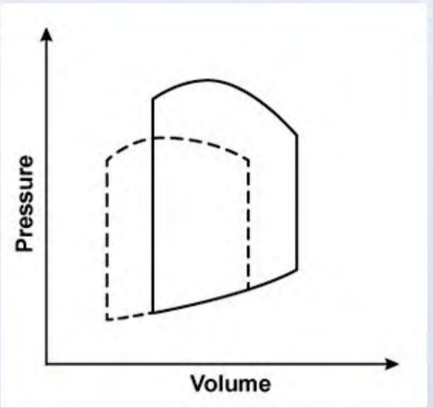


✕ D. (24%)



✓ E. (52%)



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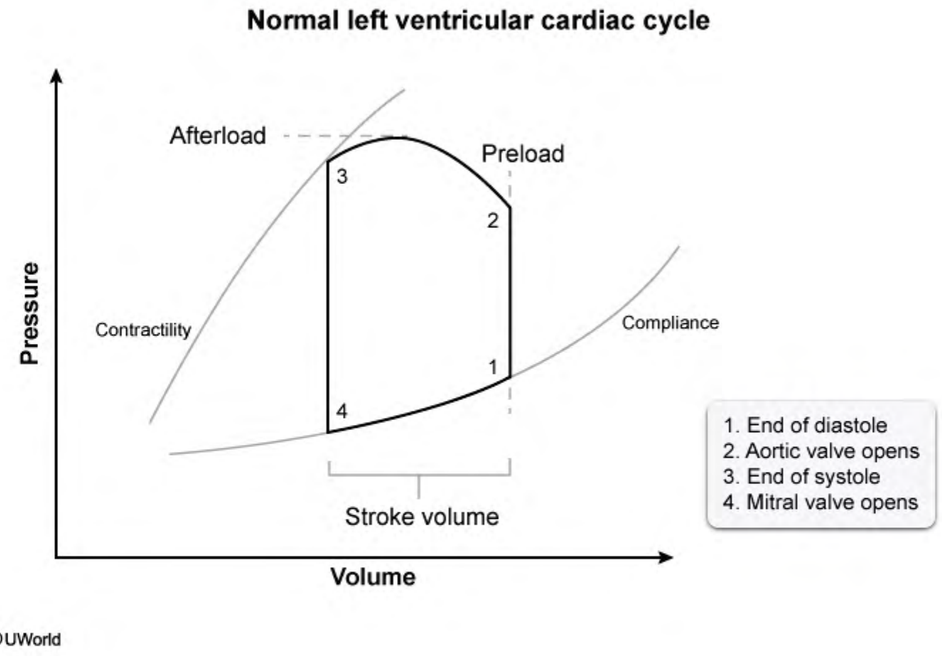

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This patient most likely has **acute heart failure** due to **systemic hypertension**, which can be treated with an infusion of **sodium nitroprusside**. Sodium nitroprusside is a short-acting agent that causes **balanced vasodilation** of the veins and arteries; as such, it **decreases** left ventricular (LV) **preload and afterload**, allowing for maintenance of cardiac output (**unchanged stroke volume**) at a lower LV pressure (decreased LV work). LV

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Of the choices given, only graph E shows a reduction in both LV end-diastolic volume (preload) and LV systolic pressure (afterload) without a decrease in stroke volume.

(Choice A) This graph depicts unchanged preload with increased stroke volume due to an increase in LV contractility (point 3 is moved upward and to the left). The slight increase in afterload occurs secondary to the increase in contractility. Such changes are expected with the administration of a positive inotrope with minimal vasodilatory/vasoconstrictive effect (eg, digoxin).

(Choice B) This graph depicts increased preload with an increase in stroke volume and no change in contractility. The increase in afterload is secondary to the increase in stroke volume (higher stroke volume increases systolic pressure). Such changes are consistent with the Frank-Starling response to increased preload.

(Choice C) This graph depicts an increase in preload and afterload with a reduction in stroke volume and no change in contractility. Such changes are detrimental in heart failure and would be expected with a vasoconstrictor affecting both the veins and arteries but with no effect on the myocardium (eg, the alpha-1 agonist phenylephrine).

(Choice D) This graph depicts a decrease in preload and stroke volume with a minimal reduction in afterload and no change in contractility. These changes are expected with predominantly venous dilators such as nitrates (eg, nitroglycerin, isosorbide mononitrate, isosorbide dinitrate). Sodium nitroprusside differs from nitrates in that it generates nitric oxide in the bloodstream rather than in the venous smooth muscle, creating a balanced vasodilatory effect on both the arteries and veins.

Educational objective:
Sodium nitroprusside is a short-acting agent that causes balanced vasodilation of the veins and arteries to decrease both left ventricular (LV) preload and afterload. The balanced vasodilation allows for maintenance of

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vasodilation of the veins and arteries; as such, it **decreases** left ventricular (LV) **preload and afterload**, allowing for maintenance of cardiac output (**unchanged stroke volume**) at a lower LV pressure (decreased LV work). LV contractility is essentially unchanged.

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(Choice D) This graph depicts a decrease in preload and stroke volume with a minimal reduction in afterload and no change in contractility. These changes are expected with predominantly venous dilators such as nitrates (eg, nitroglycerin, isosorbide mononitrate, isosorbide dinitrate). Sodium nitroprusside differs from nitrates in that it generates nitric oxide in the bloodstream rather than in the venous smooth muscle, creating a balanced vasodilatory effect on both the arteries and veins.

Educational objective:
Sodium nitroprusside is a short-acting agent that causes balanced vasodilation of the veins and arteries to decrease both left ventricular (LV) preload and afterload. The balanced vasodilation allows for maintenance of stroke volume and cardiac output at a lower LV pressure (lower cardiac work).

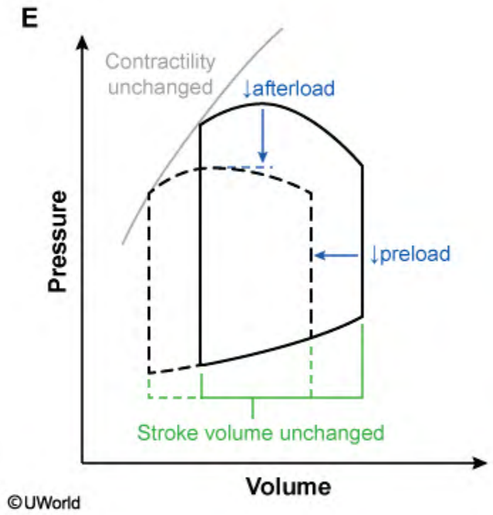
Physiology	Cardiovascular System	Diastolic dysfunction
Subject	System	Topic

https://t.me/USMLEWorldStep1

Stroke volume

Volume

Exhibit Display



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Stroke volume

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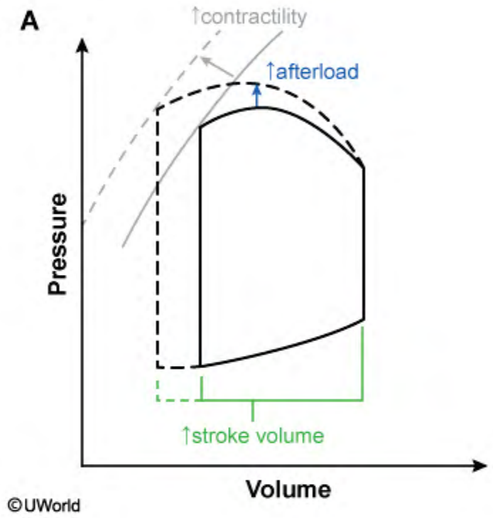

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generates nitric oxide in the bloodstream rather than in the venous smooth muscle, creating a balanced vasodilatory effect on both the arteries and veins.

Educational objective:

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This patient most likely has **acute heart failure** due to **systemic hypertension**, which can be treated with an infusio
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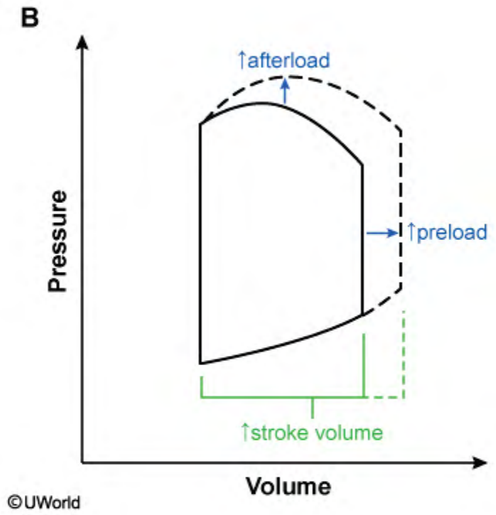

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for maintenance of cardiac output (**unchanged stroke volume**) at a lower LV pressure (decreased LV work). LV contractility is essentially unchanged.

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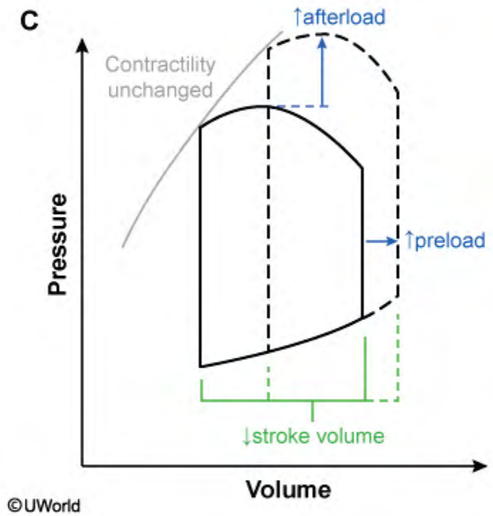

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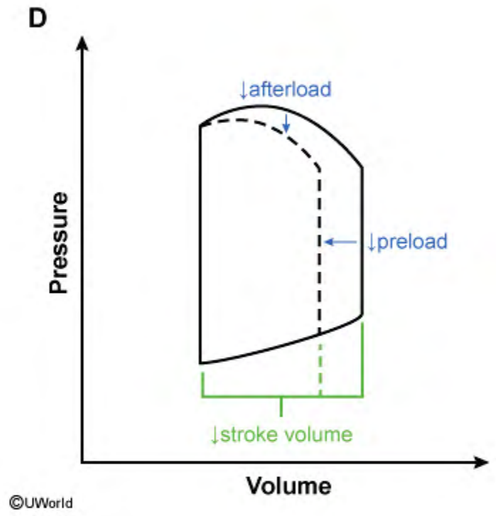

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Physiology
Subject

Cardiovascular System
System

Diastolic dysfunction
Topic

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A 48-year-old man comes to the office for a follow-up visit. He was diagnosed with hypercholesterolemia 6 months ago and has been strictly following dietary and lifestyle modifications as advised. The patient has no other medical problems. He does not use tobacco, alcohol, or illicit drugs. His father has diabetes mellitus and coronary artery disease. The patient's blood pressure is 126/70 mm Hg and BMI is 33.1 kg/m^2. Physical examination is normal. Laboratory studies show a current LDL level of 190 mg/dL. Which of the following should be obtained before starting statin therapy in this patient?

- A. Apolipoprotein-B level
- B. Complete blood count
- C. Lipoprotein lipase activity assay
- D. Liver transaminase levels
- E. Serum cortisol level
- F. Serum creatinine

Submit

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A 48-year-old man comes to the office for a follow-up visit. He was diagnosed with hypercholesterolemia 6 months ago and has been strictly following dietary and lifestyle modifications as advised. The patient has no other medical problems. He does not use tobacco, alcohol, or illicit drugs. His father has diabetes mellitus and coronary artery disease. The patient's blood pressure is 126/70 mm Hg and BMI is 33.1 kg/m^2. Physical examination is normal. Laboratory studies show a current LDL level of 190 mg/dL. Which of the following should be obtained before starting statin therapy in this patient?

- A. Apolipoprotein-B level (3%)
- ✗ B. Complete blood count (1%)
- C. Lipoprotein lipase activity assay (4%)
- ✓ D. Liver transaminase levels (69%)
- E. Serum cortisol level (0%)
- F. Serum creatinine (20%)

Incorrect
Correct answer
D

69%
Answered correctly

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Explanation

Antihyperlipidemic medications

Drug	Mechanism	Major lipid effects	Adverse effects
Statins	• Inhibit HMG-CoA reductase	• ↓↓ LDL • ↓ Triglycerides	• Hepatotoxicity • Muscle toxicity
	• ↓ Intestinal cholesterol		• ↑ Hepatotoxicity if given with

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Antihyperlipidemic medications			
Drug	**Mechanism**	**Major lipid effects**	**Adverse effects**
Statins	• Inhibit HMG-CoA reductase	• ↓↓ LDL • ↓ Triglycerides	• Hepatotoxicity • Muscle toxicity
Ezetimibe	• ↓ Intestinal cholesterol absorption	• ↓ LDL	• ↑ Hepatotoxicity if given with statins
Bile acid sequestrants	• Prevent reabsorption of bile acids in the intestine	• ↓ LDL	• Nausea, bloating, cramping • Impaired drug & fat-soluble vitamin absorption
Niacin	• ↓ Fatty acid release • ↓ VLDL synthesis • ↓ HDL clearance	• ↓ LDL • ↑↑ HDL	• Flushing & pruritus • Hepatotoxicity • Hyperuricemia/gout
Fibrates	• Activate PPAR-α • ↓ VLDL synthesis	• ↓↓ Triglycerides • ↑ HDL	• Muscle toxicity • Gallstones
Fish oil/ omega-3 fatty acids	• ↓ VLDL synthesis • ↓ Apolipoprotein B synthesis	• ↓ Triglycerides • ↑ HDL	• Fishy taste
PPAR-α = peroxisome proliferator-activated receptor-alpha.			

This patient has a severe elevation in his **LDL** level. This degree of hyperlipidemia, especially with a family history of coronary artery disease, suggests familial hypercholesterolemia and confers a very high risk for cardiovascular events. **HMG-CoA reductase inhibitors** (statins) are the first-line therapy for most patients with hypercholesterolemia and have been proven to reduce the risk of cardiac events. The statins are structural

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PPAR-α = peroxisome proliferator-activated receptor-alpha.

This patient has a severe elevation in his **LDL** level. This degree of hyperlipidemia, especially with a family history of coronary artery disease, suggests familial hypercholesterolemia and confers a very high risk for cardiovascular events. **HMG-CoA reductase inhibitors** (statins) are the first-line therapy for most patients with hypercholesterolemia and have been proven to reduce the risk of cardiac events. The statins are structural analogs of HMG-CoA and competitively inhibit HMG-CoA reductase, the enzyme responsible for the rate-limiting step in cholesterol synthesis.

Serious side effects of statins include **myopathy** and **hepatitis**. Although mild elevations of liver enzymes are common, significant liver injury (aspartate transaminase/alanine transaminase levels more than 3 times the upper limit of normal) is seen in less than 1% of patients and is usually reversible if the medication is discontinued. **Liver function tests** are recommended before starting statin therapy; otherwise, routine monitoring is not necessary unless symptoms of hepatic injury develop (eg, fatigue, malaise, anorexia).

(Choice A) Apolipoprotein-B (Apo-B) is a structural protein found in atherogenic lipoprotein particles. Each lipoprotein particle contains only a single Apo-B molecule, so measurement of Apo-B levels can quantify the LDL particle count. Patients with increased LDL particle counts are at greater risk for cardiovascular events. However, measurement is of limited clinical utility and would not change management in this case.

(Choice B) Statin medications do not cause any significant hematologic toxicity.

(Choice C) Lipoprotein lipase deficiency can cause familial hypertriglyceridemia (heterozygous deficiency) and chylomicronemia (homozygous deficiency) syndromes. These conditions present with elevated serum triglyceride levels but do not cause elevation in LDL levels.

(Choice E) Because cholesterol is a precursor in the synthesis of adrenocortical and gonadal steroid hormones, statin medications can, in theory, cause hypogonadism or hypoadrenalism. Clinically, however, there are no significant alterations in serum cortisol or gonadal steroid levels unless patients are also on other adrenal enzyme inhibitors (eg, ketoconazole, aminoglutethimide).

(Choice F) Statins have no significant renal side effects. In rare instances, they can cause rhabdomyolysis, which can precipitate acute renal failure; however, this occurs less commonly than hepatic dysfunction and would be associated with myalgias and myoglobinuria.

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Serious side effects of statins include **myopathy** and **hepatitis**. Although mild elevations of liver enzymes are common, significant liver injury (aspartate transaminase/alanine transaminase levels more than 3 times the upper limit of normal) is seen in less than 1% of patients and is usually reversible if the medication is discontinued. **Liver function tests** are recommended before starting statin therapy; otherwise, routine monitoring is not necessary unless symptoms of hepatic injury develop (eg, fatigue, malaise, anorexia).

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Educational objective:
Common side effects of HMG-CoA reductase inhibitors (statins) include muscle and liver toxicity. Hepatic transaminases should be checked prior to initiating therapy and repeated if symptoms of hepatic injury occur.

References

- Statins and the liver.

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A 77-year-old woman is brought to the emergency department with severe lightheadedness and near-syncope that began earlier in the day. Over the past several months, she has experienced occasional episodes of lightheadedness and feeling unsteady, which she attributed to "old age." The patient has not had chest pain, shortness of breath, or lower extremity swelling. She was diagnosed with hypertension 10 years ago and has been taking losartan. The patient does not take any other medications. She lives alone and is independent in her daily activities. An ECG obtained in the emergency department is shown in the exhibit. Cell degeneration in which of the following locations is most likely responsible for this patient's current condition?

- A. Interatrial septum
- B. Interventricular septum
- C. Left atrial wall
- D. Left ventricular free wall
- E. Right atrial wall

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A 77-year-old woman is brought to the emergency department with severe lightheadedness and near-syncope that
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- A. Interatrial septum (24%)
- ✗ B. Interventricular septum (15%)
- C. Left atrial wall (5%)
- D. Left ventricular free wall (3%)
- ✓ E. Right atrial wall (51%)

Incorrect
Correct answer
E

51%
Answered correctly

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Time Spent

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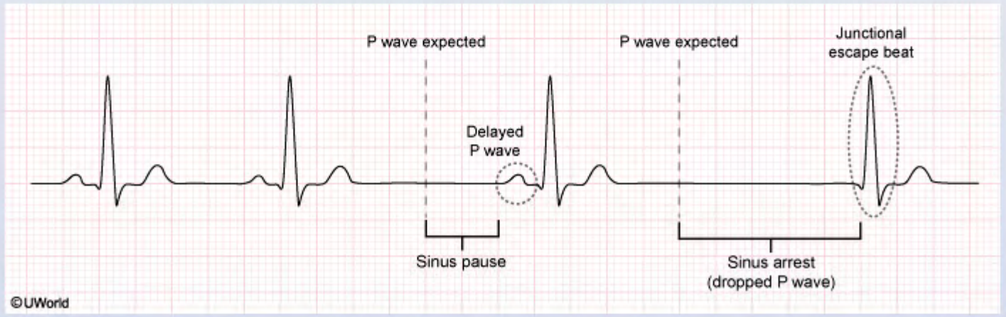
Explanation



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B. Interventricular septum (15%)

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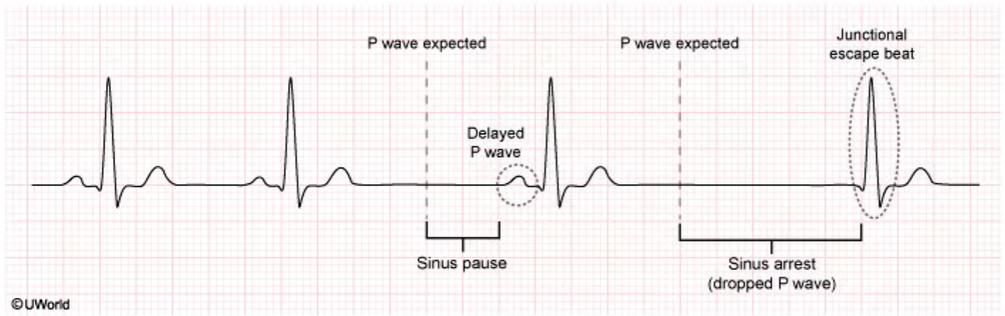

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own pacemaker if it does not receive a timely signal from the sinoatrial node, sinus arrest will lead to a **junctional escape beat** (narrow QRS complex preceded by a long pause and no P wave). Sick sinus syndrome can also

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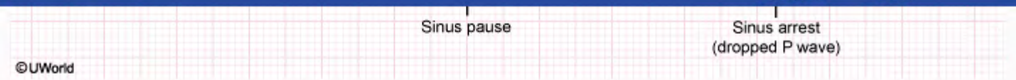

This elderly patient with recent lightheadedness and near-syncope has an ECG showing **bradycardia** with dropped P waves; these findings are consistent with **sick sinus syndrome**. Sick sinus syndrome most commonly results from **age-related degeneration** of the **sinoatrial node**, which is located on the right atrial wall and is responsible for initiating normal cardiac conduction. Impaired signaling from the sinoatrial node can markedly slow the rate of ventricular contraction, leading to **reduced cardiac output** and symptoms of dyspnea, fatigue, lightheadedness, presyncope, and syncope.

Typical ECG findings include bradycardia, **sinus pauses** (delayed P waves), and **sinus arrest** (prolonged delay of a P wave such that the P wave is dropped). Because the atrioventricular node triggers conduction based on its own pacemaker if it does not receive a timely signal from the sinoatrial node, sinus arrest will lead to a **junctional escape beat** (narrow QRS complex preceded by a long pause and no P wave). Sick sinus syndrome can also cause brief periods of atrial tachycardia (eg, atrial fibrillation) alternating with bradycardia.

(Choice A) The inferior portion of the interatrial septum contains the His bundle. Conduction abnormalities in this region can manifest as varying degrees of atrioventricular block with prolongation of the PR interval (first degree), dropped QRS complexes (second degree), or dissociation of P waves and QRS complexes (third degree).

(Choice B) Conduction through the interventricular septum mostly involves the left and right bundle branches. Conduction abnormalities in this region can lead to bundle branch blocks; the QRS complex is widened in bundle branch block due to impaired synchronization of ventricular depolarization.

(Choice C) After initiation in the sinoatrial node, conduction propagates through the cardiomyocytes of the left atrial wall to stimulate left atrial contraction. Abnormal electrical foci can develop in the nearby pulmonary veins, which is a common trigger for atrial fibrillation.

(Choice D) Conduction abnormalities may originate in ischemic areas of the left ventricular free wall, leading to ventricular tachycardia.

Educational objective:
Sick sinus syndrome results from degeneration (usually age-related) of the sinoatrial node, leading to impaired conduction and reduced cardiac output with symptoms of dyspnea, fatigue, lightheadedness, presyncope, and

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Educational objective:
Sick sinus syndrome results from degeneration (usually age-related) of the sinoatrial node, leading to impaired conduction and reduced cardiac output with symptoms of dyspnea, fatigue, lightheadedness, presyncope, and syncope. ECG typically demonstrates bradycardia with sinus pauses (delayed P waves), sinus arrest (dropped P waves), and junctional escape beats.

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Delayed
P wave

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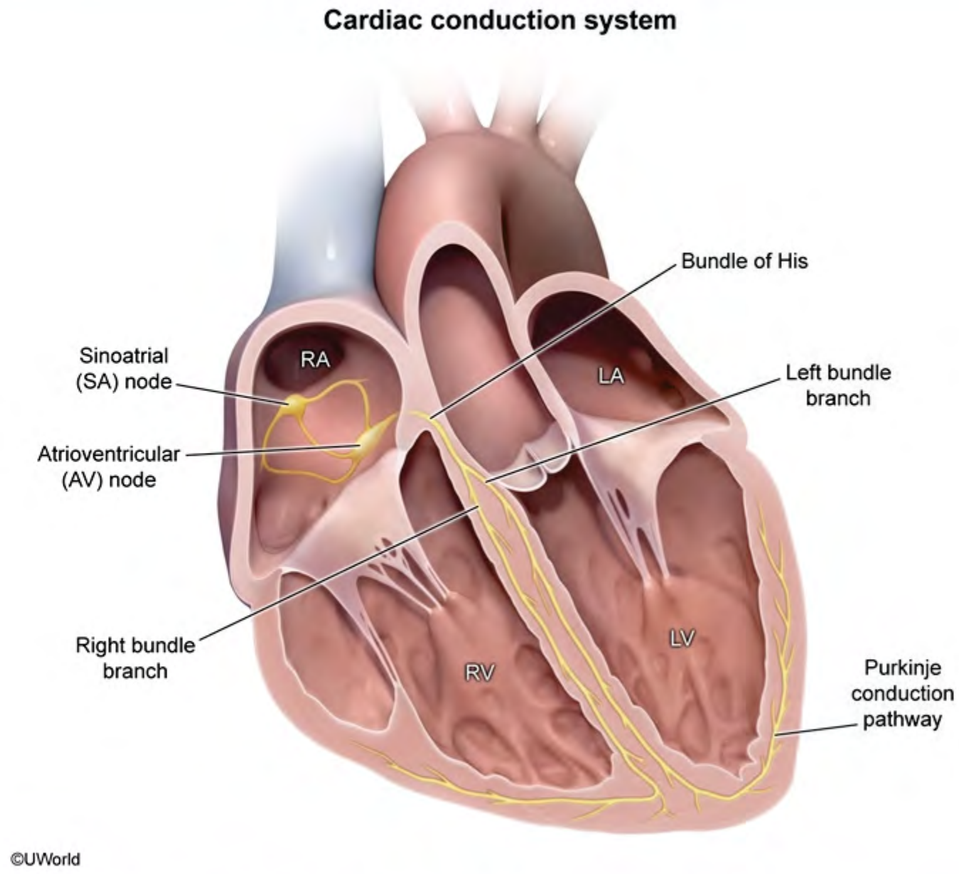

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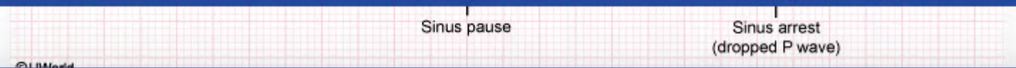

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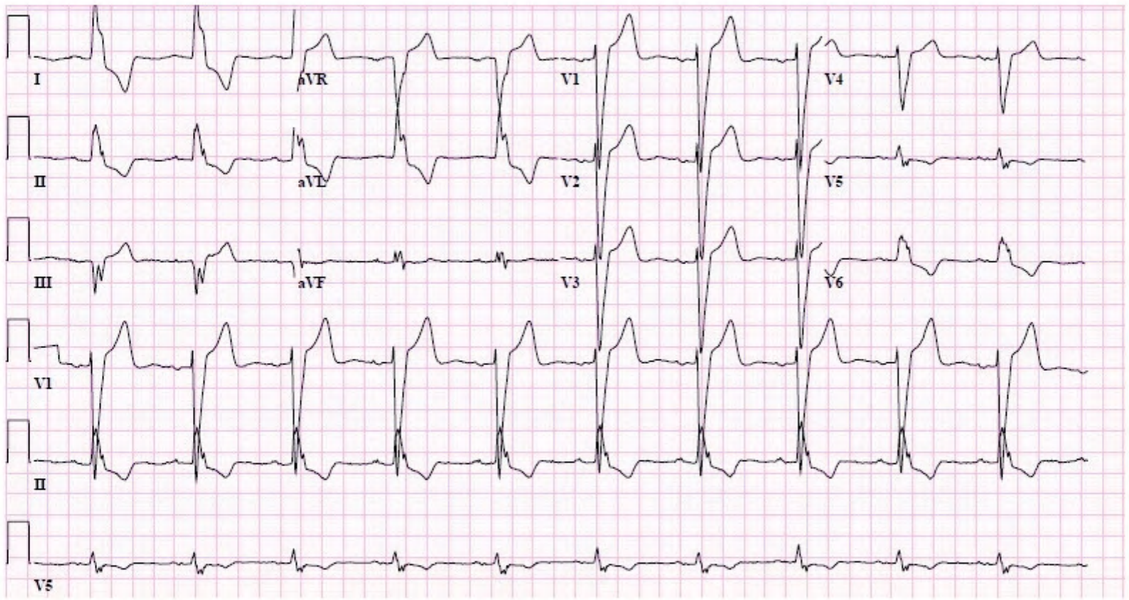

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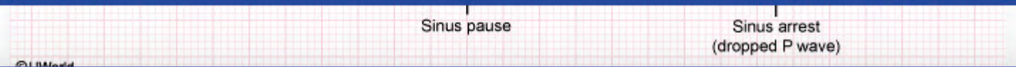

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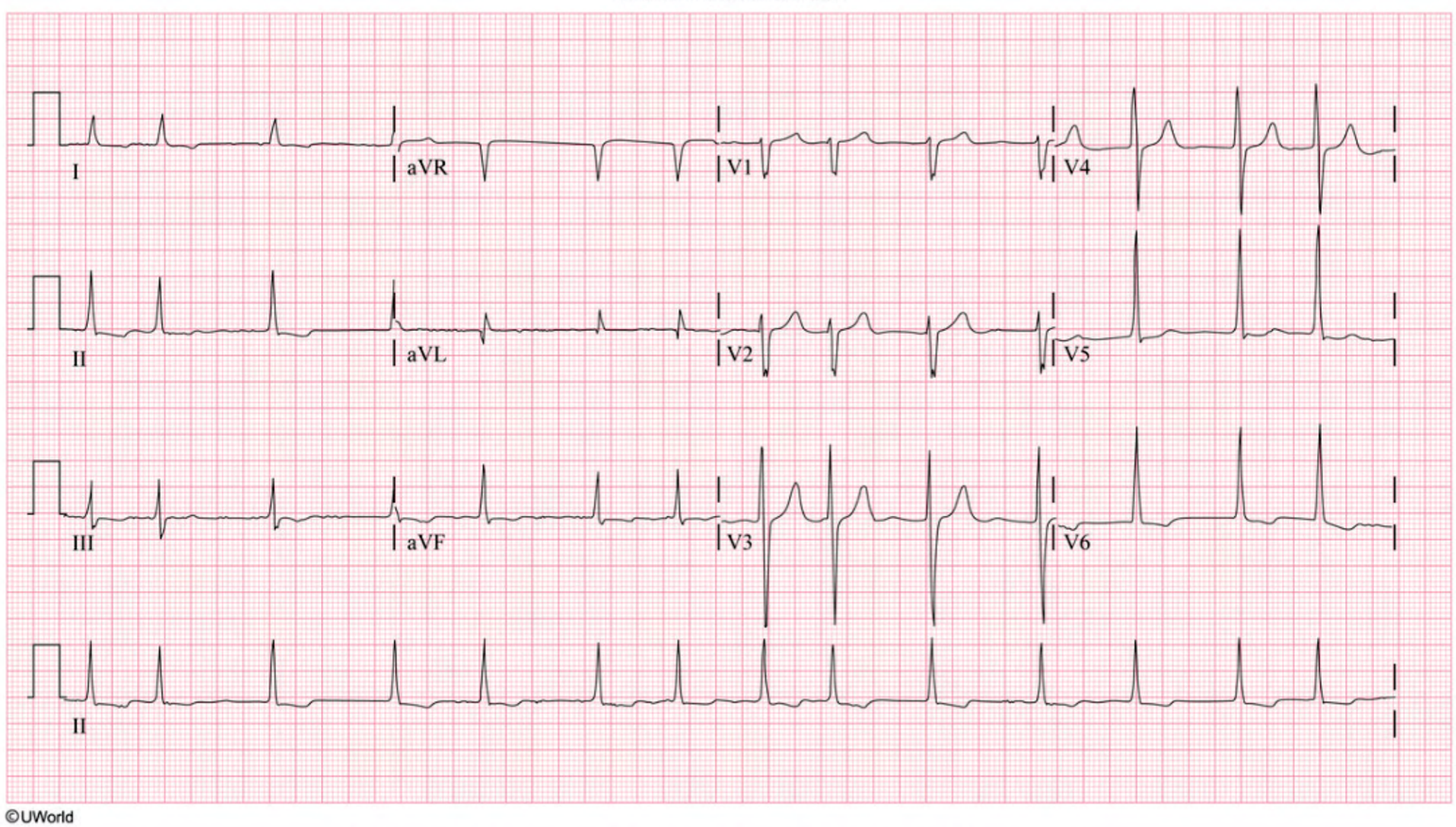

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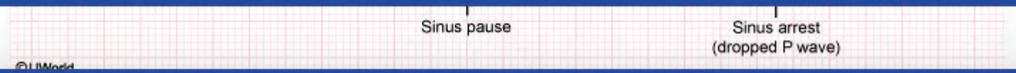

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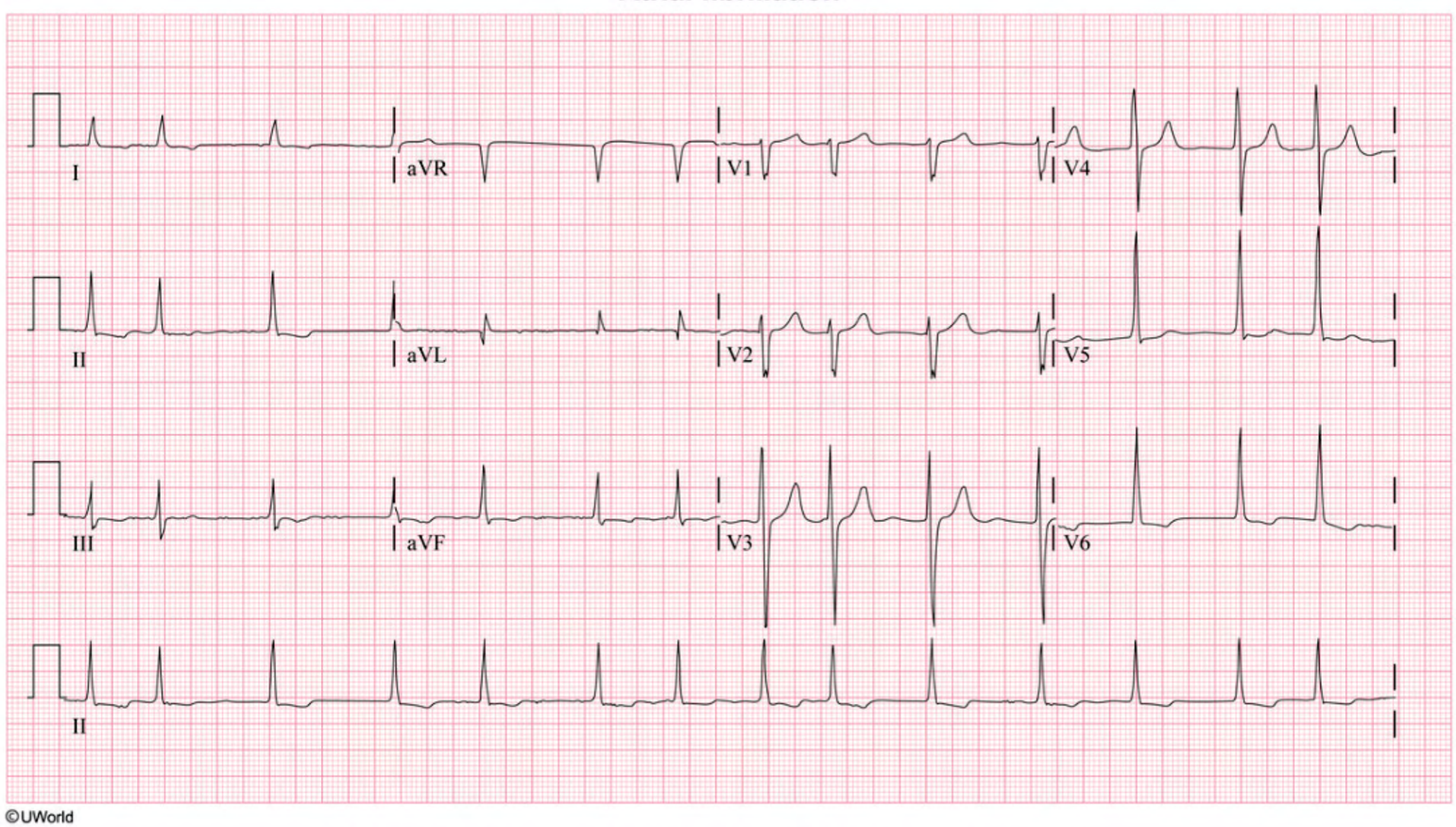

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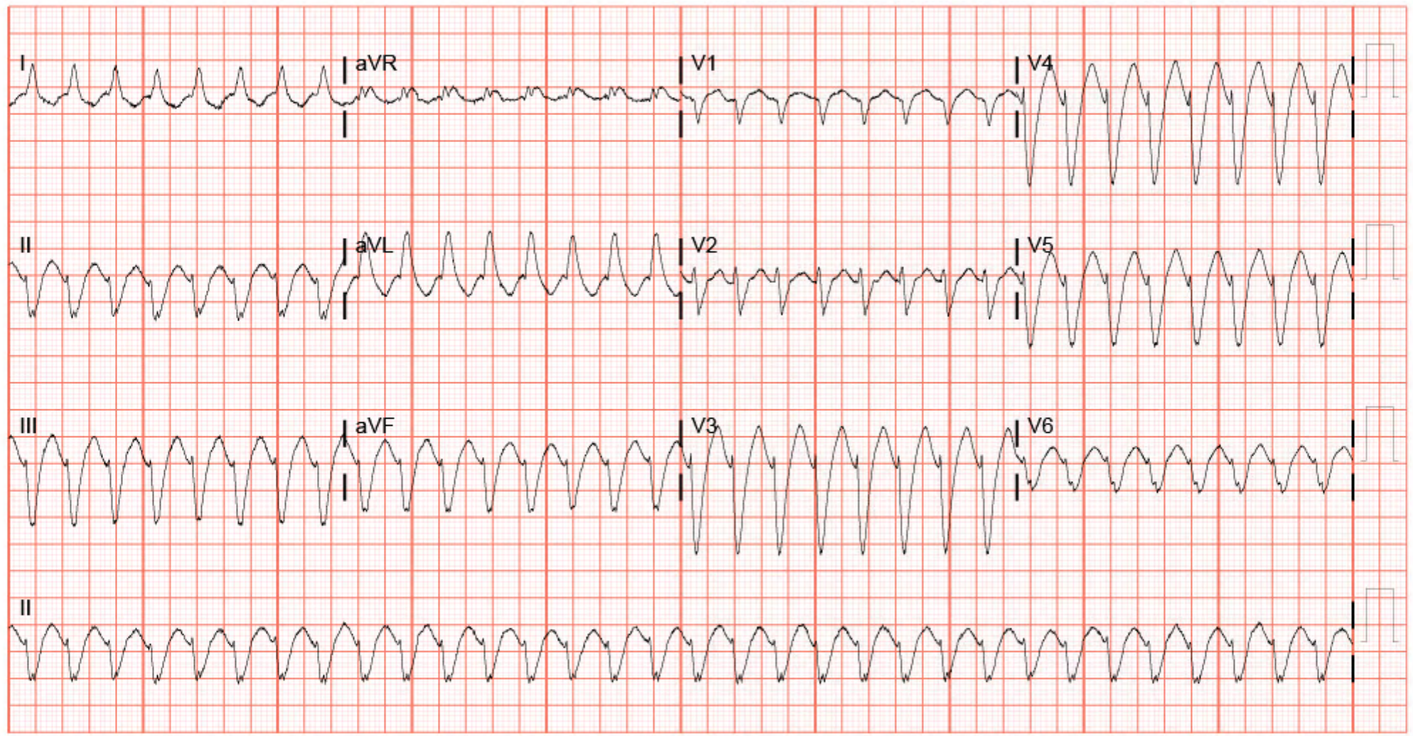
Sinus pause

Sinus arrest
(dropped P wave)

Exhibit Display



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A 63-year-old man comes to the office due to exertional dyspnea that has been progressively worsening over the last 3 weeks. He sleeps in a recliner with his head elevated because he gets short of breath when lying flat in bed. Medical history includes gout, dyslipidemia, and hypertension. Blood pressure is 154/89 mm Hg and pulse is 85/min. Physical examination shows distended jugular veins in the semi-recumbent position. An apical heave is present on cardiac examination. There is pitting edema in the bilateral lower extremities. Levels of which of the following substances will be higher in the pulmonary vein compared to the pulmonary artery in this patient?

- A. Aldosterone
- B. Angiotensin I
- C. Angiotensin II
- D. Angiotensinase
- E. Angiotensinogen
- F. Arginine vasopressin
- G. Prostaglandin E1
- H. Renin

Submit

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A 63-year-old man comes to the office due to exertional dyspnea that has been progressively worsening over the last 3 weeks. He sleeps in a recliner with his head elevated because he gets short of breath when lying flat in bed. Medical history includes gout, dyslipidemia, and hypertension. Blood pressure is 154/89 mm Hg and pulse is 85/min. Physical examination shows distended jugular veins in the semi-recumbent position. An apical heave is present on cardiac examination. There is pitting edema in the bilateral lower extremities. Levels of which of the following substances will be higher in the pulmonary vein compared to the pulmonary artery in this patient?

- A. Aldosterone (1%)
- ✗ B. Angiotensin I (14%)
- ✓ C. Angiotensin II (61%)
- D. Angiotensinase (2%)
- E. Angiotensinogen (4%)
- F. Arginine vasopressin (3%)
- G. Prostaglandin E1 (10%)
- H. Renin (2%)

Incorrect
Correct answer
C

61%
Answered correctly

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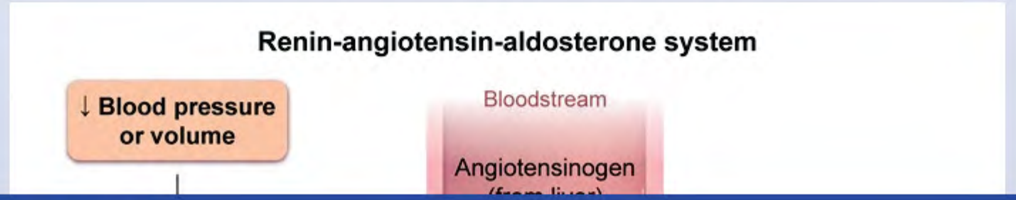
Explanation



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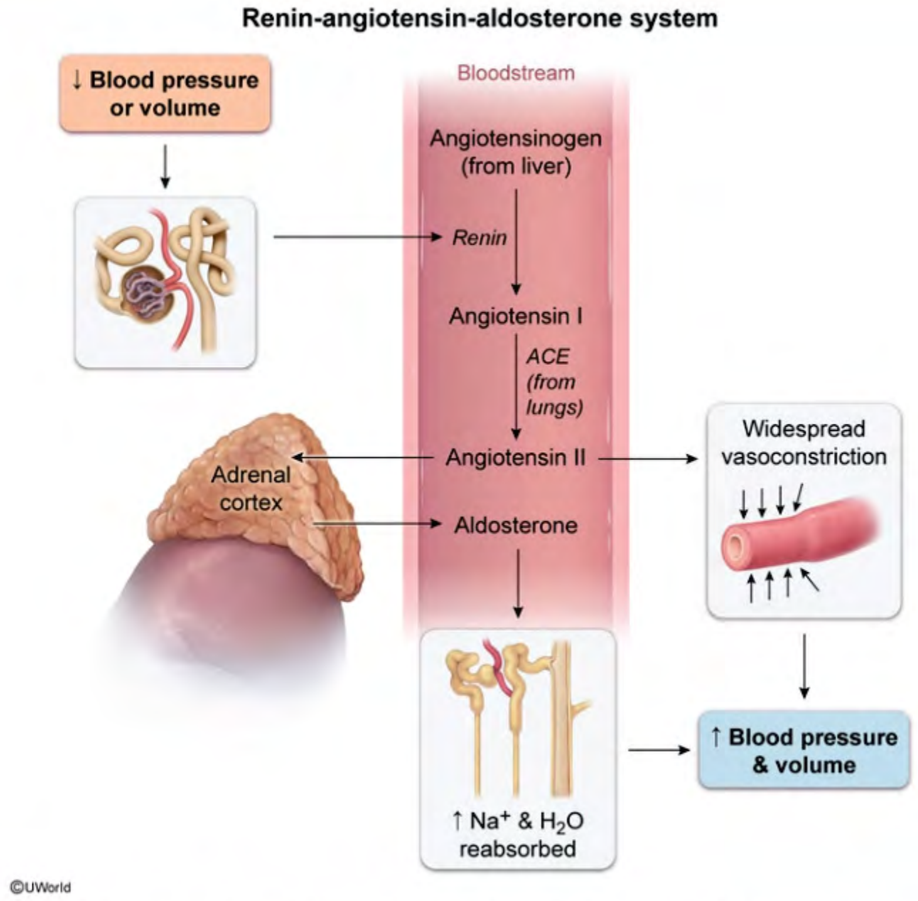

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This patient's exertional dyspnea, orthopnea (dyspnea when lying flat), jugular venous distension, cardiac heave, and peripheral edema are consistent with **decompensated heart failure**. The decreased cardiac output in heart failure reduces renal perfusion, which stimulates **renin secretion** by the juxtaglomerular cells of the kidney **(Choice H)** in a maladaptive effort to maintain effective blood volume and organ perfusion.

Renin acts to convert angiotensinogen (produced by the liver) into angiotensin I in the systemic circulation **(Choices B and E)**. Angiotensin I is then converted to **angiotensin II**, primarily within the **small pulmonary vessels**, by endothelial-bound angiotensin-converting enzyme. Because of the pulmonary location of angiotensin conversion, levels of angiotensin II are **higher in the pulmonary vein** (on the way out of the lungs) than in the pulmonary artery (on the way into the lungs).

Angiotensin II is a potent vasoconstrictor of the systemic circulation and causes a rise in blood pressure (increased afterload) that worsens cardiac output. It also acts on the adrenal glands to stimulate aldosterone release, causing sodium retention and further worsening of volume overload **(Choice A)**.

(Choice D) The angiotensinases are a group of enzymes found in multiple tissues and are responsible for the breakdown of angiotensin II and other structurally similar enzymes.

(Choice F) Arginine vasopressin (antidiuretic hormone) is released from the posterior pituitary in response to high serum osmolality or decreased arterial blood volume. Because reduced cardiac output causes the body to perceive hypovolemia, antidiuretic hormone secretion is typically increased in heart failure.

(Choice G) Prostaglandins are produced in multiple cell types, including endothelial cells, mast cells, and macrophages. Prostaglandin E1 is a potent vasodilator that maintains patency of the ductus arteriosus and causes afferent arteriolar vasodilation in the kidneys.

Educational objective:
The reduced cardiac output in heart failure leads to decreased renal perfusion and consequent stimulation of the renin-angiotensin-aldosterone system in a maladaptive effort to maintain effective blood volume. Inactive angiotensin I is converted into active angiotensin II by endothelial-bound angiotensin-converting enzyme in the lungs.

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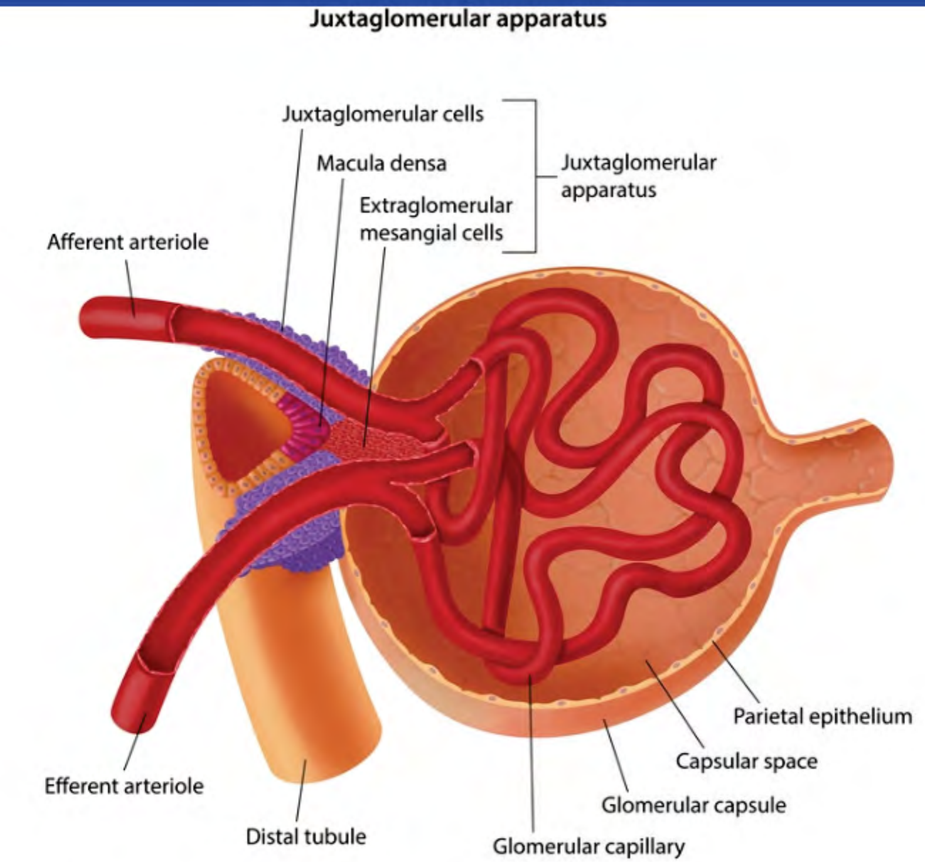

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A 52-year-old man is being evaluated for nonspecific, chronic abdominal pain. As part of the workup, he undergoes an abdominal CT scan with intravenous contrast, which reveals no significant findings. An axial image from the scan is shown below.

Which of the following statements best describes the structure indicated by the arrow?

- A. It drains into the left subclavian vein in the thorax.
- B. It drains through the cystic duct into the common bile duct.
- C. It gives rise to the right and left renal arteries.
- D. It is formed by the union of the common iliac veins.
- E. It joins the splenic vein to form the portal vein.

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- E. It joins the splenic vein to form the portal vein.

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from the scan is shown below:

Which of the following statements best describes the structure indicated by the arrow?

- ✗ A. It drains into the left subclavian vein in the thorax. (3%)
- B. It drains through the cystic duct into the common bile duct. (10%)
- C. It gives rise to the right and left renal arteries. (5%)
- ✓ D. It is formed by the union of the common iliac veins. (70%)
- E. It joins the splenic vein to form the portal vein. (10%)

Incorrect
Correct answer
D

70%
Answered correctly

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Incorrect
Correct answer
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70%
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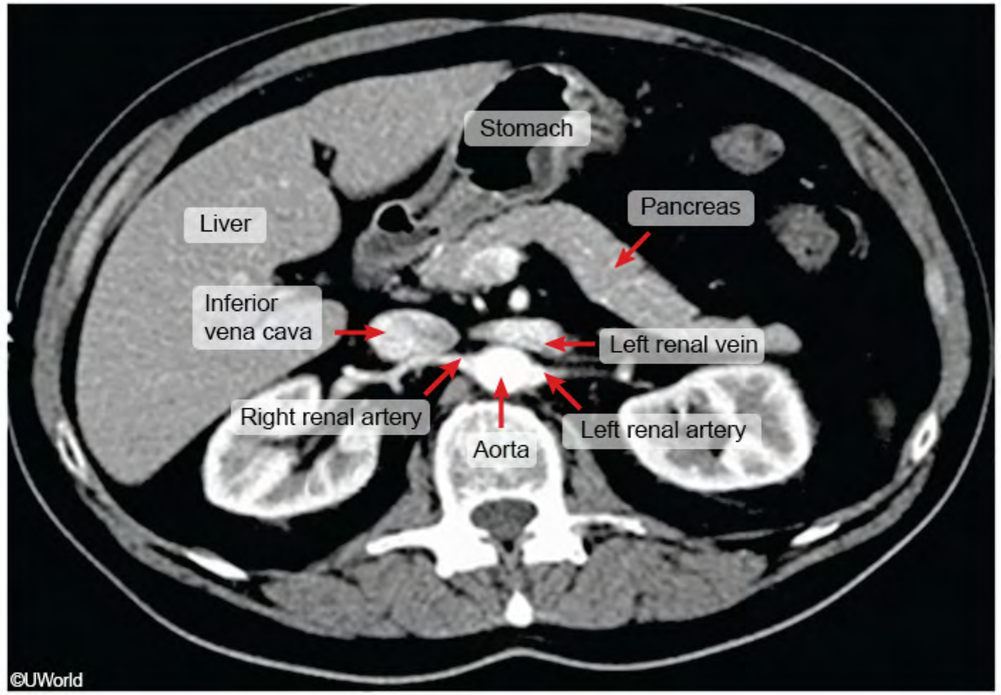

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(Choice A) The thoracic duct drains lymph from the entire left side of the body and all regions inferior to the

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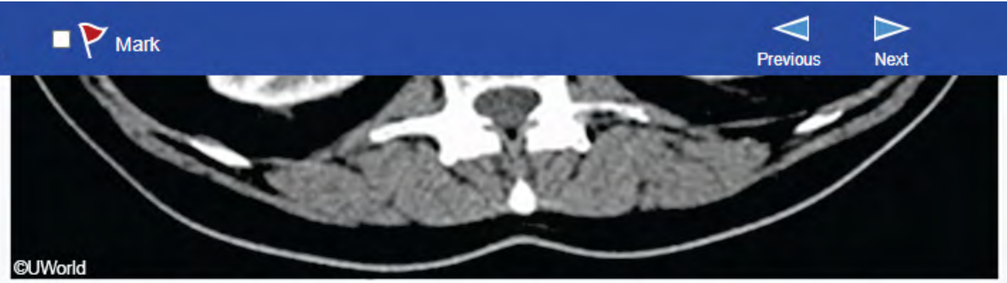

This axial CT image reveals the abdomen near the L1 vertebral level. At this level, the **inferior vena cava (IVC)** lies just anterior to the right renal artery and to the right of the aorta. The IVC is formed by the **union of the right and left common iliac veins** at the level of L4-L5 and drains into the right atrium just above the level of the diaphragm at T8. The IVC returns venous blood to the right atrium from the lower extremities, portal system, and abdominal and pelvic viscera.

(Choice A) The thoracic duct drains lymph from the entire left side of the body and all regions inferior to the umbilicus (including the entire gastrointestinal tract). It enters the thorax through the aortic hiatus and empties into the left subclavian vein near its junction with the internal jugular vein.

(Choice B) The cystic duct drains bile from the gallbladder into the common bile duct. The gallbladder is typically visualized just below the liver in the anterior right abdomen but is not well seen in this image.

(Choice C) The image reveals the abdominal aorta giving rise to the right and left renal arteries at the L1 vertebral level. The abdominal aorta lies posteromedial to the IVC within the abdomen.

(Choice E) The superior mesenteric vein joins the splenic vein to form the portal vein. The portal vein can be seen vaguely near the head of the pancreas in this cross-sectional image.

Educational objective:
The inferior vena cava is formed by the union of the right and left common iliac veins at the level of L4-L5. The renal arteries and veins lie at the level of L1. The inferior vena cava returns venous blood to the heart from the lower extremities, portal system, and abdominal and pelvic viscera.

Anatomy	Cardiovascular System	Cardiovascular anatomy
Subject	System	Topic

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A 34-year-old woman has recurrent throbbing headaches that occur several times a month and cause significant distress. They are accompanied by nausea and photophobia. She has had 5 emergency department visits for these headaches over the last 6 months. During office visits, her blood pressure ranges from 140/90 mm Hg to 150/95 mm Hg. Neurologic examination is unremarkable. The patient is prescribed metoprolol and is advised that the medication is expected to decrease the frequency of headaches and also lower blood pressure in the long term. Which of the following best explains this medication's effect on blood pressure?

- A. Decreased level of circulating catecholamines
- B. Decreased level of circulating renin
- C. Increased atrioventricular nodal conduction rate
- D. Increased renal sodium and water retention
- E. Long-term increase in peripheral vascular resistance

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- ✗ A. Decreased level of circulating catecholamines (14%)
- ✓ B. Decreased level of circulating renin (73%)
- C. Increased atrioventricular nodal conduction rate (6%)
- D. Increased renal sodium and water retention (1%)
- E. Long-term increase in peripheral vascular resistance (4%)

Incorrect
Correct answer
B

73%
Answered correctly

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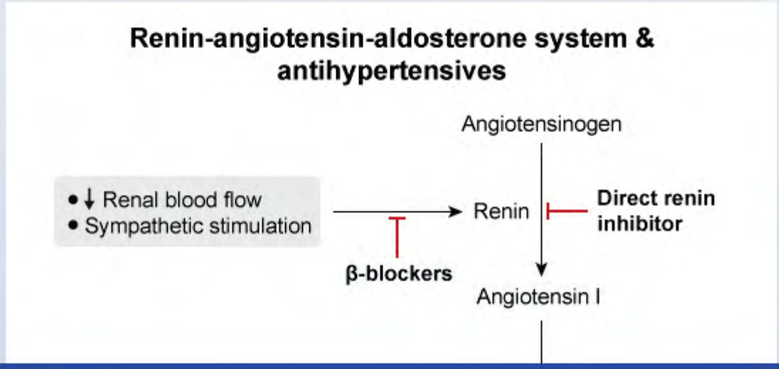
Explanation



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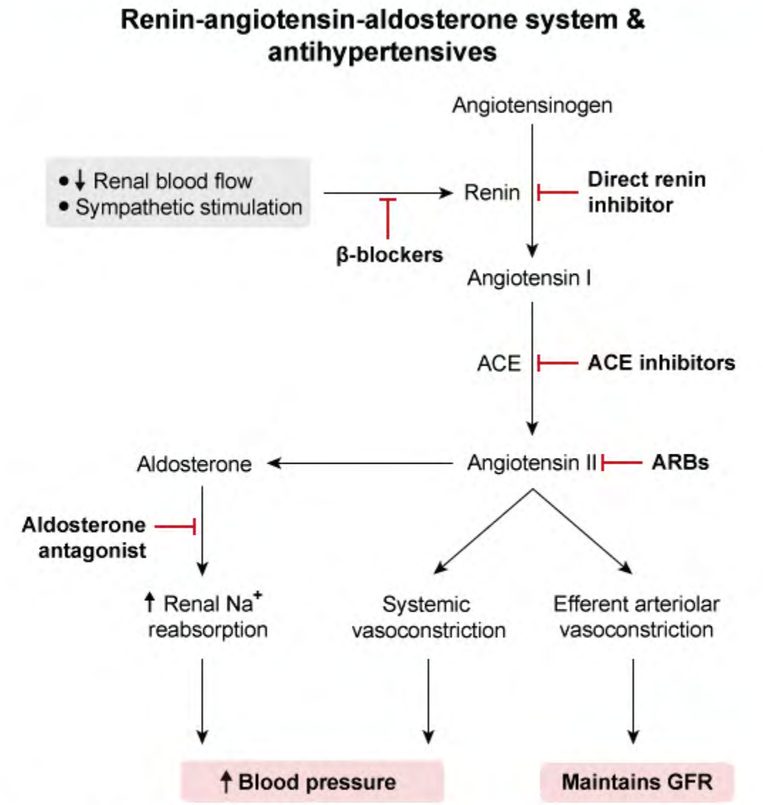

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Beta blockers are useful for treating hypertensive patients with comorbid conditions such as migraine, essential tremor, angina pectoris/prior myocardial infarction, and atrial fibrillation. Beta blockers lower blood pressure via 2 mechanisms:

1. Reducing myocardial contractility and heart rate
2. Decreasing renin release by the kidney

Renin release is mediated in part through sympathetic stimulation of beta-1 receptors located on juxtaglomerular cells. Therefore, beta-adrenergic blocking drugs (eg, metoprolol) act to **inhibit renin release**, which in turn reduces the conversion of angiotensinogen to angiotensin I and reduces the levels of angiotensin II (a potent vasoconstrictor) and aldosterone (decreasing renal sodium and water retention) **(Choice D)**.

(Choice A) Beta blockers block the action of endogenous catecholamines on adrenergic beta receptors but have no significant effect on circulating levels of catecholamine.

(Choice C) Beta-adrenergic receptor antagonists depress conduction through the atrioventricular (AV) node and can cause varying degrees of AV block.

(Choice E) Long-term use of beta blockers will cause either no change or modest decreases in the peripheral vascular resistance due to their effects on the renin-angiotensin-aldosterone system.

Educational objective:
Beta blockers inhibit release of renin from renal juxtaglomerular cells through antagonism of beta-1 receptors on these cells. Inhibition of renin release prevents activation of the renin-angiotensin-aldosterone pathway, which results in decreased vasoconstriction and decreased renal sodium and water retention.

References

- Beta-adrenergic receptor blockade as a therapeutic approach for suppressing the renin-angiotensin-aldosterone system in normotensive and hypertensive subjects.

Pharmacology	Cardiovascular System	Primary hypertension
Subject	System	Topic

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A physician is conducting a clinical trial for a new hemodynamic drug developed for a variety of indications. The drug's pharmacodynamic profile indicates that it is a pure adrenergic agonist with no significant antagonistic properties. It is infused into a volunteer, and hemodynamic parameters are recorded in the graphs below.

Administration of the drug to healthy individuals would most likely result in which of the following physiologic changes?

- A. Decreased atrioventricular node conduction velocity
- B. Decreased cardiac afterload
- C. Decreased pulmonary capillary wedge pressure
- D. Decreased venous tone
- E. Increased renal perfusion

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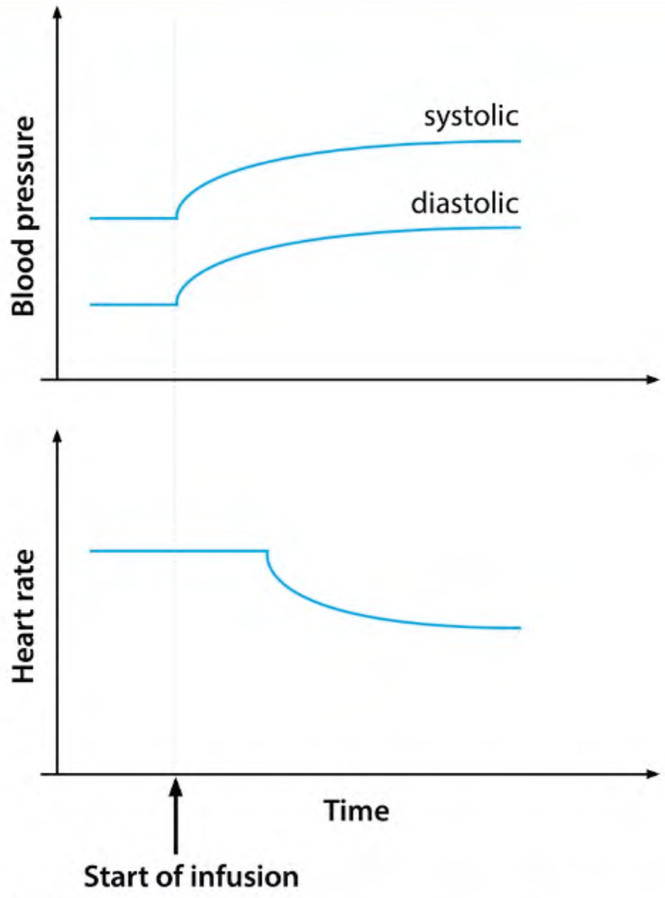

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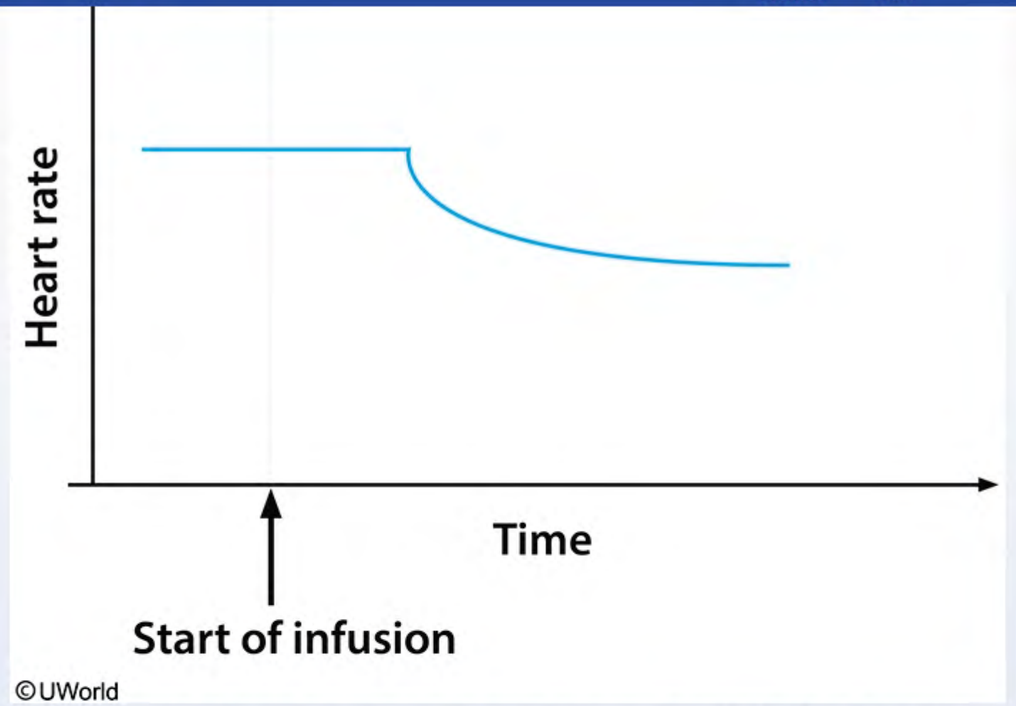

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- B. Decreased cardiac afterload
- C. Decreased pulmonary capillary wedge pressure
- D. Decreased venous tone
- E. Increased renal perfusion

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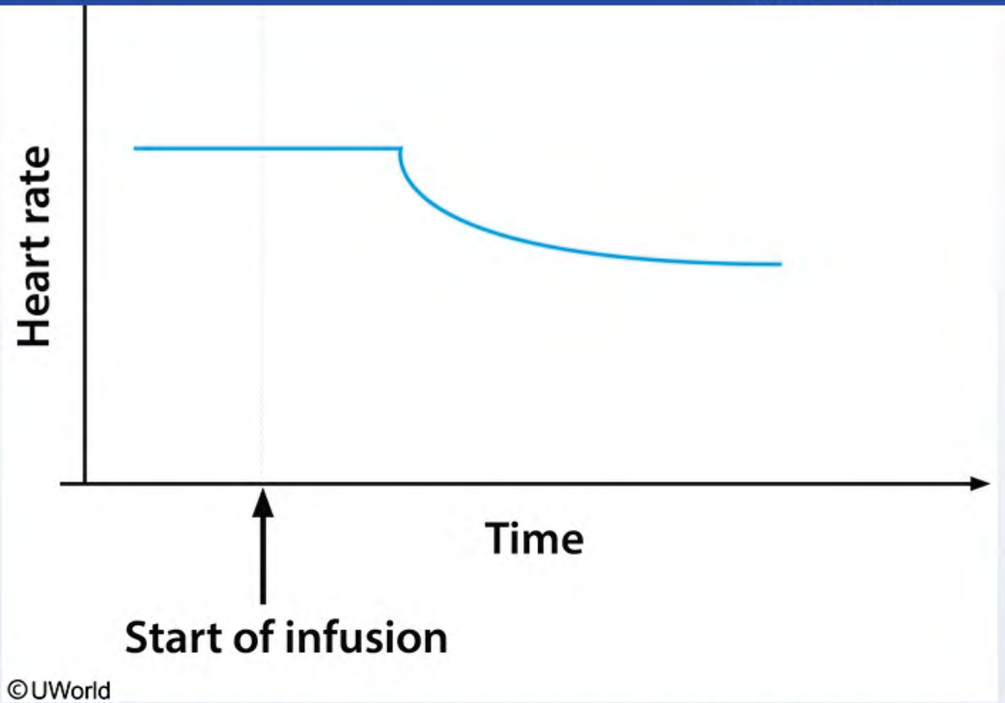

Administration of the drug to healthy individuals would most likely result in which of the following physiologic changes?

- ✓ A. Decreased atrioventricular node conduction velocity (56%)
- ✗ B. Decreased cardiac afterload (7%)
- C. Decreased pulmonary capillary wedge pressure (7%)
- D. Decreased venous tone (7%)
- E. Increased renal perfusion (20%)

Incorrect
Correct answer
A

56%
Answered correctly

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Explanation

Direct effects of α-adrenergic agonists	
α_1 mediated	**α_2 mediated**
• Vascular smooth muscle contraction • Mydriasis • ↑ Internal urethral sphincter tone & prostate contraction	• CNS mediated ↓ in blood pressure • ↓ Intraocular pressure • ↓ Lipolysis • ↓ Presynaptic norepinephrine release • ↑ Platelet aggregation

Administration of the drug caused an increase in systolic and diastolic blood pressures and a reflexive (delayed) decrease in heart rate; these are the typical effects of a **selective α_1-adrenergic agonist** (eg, phenylephrine, methoxamine). Activation of α_1-adrenoreceptors (in blood vessels of skin and viscera) leads to **vasoconstriction** and increased systemic blood pressure. Stimulation of baroreceptors in the carotid sinus and aortic arch causes a **reflexive increase in vagal influence** on the heart. The increased vagal tone results in inhibition of pacemaker activity of the sinoatrial node, slowed conduction through the atrioventricular node, and decreased myocardial contractility.

(Choice B) The vasoconstriction caused by α_1-adrenergic agonists increases blood pressure and cardiac afterload, requiring increased myocardial effort to expel the blood.

(Choices C and D) Pulmonary capillary wedge pressure reflects left atrial pressure. It is increased after administration of α_1-adrenergic agonists due to increases in cardiac afterload (elevates left ventricular end-diastolic pressure) and venous tone (results in augmented venous return).

(Choice E) α_1-adrenergic agonists cause vasoconstriction of the renal and splanchnic arteries, decreasing perfusion to the kidneys and other viscera.

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• Vascular smooth muscle contraction • Mydriasis • ↑ Internal urethral sphincter tone & prostate contraction	• CNS mediated ↓ in blood pressure • ↓ Intraocular pressure • ↓ Lipolysis • ↓ Presynaptic norepinephrine release • ↑ Platelet aggregation

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(Choice B) The vasoconstriction caused by α_1-adrenergic agonists increases blood pressure and cardiac afterload, requiring increased myocardial effort to expel the blood.

(Choices C and D) Pulmonary capillary wedge pressure reflects left atrial pressure. It is increased after administration of α_1-adrenergic agonists due to increases in cardiac afterload (elevates left ventricular end-diastolic pressure) and venous tone (results in augmented venous return).

(Choice E) α_1-adrenergic agonists cause vasoconstriction of the renal and splanchnic arteries, decreasing perfusion to the kidneys and other viscera.

Educational objective:
α-adrenergic agonists increase systolic and diastolic blood pressure by stimulating α_1-adrenoreceptors in the vascular walls, causing vasoconstriction. The elevated systemic blood pressure then causes a reflexive increase in vagal tone, resulting in decreased heart rate and slowed atrioventricular node conduction.

References

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A 62-year-old man is brought to the emergency department by a friend due to severe shortness of breath. He is agitated and gasping for air and is unable to provide history. His friend says the patient has a history of chronic alcohol use and attends Alcoholics Anonymous meetings. He is unaware of any other medical history. Temperature is 36.7 C (98 F), blood pressure is 110/70 mm Hg, pulse is 104/min, and respirations are 32/min. Due to worsening respiratory distress, immediate endotracheal intubation is performed and mechanical ventilation is begun. The patient is admitted to the intensive care unit and, despite appropriate therapy, dies several hours later. Autopsy examination is performed, and histopathology of the lungs reveals engorged pulmonary capillaries and abundant intraalveolar, acellular, pink material that is more prominent at the bases. This patient most likely had which of the following conditions?

- A. Aspiration pneumonia
- B. Centriacinar emphysema
- C. Hypersensitivity pneumonitis
- D. Pulmonary arterial hypertension
- E. Pulmonary edema
- F. Pulmonary infarction

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- ✗ A. Aspiration pneumonia (19%)
- B. Centriacinar emphysema (4%)
- C. Hypersensitivity pneumonitis (5%)
- D. Pulmonary arterial hypertension (19%)
- ✓ E. Pulmonary edema (43%)
- F. Pulmonary infarction (6%)

Incorrect
Correct answer
E

43%
Answered correctly

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Explanation

This patient's histopathologic findings of engorged alveolar capillaries (reflecting increased pulmonary venous pressure) and pink, acellular material within the alveoli (transuded plasma across the alveolar-capillary membrane) are most consistent with **acute pulmonary edema**. Given the patient's history of **chronic alcohol use**, he likely had underlying **dilated cardiomyopathy**. Subsequent decompensation then led to increased alveolar capillary

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This patient's histopathologic findings of engorged alveolar capillaries (reflecting increased pulmonary venous pressure) and pink, acellular material within the alveoli (transuded plasma across the alveolar-capillary membrane) are most consistent with **acute pulmonary edema**. Given the patient's history of **chronic alcohol use**, he likely had underlying **dilated cardiomyopathy**. Subsequent decompensation then led to increased alveolar capillary hydrostatic pressure, causing pulmonary congestion and acute respiratory failure.

(Choice A) Aspiration pneumonia may be chemical (eg, aspiration of gastric contents) or bacterial (eg, aspiration of oral flora). Histopathology of acute pneumonia shows neutrophil-rich, inflammatory exudates within the bronchioles and alveolar spaces.

(Choice B) Centriacinar emphysema most commonly results from heavy smoking. Enlarged airspaces with macrophage-predominant infiltrates are expected.

(Choice C) Hypersensitivity pneumonitis results from exposure to an environmental irritant (eg, mold). Histopathology shows lymphocyte-predominant, interstitial inflammation, often accompanied by non-necrotizing granulomas.

(Choice D) Histopathology in pulmonary arterial hypertension typically shows hypertrophy of the pulmonary vascular smooth muscle.

(Choice F) Pulmonary infarction yields hemorrhagic, ischemic necrosis of the lung parenchyma. Histopathology shows a well-demarcated area of alveolar and vascular wall necrosis with cells that lack nuclei, and the alveolar spaces may contain red blood cells.

Educational objective:
Acute cardiogenic pulmonary edema results from increased pulmonary venous pressure. The alveolar capillaries become engorged with blood and there is a transudation of fluid plasma across the alveolar-capillary membrane, appearing as pink, acellular material within the alveoli.

Pathology	Cardiovascular System	Acute heart failure
Subject	System	Topic

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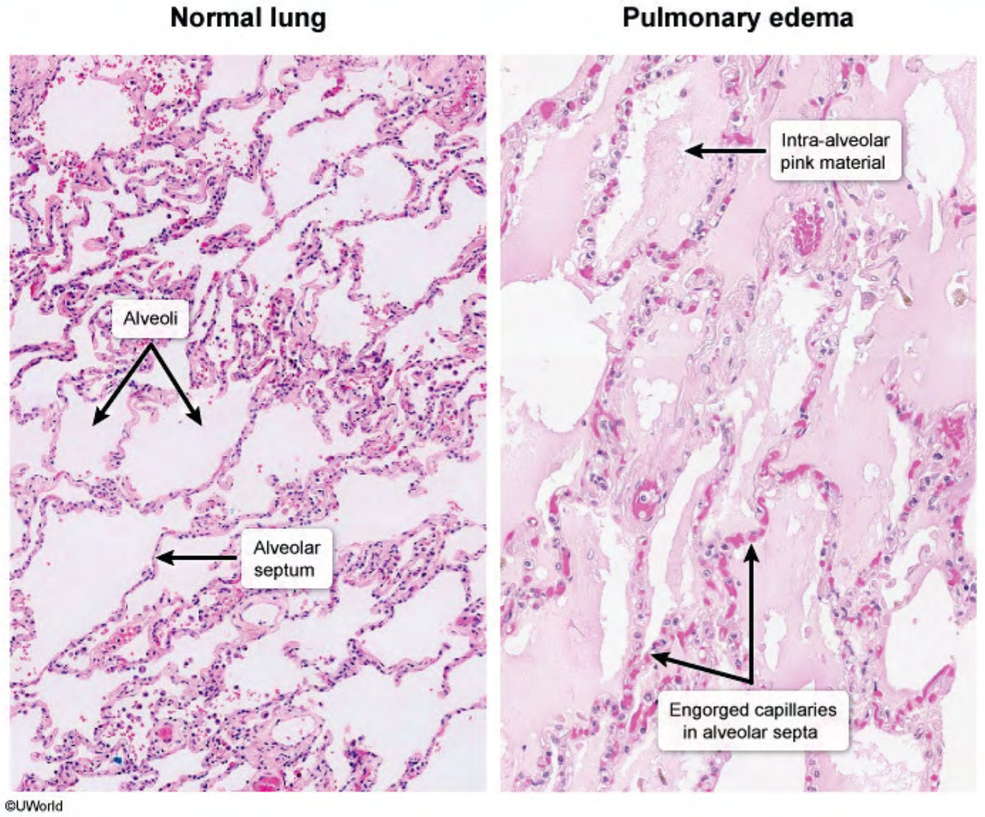

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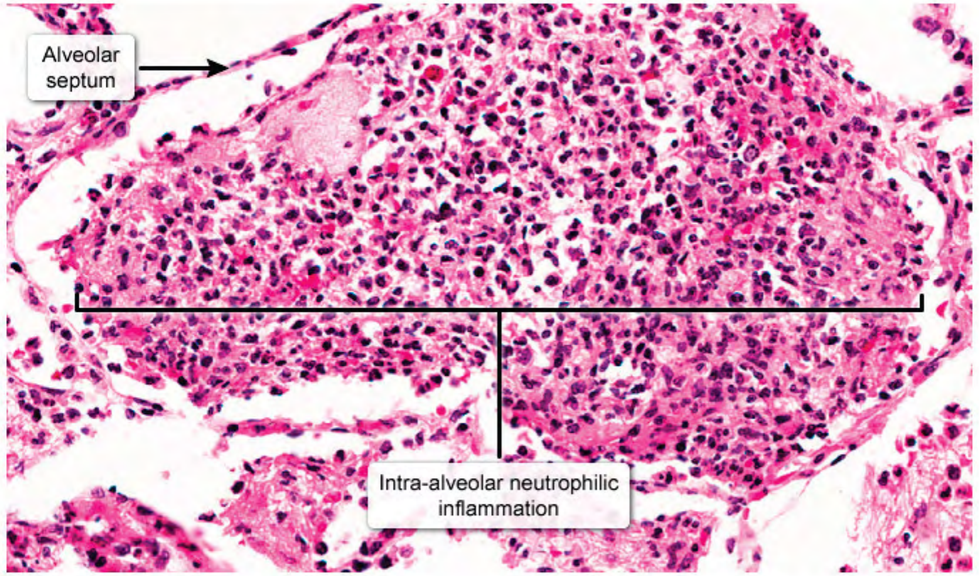

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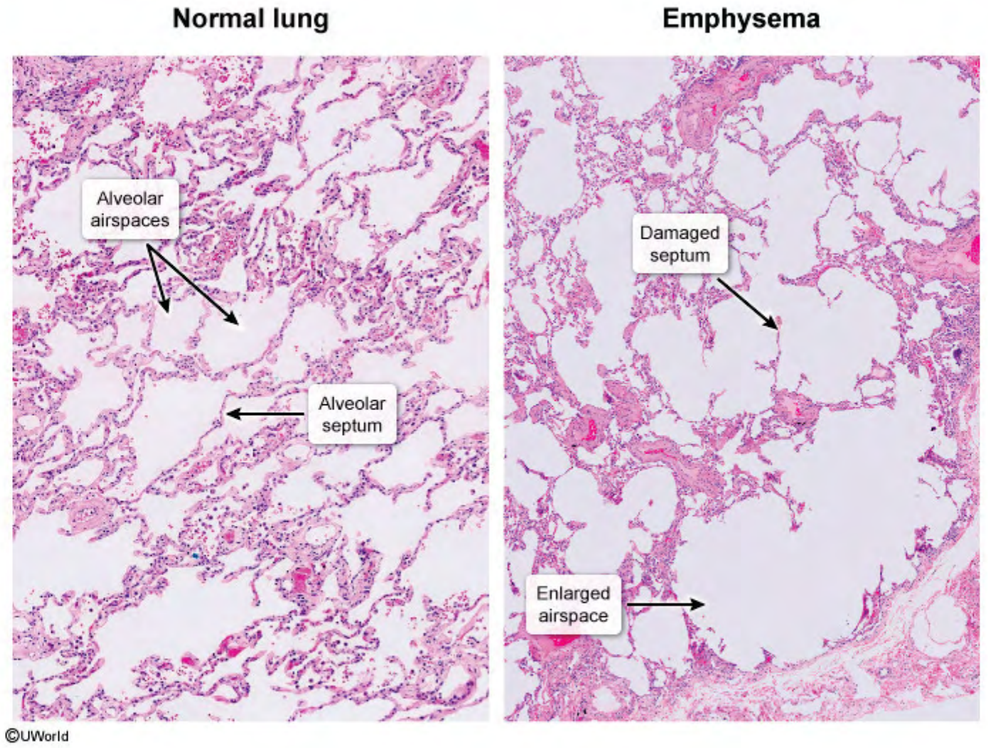

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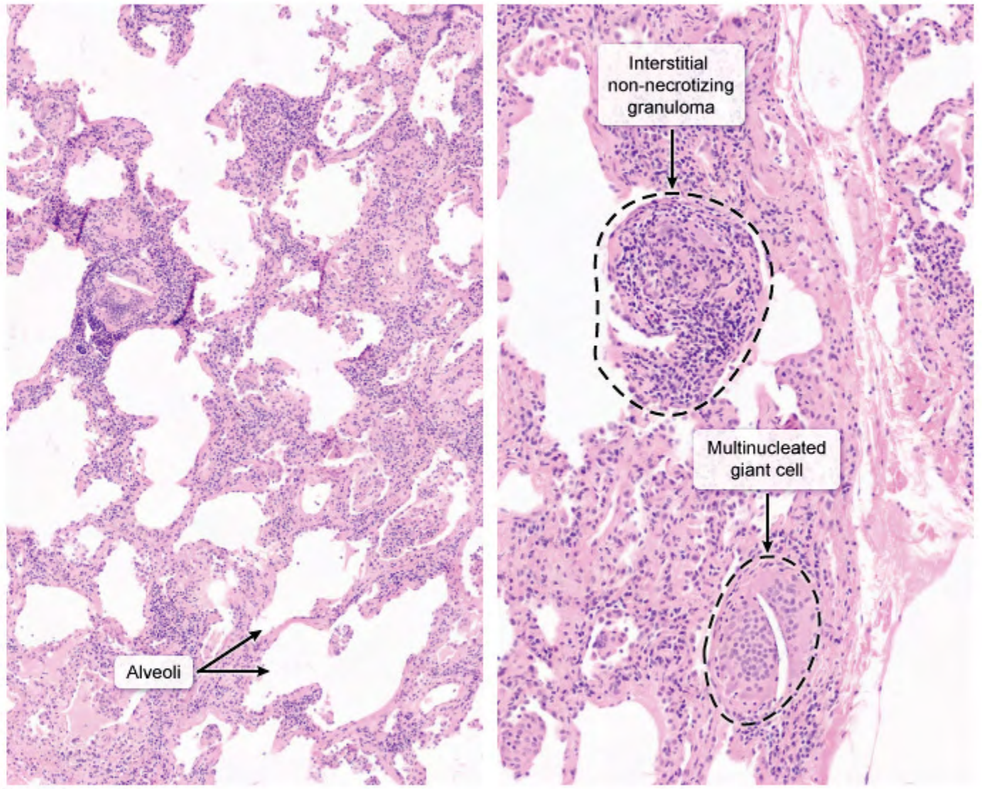
Hypersensitivity pneumonitis



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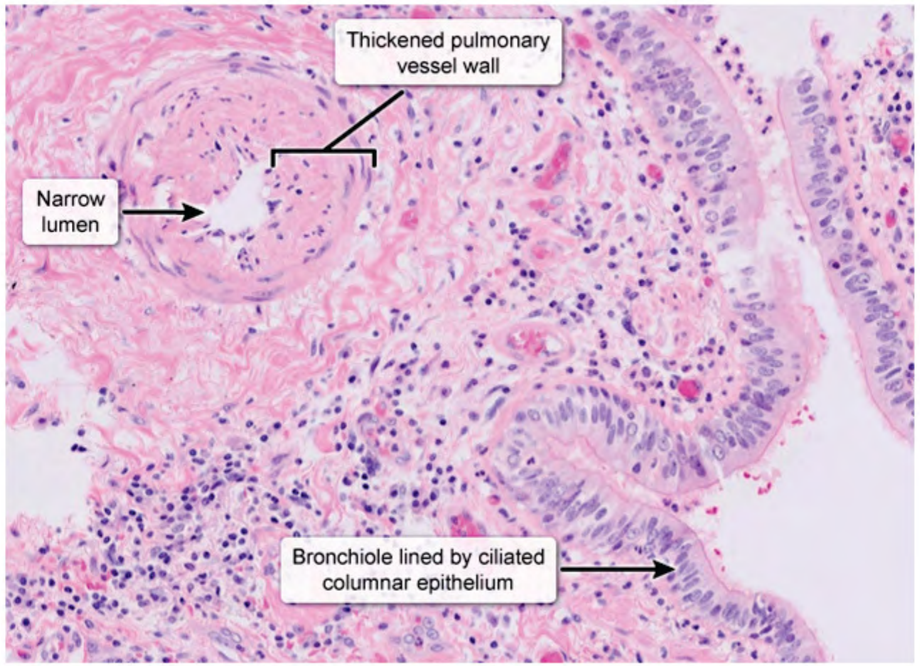
Pulmonary hypertension



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A 68-year-old man comes to the office due to several weeks of progressive exertional dyspnea and lower extremity edema. Medical history is significant for non-Hodgkin lymphoma, which is in remission after chemotherapy 8 years ago. Blood pressure is 126/76 mm Hg and pulse is 88/min. Physical examination reveals bibasilar lung crackles and 1+ bilateral lower extremity edema. Echocardiography shows biventricular dilation and a left ventricular ejection fraction of 35%. Stress myocardial perfusion scan is negative for inducible ischemia. After initial stabilization, long-term use of which of the following medications will most likely improve survival in this patient?

- A. Amiodarone
- B. Amlodipine
- C. Carvedilol
- D. Digoxin
- E. Diltiazem
- F. Flecainide
- G. Furosemide

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- A. Amiodarone (2%)
- ✗ B. Amlodipine (4%)
- ✓ C. Carvedilol (52%)
- D. Digoxin (16%)
- E. Diltiazem (3%)
- F. Flecainide (1%)
- G. Furosemide (18%)

Incorrect
Correct answer
C

52%
Answered correctly

04 secs
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Explanation

Pharmacotherapy for chronic heart failure with reduced ejection fraction	
Agent	**Mortality benefit**
Angiotensin receptor-neprilysin inhibitor (eg, sacubitril-valsartan) OR	

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Pharmacotherapy for chronic heart failure with reduced ejection fraction

Agent	Mortality benefit
Angiotensin receptor-neprilysin inhibitor (eg, sacubitril-valsartan) OR **ACE inhibitor** (eg, lisinopril) OR **Angiotensin II receptor blocker** (eg, losartan)	Yes
Beta blocker (eg, metoprolol, carvedilol)	Yes
Mineralocorticoid receptor antagonist (eg, spironolactone, eplerenone)	Yes
Sodium-glucose cotransporter-2 inhibitor (eg, dapagliflozin, empagliflozin)	Yes
Diuretic (eg, furosemide, metolazone)	No*
Digoxin	No**

*Improves symptoms & reduces hospitalization.
**Reduces hospitalization.

This patient's clinical presentation is consistent with decompensated **heart failure with reduced ejection fraction** (HFrEF) (left ventricular [LV] **systolic dysfunction** with ejection fraction <40%). Given the absence of evidence of ischemia, this patient's presentation is most likely due to dilated cardiomyopathy as an adverse effect of chemotherapy for non-Hodgkin lymphoma.

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Reduces hospitalization.

This patient's clinical presentation is consistent with decompensated **heart failure with reduced ejection fraction** (HFrEF) (left ventricular [LV] **systolic dysfunction** with ejection fraction <40%). Given the absence of evidence of ischemia, this patient's presentation is most likely due to dilated cardiomyopathy as an adverse effect of chemotherapy for non-Hodgkin lymphoma.

Beta blockers (eg, carvedilol, metoprolol) have been shown to **improve long-term survival** in patients with HFrEF. Much of the benefit of beta blockers in the treatment of HFrEF likely owes to **neurohormonal blockade of sympathetic drive**. This results in reduced heart rate and myocardial oxygen demand as well as decreased levels of circulating vasoconstrictors (eg, epinephrine, renin, endothelin), leading to reduced peripheral resistance (afterload) and myocardial work. These effects are cardioprotective, helping to **reduce cardiomyocyte death** and **prevent deleterious cardiac remodeling**.

Beta blockers should not be initiated in patients with acute decompensated heart failure (ie, heart failure requiring hospitalization), as they can further impair cardiac output; they should be introduced slowly after the patient has been stabilized. Other drugs that improve long-term survival in patients with HFrEF include ACE inhibitors, angiotensin II receptor blockers, angiotensin receptor-neprilysin inhibitors (eg, sacubitril-valsartan), and aldosterone antagonists (eg, spironolactone, eplerenone).

(Choice A) Amiodarone is a class III antiarrhythmic agent used for the management of atrial fibrillation and ventricular tachycardia, but it is not used for heart failure.

(Choices B and E) Dihydropyridine calcium channel blockers (eg, amlodipine) do not improve survival in patients with HFrEF. Nondihydropyridine calcium channel blockers (eg, diltiazem, verapamil) are specifically contraindicated in HFrEF due to the potential for reduced contractility and further worsening of LV systolic dysfunction.

(Choice D) Digoxin is sometimes used as an adjunctive therapy in patients with HFrEF. It improves symptoms and reduces hospitalizations, but it has not been shown to improve survival.

(Choice F) Flecainide is a class IC antiarrhythmic drug that is occasionally used for management of atrial fibrillation in patients with a structurally normal heart. It has no role in the management of heart failure.

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HFrEF. Much of the benefit of beta blockers in the treatment of HFrEF likely owes to **neurohormonal blockade of sympathetic drive**. This results in reduced heart rate and myocardial oxygen demand as well as decreased levels of circulating vasoconstrictors (eg, epinephrine, renin, endothelin), leading to reduced peripheral resistance (afterload) and myocardial work. These effects are cardioprotective, helping to **reduce cardiomyocyte death** and **prevent deleterious cardiac remodeling**.

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(Choice F) Flecainide is a class IC antiarrhythmic drug that is occasionally used for management of atrial fibrillation in patients with a structurally normal heart. It has no role in the management of heart failure.

(Choice G) Loop diuretics (eg, furosemide) are first-line agents for volume control in patients with heart failure. They improve symptoms and reduce hospitalizations, but they have not been shown to improve long-term survival.

Educational objective:
Drugs that have been shown to improve long-term survival in patients with heart failure with reduced ejection fraction include beta blockers, ACE inhibitors, angiotensin II receptor blockers, angiotensin receptor-neprilysin inhibitors, and aldosterone antagonists.

References

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A 43-year-old man comes to the emergency department after experiencing 4 episodes of coffee ground emesis that started earlier this morning. He also describes epigastric pain over the last 3-4 months that was relieved by over-the-counter antacids. He has no other past medical history. The patient has smoked a pack of cigarettes daily for the last 20 years but does not use alcohol or illicit drugs. Blood pressure is 70/40 mm Hg and pulse is 130/min and regular. Extremities are cool to the touch with loss of skin turgor. Intravenous access is obtained via 2 large-bore peripheral catheters, and rapid infusion of 2 liters of normal saline is initiated. This intervention is most likely to increase which of the following hemodynamic parameters?

- A. Aortic wall elasticity
- B. Diastolic ventricular compliance
- C. End-diastolic sarcomere length
- D. Heart rate
- E. Plasma renin activity
- F. Total peripheral resistance
- G. Ventricular muscle contraction velocity

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- A. Aortic wall elasticity (0%)
- ✗ B. Diastolic ventricular compliance (5%)
- ✓ C. End-diastolic sarcomere length (70%)
- D. Heart rate (1%)
- E. Plasma renin activity (2%)
- F. Total peripheral resistance (15%)
- G. Ventricular muscle contraction velocity (2%)

Incorrect
Correct answer
C

70%
Answered correctly

04 secs
Time Spent

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Explanation

This patient's hypotension, tachycardia, and cool extremities are indicative of hypovolemic shock most likely due to upper gastrointestinal bleeding from peptic ulcer disease (coffee ground emesis due to oxidation of heme iron by exposure to gastric acid). Decreased intravascular volume from significant blood loss **activates** the **sympathetic nervous system** and **renin-angiotensin-aldosterone system** (RAAS), which help to maintain systemic blood pressure via the following compensatory mechanisms:

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1. Stimulation of the heart results in increased contractility and heart rate, helping to maintain cardiac output.
2. Constriction of the arteriolar beds serves to increase total peripheral resistance and maintain end-organ perfusion. This also shunts blood away from the extremities and skin and toward the vital organs.
3. Constriction of the systemic veins increases venous return to the heart, thereby assisting in maintaining preload.
4. Increased renal sodium and water retention help to prevent further volume loss.

In the treatment of hypovolemic shock, the most important intervention other than identifying and eliminating the source of bleeding is **rapid infusion of blood** products and crystalloid solutions such as **normal saline**. By infusing intravenous fluids, intravascular volume and ventricular preload can be increased rapidly. The increase in preload stretches the myocardium and **increases the end-diastolic sarcomere length**, leading to an increase in stroke volume and cardiac output by the Frank-Starling mechanism.

(Choice A) Aortic wall elasticity is based on the intrinsic structure of the aortic media and is not affected by fluid infusions.

(Choice B) The diastolic ventricular compliance is determined by intrinsic properties of the myocardium and is unaffected by crystalloid infusions. Conditions that decrease ventricular compliance include left ventricular hypertrophy (aortic stenosis, hypertension), hypertrophic cardiomyopathy, and infiltrative disorders (amyloidosis, sarcoidosis).

(Choice D) Adequate fluid resuscitation in a hypovolemic patient decreases the sympathetic drive and endogenous catecholamine release, resulting in decreased heart rate.

(Choice E) Plasma renin activity increases in patients with hypovolemic shock due to activation of RAAS and would be expected to decrease after intravenous fluid resuscitation.

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preload.

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(Choice E) Plasma renin activity increases in patients with hypovolemic shock due to activation of RAAS and would be expected to decrease after intravenous fluid resuscitation.

(Choices F and G) In a patient with hypovolemic shock, total peripheral resistance and myocardial contraction velocity are high due to sympathetic activation. Administration of fluids will reduce sympathetic activation and decrease both of these parameters.

Educational objective:
Intravenous fluids increase the intravascular and left ventricular end-diastolic volumes. The increase in preload stretches the myocardium and increases the end-diastolic sarcomere length, leading to an increase in stroke volume and cardiac output by the Frank-Starling mechanism.

Physiology	Cardiovascular System	Hypovolemia
Subject	System	Topic

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(Choice D) Adequate fluid resuscitation in a hypovolemic patient decreases the sympathetic drive and

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Hemodynamic measurements in shock			
Parameter	**Hypovolemic shock**	**Cardiogenic shock**	**Septic shock**
Right atrial pressure (preload)	↓	↑	Normal to slight ↓
Pulmonary capillary wedge pressure (preload)	↓	↑	Normal to slight ↓
Cardiac output (pump function)	↓	↓↓	↑
Systemic vascular resistance (afterload)	↑	↑	↓

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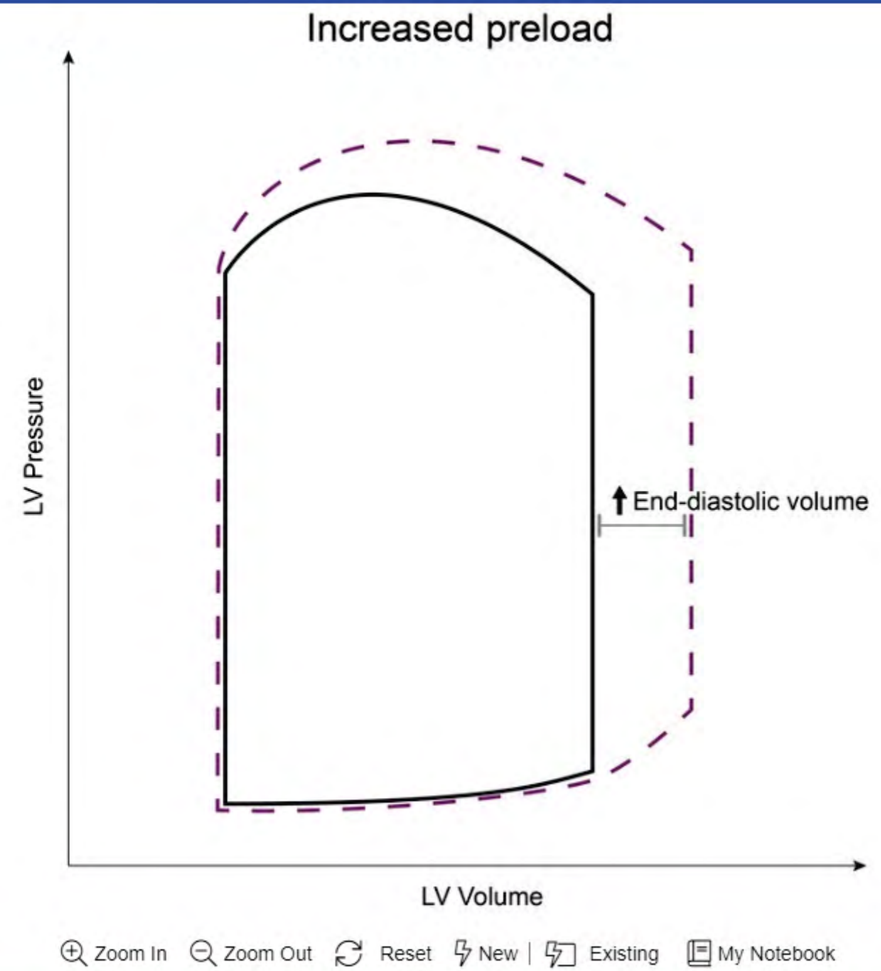

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An 80-year-old man is brought to the hospital due to increasing chest pain. Over the past 6 weeks, he has had dry cough, dysphagia, and a change in voice quality. The patient has a long history of hypertension. He is an ex-smoker with a 35-pack-year history. On physical examination, the patient appears anxious, pale, and diaphoretic. Blood pressure is 80/60 mm Hg, pulse is 120/min, and respirations are 20/min. While being evaluated in the emergency department, he develops cardiac arrest with pulseless electrical activity and cannot be resuscitated. Autopsy is most likely to show which of the following findings?

- A. Fat globules in the pulmonary vasculature
- B. Left ventricular free-wall rupture
- C. Longitudinal tear in the esophagus
- D. Ruptured pulmonary bleb
- E. Ruptured thoracic aortic aneurysm

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- A. Fat globules in the pulmonary vasculature (2%)
- ✗ B. Left ventricular free-wall rupture (15%)
- C. Longitudinal tear in the esophagus (7%)
- D. Ruptured pulmonary bleb (5%)
- ✓ E. Ruptured thoracic aortic aneurysm (68%)

Incorrect
Correct answer
E

68%
Answered correctly

04 secs
Time Spent

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Explanation

Thoracic aortic aneurysm	
Risk factors	• Advanced age (>60) • Smoking, hypertension, family history • Connective tissue disease (eg, Marfan syndrome)
	• Majority involve ascending aorta • Usually asymptomatic

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Thoracic aortic aneurysm	
Risk factors	• Advanced age (>60) • Smoking, hypertension, family history • Connective tissue disease (eg, Marfan syndrome)

Clinical presentation	• Majority involve ascending aorta
	• Usually asymptomatic
	• Expansion:
	  ◦ Vague chest/back pain
	  ◦ Compression of structures (eg, hoarseness, dysphagia)
	• Rupture/dissection:
	  ◦ Severe pain, hemodynamic instability

Imaging	• CXR: wide mediastinum, large aortic knob, tracheal deviation

CXR = chest x-ray.

Thoracic aortic aneurysm (TAA) can involve the ascending or descending aorta above the diaphragm. It is usually asymptomatic until it grows large enough to encroach on nearby structures or cause rupture. This patient likely experienced progressive TAA expansion with symptomatic **compression** of **surrounding structures**, followed by acute rupture that led to shock and cardiac arrest.

TAA usually results from age-related degenerative changes in the medial layer of the aorta; these changes are likely accelerated by risk factors, including dyslipidemia, hypertension, tobacco use, and family history. Connective tissue diseases such as Marfan or Ehlers-Danlos syndrome also increase the risk.

The most common symptomatic presentation of TAA is pain, which is typically localized to the chest and back. If the TAA impinges upon the **esophagus**, it can also cause dysphagia. Similarly, compression of the **left recurrent**

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The most common symptomatic presentation of TAA is pain, which is typically localized to the chest and back. If the TAA impinges upon the **esophagus**, it can also cause dysphagia. Similarly, compression of the **left recurrent laryngeal nerve** or left vagus nerve results in hoarseness, whereas compression of the **phrenic nerve** can cause hemidiaphragmatic paralysis. Respiratory manifestations, including wheeze, cough, hemoptysis, and dyspnea may occur due to **tracheobronchial obstruction**. Other complications include heart failure due to aortic valve regurgitation and superior vena cava syndrome from venous compression and occlusion.

Chest x-ray may suggest the diagnosis of TAA, demonstrating a widened mediastinum, enlarged aortic knob, and tracheal deviation.

(Choice A) Pulmonary fat embolism most commonly results in acute dyspnea, tachypnea, and hypoxemia. Patients commonly develop neurologic abnormalities and a petechial rash, both of which this patient lacks.

(Choice B) Ventricular free-wall rupture is a life-threatening complication of ST elevation myocardial infarction that typically occurs 5 days to 2 weeks after the event. It can also result in shock and cardiac arrest, but it would not explain this patient's subacute cough, voice change, or dysphagia.

(Choice C) Patients with a Mallory-Weiss tear have hematemesis and epigastric or back pain. These esophageal tears are usually preceded by episodes of nonbloody vomiting or retching.

(Choice D) Spontaneous pneumothorax results from the rupture of a pulmonary bleb and can occur with or without a preceding history of lung disease. Most patients describe acute chest pain and dyspnea. Although it can rarely progress to a tension pneumothorax with obstructive shock, such a condition would not explain this patient's

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Educational objective:
Thoracic aortic aneurysms are usually asymptomatic until they grow large enough to compress surrounding structures or cause rupture. The most common symptomatic presentation is chest or back pain, but compression of nearby structures can cause dysphagia, hoarseness, cough, or dyspnea.

References

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TAA usually results from age-related degenerative changes in the medial layer of the aorta; these changes are likely accelerated by risk factors, including dyslipidemia, hypertension, tobacco use, and family history. Connective tissue

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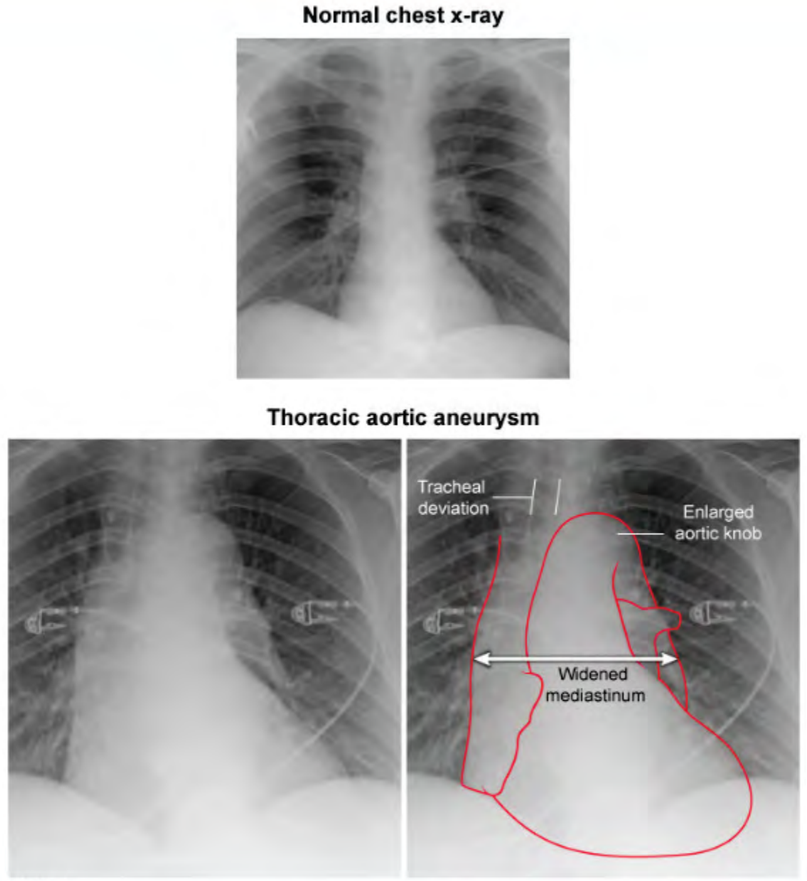

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A 62-year-old woman with no significant medical history is brought to the emergency department due to fatigue and substernal chest pain that began 6 hours ago. On arrival, ECG is consistent with ST-segment elevation myocardial infarction. Coronary angiography shows right-dominant coronary circulation and a thrombotic occlusion of the proximal right coronary artery, which supplies blood to the patient's right ventricle and the inferior and posterior portions of the left ventricle. A drug-eluting stent is placed, and she is discharged 2 days later without complication. After 6 weeks, the patient follows up in the clinic and undergoes echocardiography that shows left ventricular contractile dysfunction with normal right ventricular function. Which of the following characteristics of the right ventricle compared to the left ventricle best explains these echocardiography findings?

- A. Higher resting oxygen extraction
- B. Less dependence on collateral circulation
- C. Less ischemic preconditioning
- D. More active fibroblast proliferation
- E. Perfusion during both systole and diastole

Submit

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- ✗ A. Higher resting oxygen extraction (9%)
- B. Less dependence on collateral circulation (28%)
- C. Less ischemic preconditioning (20%)
- D. More active fibroblast proliferation (3%)
- ✓ E. Perfusion during both systole and diastole (37%)

Incorrect
Correct answer
E

37%
Answered correctly

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Time Spent

2023
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Explanation

Characteristics of the left & right ventricle		
	Left ventricle	**Right ventricle**
Muscle mass	High	Low
Resting oxygen extraction	High	Low
Coronary perfusion	During diastole only	Throughout cardiac cycle

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Explanation

Characteristics of the left & right ventricle		
	Left ventricle	**Right ventricle**
Muscle mass	High	Low
Resting oxygen extraction	High	Low
Coronary perfusion	During diastole only	Throughout cardiac cycle
Collateral circulation	Less developed	More developed
Ischemic preconditioning	Low	High

Myocardial infarction (MI) involving the left ventricle (LV) commonly leads to scarring and a sustained reduction in contractile function; however, **contractile function of the right ventricle** (RV) almost always **returns to normal** following MI. Some of the reasons for protection of the RV from infarction include:

- The relatively **small muscle mass and afterload** of the RV create less oxygen demand and necessitate lower oxygen extraction at rest. This allows for a **large capacity to increase oxygen extraction** during periods of ischemia.
- The relatively low systolic pressure of the RV (eg, ≤25 mm Hg) allows for **coronary perfusion throughout the cardiac cycle**; during MI, this enables the RV to pull blood flow from collateral vessels during both systole and diastole. In contrast, the high systolic pressure of the LV (eg, 120 mm Hg) blocks coronary blood flow of the LV walls during systole, allowing for perfusion during diastole only.

These factors likely contribute to the typical clinical course of MI involving the RV, which is acute RV contractile dysfunction often leading to profound hypotension and hemodynamic instability; acute mortality risk is high, but there is minimal long-term RV dysfunction for those who survive.

(Choice A) The large muscle mass of the LV necessitates higher oxygen extraction at rest, with relatively little capacity to increase oxygen extraction during periods of ischemia.

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These factors likely contribute to the typical clinical course of MI involving the RV, which is acute RV contractile dysfunction often leading to profound hypotension and hemodynamic instability; acute mortality risk is high, but there is minimal long-term RV dysfunction for those who survive.

(Choice A) The large muscle mass of the LV necessitates higher oxygen extraction at rest, with relatively little capacity to increase oxygen extraction during periods of ischemia.

(Choice B) The most robust collateral flow occurs from the left to the right coronary arteries, suggesting the RV has a more developed collateral circulation and may be more dependent on collateral flow than the LV.

(Choice C) Ischemic preconditioning refers to the myocardium being repeatedly exposed to mildly ischemic states (eg, during periods of exercise), which stimulates adjustments (eg, collateral circulation development, cellular improvements in oxygen utilization) that allow future ischemic states to be better tolerated. The RV is thought to experience heightened ischemic preconditioning compared to the LV, contributing to protection of the RV from infarction.

(Choice D) Fibroblast proliferation is more prominent in the muscular LV, where repair of a larger mass of tissue is needed.

Educational objective:
The right ventricle (RV) is relatively protected from myocardial infarction (MI), and contractile function of the RV usually returns to normal following MI. Factors contributing to this protection include relatively small muscle mass with high capacity to increase oxygen extraction, perfusion throughout the cardiac cycle, and heightened ischemic preconditioning.

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The right ventricle (RV) is relatively protected from myocardial infarction (MI), and contractile function of the RV usually returns to normal following MI. Factors contributing to this protection include relatively small muscle mass

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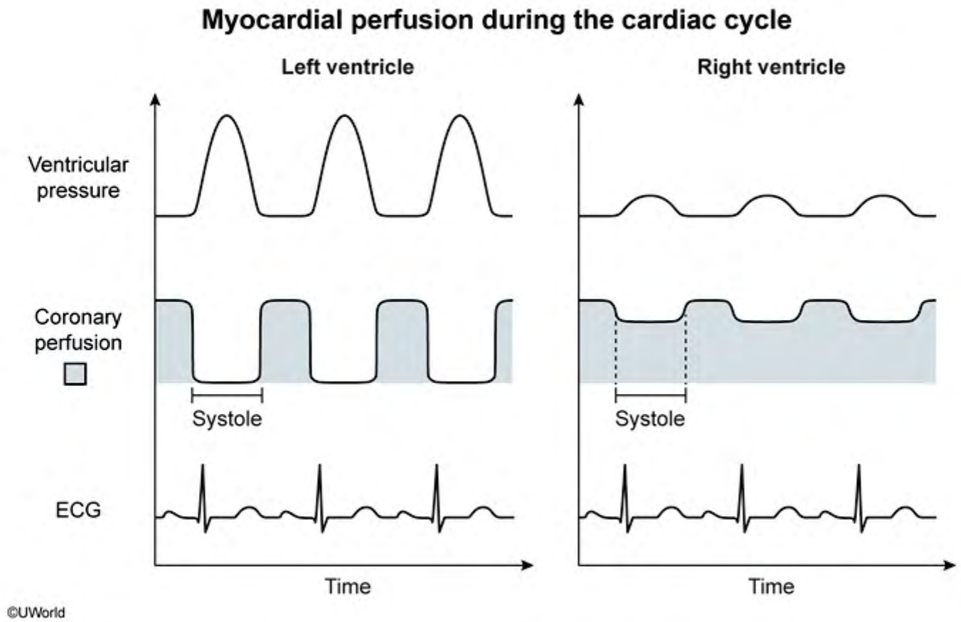

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A 16-year-old boy is evaluated in the office due to leg cramps. He plays soccer but frequently stops in the middle of practice due to cramps in both legs. The patient is afebrile. Blood pressure is 170/90 mm Hg bilaterally in the upper extremities. The point of maximal impulse is nondisplaced. S1 is present and S2 is physiologically split. An S4 is heard, as well as a continuous murmur best auscultated over the back at the left interscapular area. There is no appreciable murmur on auscultation of the anterior chest. Distal pulses in the radial arteries are prominent. Dorsalis pedis and posterior tibial pulses are difficult to palpate. The rest of the physical examination is unremarkable. Which of the following is the most likely cause of this patient's murmur?

- A. Increased blood flow across the tricuspid valve
- B. Increased blood flow through collateral blood vessels
- C. Increased cardiac output due to low systemic vascular resistance
- D. Increased pulmonary arterial blood flow
- E. Persistence of the ductus arteriosus

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Explanation

Coarctation of the aorta	
Pathology	• Thickening of tunica media of aortic arch • Can be associated with Turner syndrome or bicuspid aortic valve
	• ↑ BP & strong pulses in upper extremities • ↓ BP & weak pulses in lower extremities • Neonates (severe narrowing)

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Coarctation of the aorta	
Pathology	• Thickening of tunica media of aortic arch • Can be associated with Turner syndrome or bicuspid aortic valve

Clinical features	• ↑ BP & strong pulses in upper extremities
	• ↓ BP & weak pulses in lower extremities
	• Neonates (severe narrowing)
	  ◦ Differential cyanosis affecting lower extremities*
	  ◦ Heart failure & shock after ductal closure
	• Children/adults (mild narrowing)
	  ◦ Lower extremity claudication
	  ◦ Palpable pulsations of intercostal vessels (collaterals)
	  ◦ Secondary hypertension (upper arms & head)

*Patent ductus arteriosus shunts blood into descending aorta. **BP** = blood pressure.	

This patient has **upper extremity hypertension** with diminished lower extremity pulses. These findings are consistent with **coarctation of the aorta** (COA), a congenital defect involving aortic narrowing that is usually just distal to the left subclavian artery. The defect leads to increased aortic pressure proximally and decreased flow distally.

Patients with severe COA may develop cardiogenic shock in the neonatal period due critical aortic obstruction. In contrast, those with milder COA typically present in childhood or adulthood as enough blood is able to pass through the defect to prevent development of heart failure; findings include hypertension and bounding pulses in the upper extremities with **hypotension and diminished pulses** in the **lower extremities**. Decreased distal perfusion can also lead to lower extremity **claudication** (leg pain/cramps with exertion).

Over time, a compensatory network of collateral vessels that bypasses the coarctation can develop. For instance, distal branches of the ascending aorta (eg, internal thoracic artery) can supply the intercostal arteries below the

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through the defect to prevent development of heart failure; findings include hypertension and bounding pulses in the upper extremities with **hypotension and diminished pulses** in the **lower extremities**. Decreased distal perfusion can also lead to lower extremity **claudication** (leg pain/cramps with exertion).

Over time, a compensatory network of collateral vessels that bypasses the coarctation can develop. For instance, distal branches of the ascending aorta (eg, internal thoracic artery) can supply the intercostal arteries below the obstruction; the relatively low pressure distally allows retrograde flow through the intercostal arteries into the descending aorta. This flow through **collateral blood vessels** frequently causes a **continuous murmur** that can be heard over the back in the **interscapular area**; patients may also have a systolic murmur caused by flow through the coarctation itself. Concentric left ventricular hypertrophy can also develop due to pressure overload, creating an **S4**.

(Choices A and D) An atrial septal defect is a left-to-right intracardiac shunt that leads to right-sided volume overload, causing increased flow through the tricuspid valve and pulmonary arteries. Examination reveals a fixed and widely split S2 and a systolic ejection murmur best heard over the second left intercostal space (ie, pulmonic flow murmur).

(Choice C) Increased cardiac output due to low systemic vascular resistance (SVR) can occur during septic shock. SVR is effectively increased in COA due to the aortic obstruction.

(Choice E) A patent ductus arteriosus (PDA) typically causes a continuous murmur, but it is best heard over the left infraclavicular area (anterior chest). In addition, a PDA would not explain this patient's prominent difference in upper and lower extremity pulse strength.

Educational objective:
Aortic coarctation classically leads to lower extremity claudication and increased blood pressure in the arms compared to the legs. Over time, if the defect remains unrepaired, a compensatory collateral circulation develops and supplies blood to the descending aorta. Blood flow through collateral vessels creates a continuous murmur heard over the interscapular area.

References

- Coarctation of the aorta.
- Coarctation of the aorta and the nature of collateral circulation

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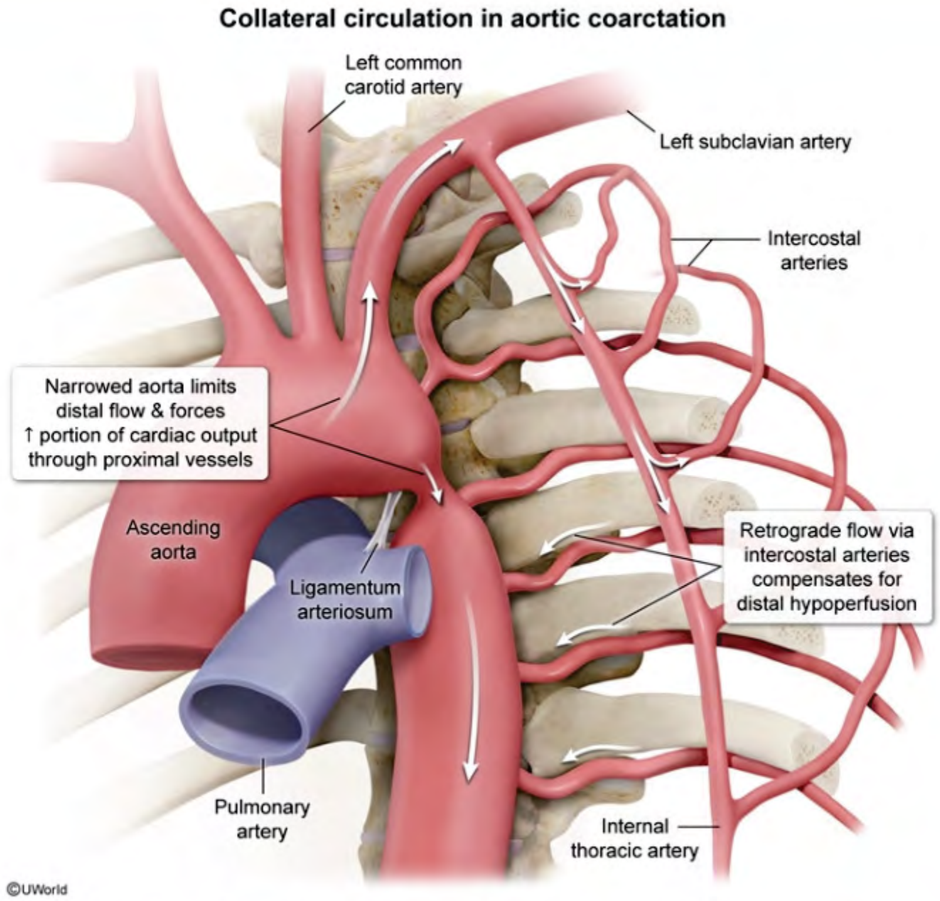

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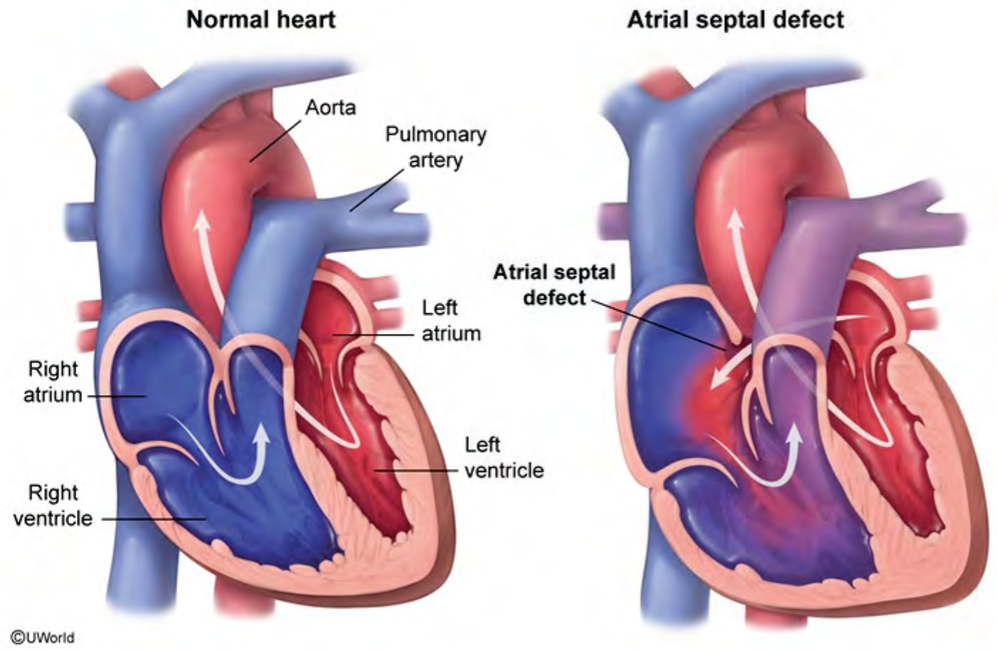

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Educational objective:

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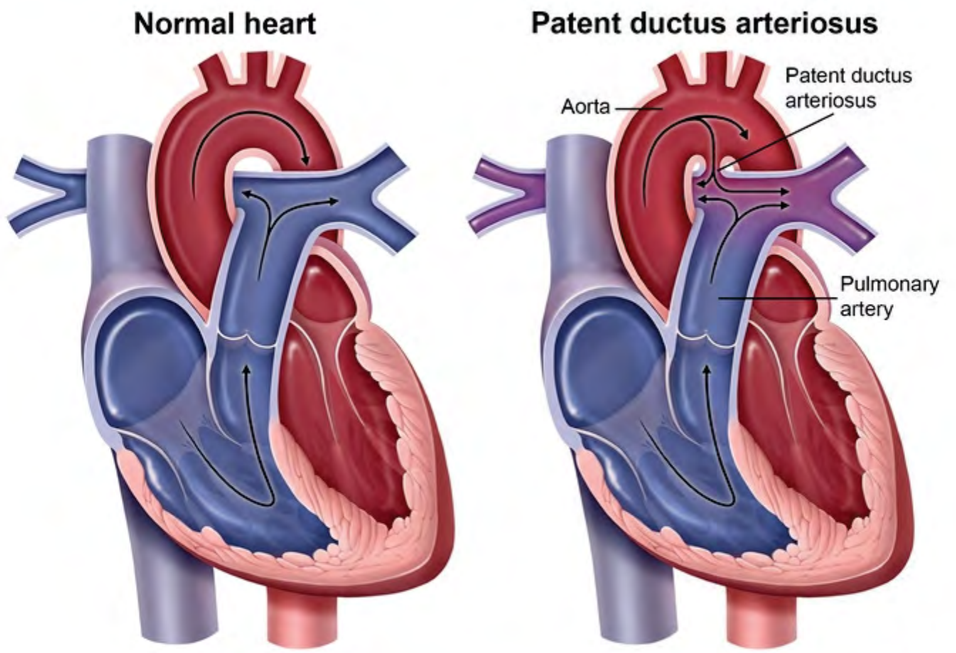

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A 53-year-old man comes to the office due to difficulty breathing and increasing fatigue. He has shortness of breath at night and has been sleeping in a recliner to help relieve his dyspnea. Medical history is significant for hypertension and hyperlipidemia. Two months ago, the patient suffered a myocardial infarction that was not revascularized due to a delay in seeking treatment. He quit smoking afterward, but had previously smoked a pack of cigarettes daily for 25 years. On cardiac auscultation, a low-frequency diastolic heart sound is heard shortly after the second heart sound when the patient lies in the left lateral decubitus position. Cardiac imaging shows hypokinesis of the left ventricular free wall. Which of the following is most likely to accentuate this patient's abnormal auscultation finding?

- A. Amyl nitrite inhalation
- B. Furosemide injection
- C. Having the patient stand up
- D. Listening at end expiration
- E. Valsalva strain phase

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- ✗ A. Amyl nitrite inhalation (9%)
- B. Furosemide injection (4%)
- C. Having the patient stand up (12%)
- ✓ D. Listening at end expiration (37%)
- E. Valsalva strain phase (36%)

Incorrect
Correct answer
D

37%
Answered correctly

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Explanation

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation

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Explanation

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive diastolic filling	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation
S4	• Heard just before S1 • Caused by blood striking a stiff LV wall during atrial contraction	• Concentric LV hypertrophy • Restrictive cardiomyopathy • Acute myocardial infarction
*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70). **EF** = ejection fraction; **LV** = left ventricular.		

This patient has decompensated heart failure (fatigue, dyspnea) with an audible **S3**, a low-frequency sound that occurs during early diastole, just **after S2**. An S3 is likely caused by reverberation of sound within the ventricular cavity during rapid **passive ventricular filling**.

S3 can be a normal finding in healthy children and young adults; however, in patients age >40 it is considered abnormal and suggests **ventricular volume overload** or enlargement. It is often heard with aortic or mitral regurgitation or heart failure with reduced ejection fraction (eg, dilated cardiomyopathy, ischemic heart disease). In this patient's case, imaging is consistent with left ventricular systolic dysfunction secondary to the patient's recent myocardial infarction.

An S3, and the other commonly heard **left ventricular gallop**, S4 (caused by blood striking a stiff ventricular wall during atrial contraction at the end of diastole), are best heard with the bell of the stethoscope over the **cardiac apex** while the patient is in the left lateral decubitus position. Listening at **end expiration** makes the sound even more audible by decreasing lung volume and bringing the heart closer to the chest wall.

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S4	• Heard just before S1 • Caused by blood striking a stiff LV wall during atrial contraction	• Concentric LV hypertrophy • Restrictive cardiomyopathy • Acute myocardial infarction

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(Choices A, B, C, and E) Medications and maneuvers that decrease venous return to the heart reduce ventricular blood volume and are likely to *lessen* the intensity of an S3. Amyl nitrate inhalation causes dilation of venous capacitance veins to decrease venous return, furosemide injection causes brisk diuresis to reduce intravascular blood volume, standing causes blood to pool in the legs to reduce venous return, and Valsalva strain phase increases intrathoracic pressure to compress the vena cava and reduce venous return.

Educational objective:
An S3 is a low-frequency sound occurring during early diastole, just after S2. Left ventricular gallops (S3 and S4) are best heard with the bell of the stethoscope over the cardiac apex while the patient is in the left lateral decubitus position at end expiration.

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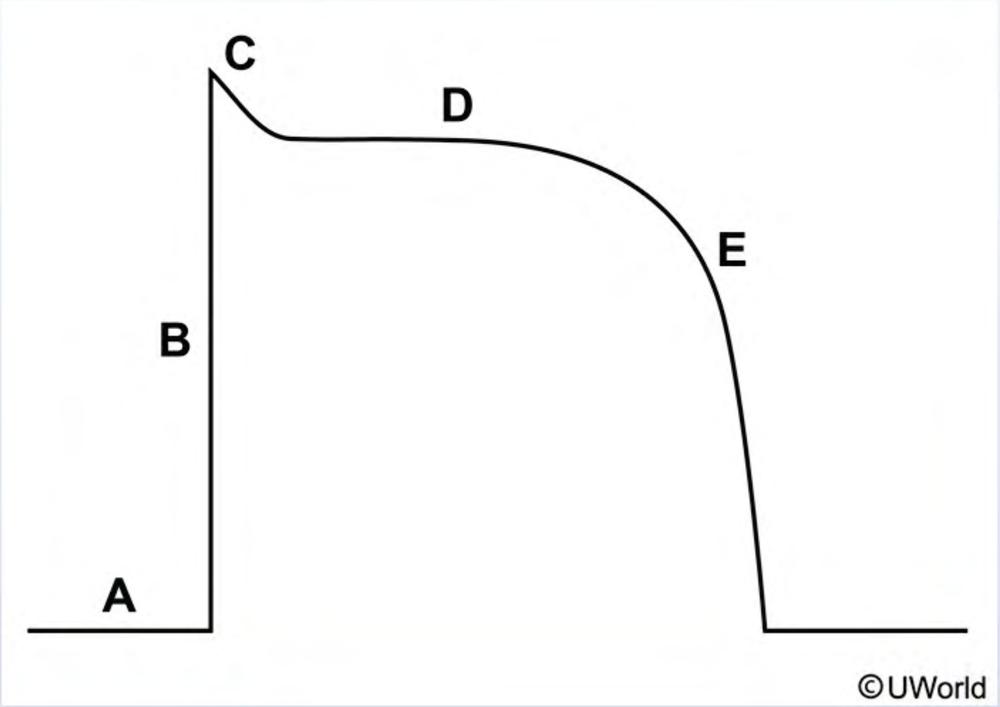
A 45-year-old man comes to the clinic due to recurrent palpitations accompanied by chest discomfort and shortness of breath. A year ago, he was diagnosed with paroxysmal atrial fibrillation treated with rate control using a beta blocker. Past medical history is also significant for hypertension and obesity. Echocardiogram shows left atrial enlargement, normal left ventricular ejection fraction, and no significant valvular disease. 24-hour Holter monitoring reveals bursts of atrial fibrillation associated with the patient's symptoms. He is initiated on dofetilide to maintain normal sinus rhythm. This medication exerts its main effect on which portion of the action potential curve?



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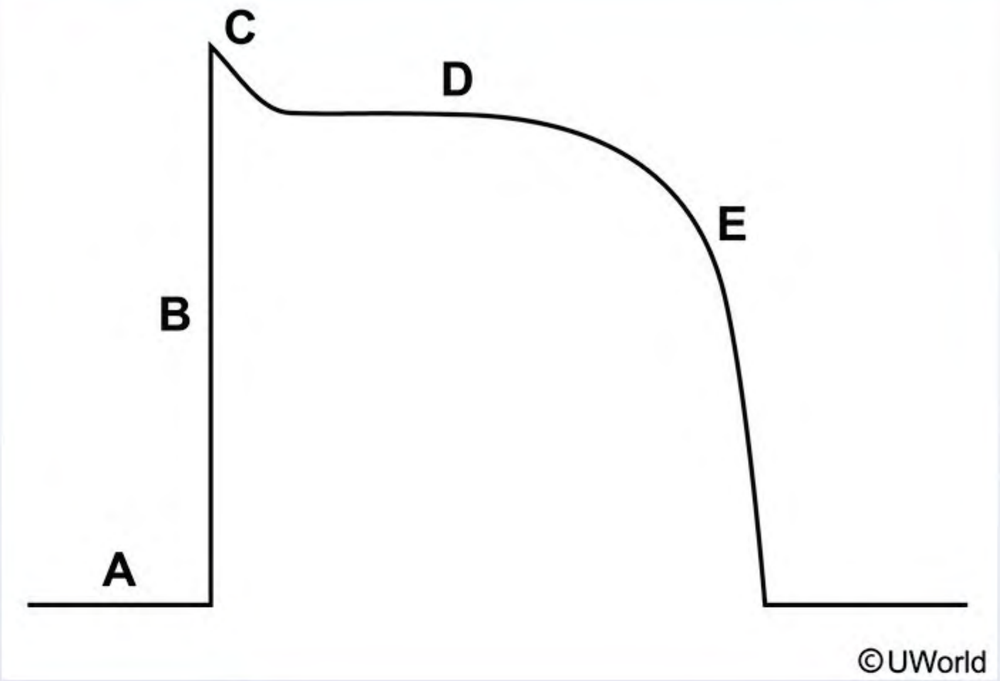

- A. A
- B. B
- C. C
- D. D
- E. E

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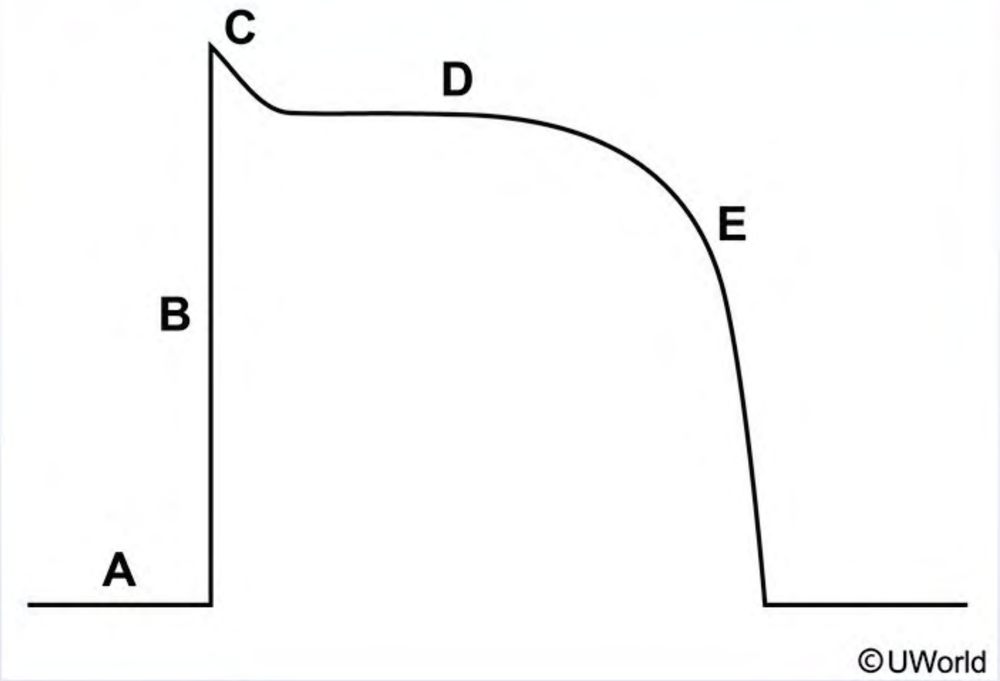

- A. A (3%)
- ✗ B. B (18%)
- C. C (2%)
- D. D (14%)
- ✓ E. E (61%)

Incorrect
Correct answer
E

61%
Answered correctly

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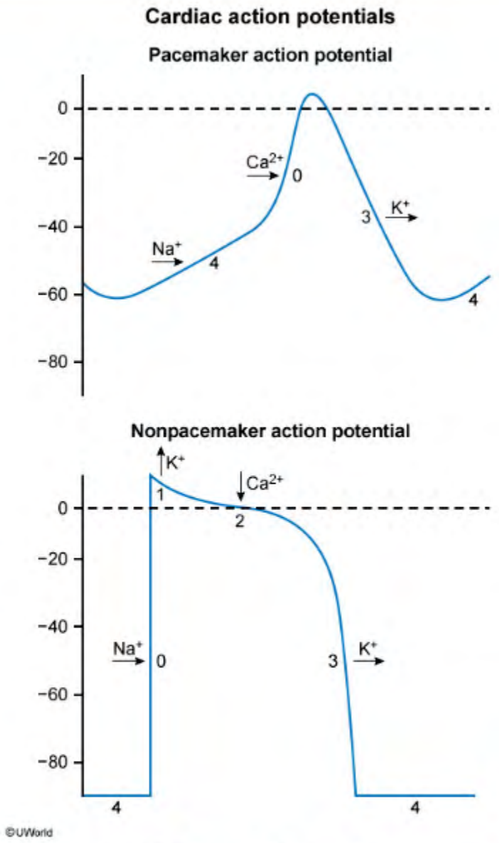

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The typical myocardial action potential of "nonpacemaker" or cardiac myocytes includes several phases:

1. **Phase 0 (rapid depolarization)** – occurs when the resting membrane potential reaches threshold; it is characterized by rapid sodium entry into the cell due to activation and opening of voltage-dependent sodium channels. Class I antiarrhythmic drugs (eg, procainamide, flecainide, lidocaine) block these sodium channels and inhibit the initial depolarization phase (phase 0) of the action potential.
2. **Phase 1 (early repolarization)** – caused primarily by activation of transient outward potassium currents along with a rapid decline in inward sodium current. This phase is not modulated to an appreciable extent by antiarrhythmic medications.
3. **Phase 2 (plateau phase)** – Stable membrane potential due to a balance of "late" inward calcium current and outward potassium current. Calcium channel blockers (verapamil, diltiazem) are class IV antiarrhythmic drugs that inhibit the L-type calcium channels during this phase. These medications slow the sinus rate, prolong conduction through the atrioventricular node, and depress myocardial contractility (negative inotropic effect).
4. **Phase 3 (late repolarization)** – characterized by the opening of **potassium channels** and the efflux of potassium ions from the cell (delayed rectifier potassium currents IKr and IKs) in conjunction with the decay of inward calcium current. **Class III** antiarrhythmic drugs (**amiodarone, sotalol, dofetilide**) predominantly block potassium channels and inhibit the outward potassium current during this phase, leading to prolongation of repolarization, action potential duration, and the QT interval on ECG. These medications are often used for **maintenance of sinus rhythm** in patients with **paroxysmal atrial fibrillation**.
5. **Phase 4 (recovery)** – normal resting membrane potential is between −80 and −95 mV and is generated by open potassium channels (inward rectifier current). Antiarrhythmic drugs do not affect phase 4 of the myocyte action potential, but beta blockers and calcium channel blockers can slow diastolic depolarization (phases 4 and 0) in pacemaker cells by decreasing the slope of depolarization.

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Educational objective:
Class III antiarrhythmic drugs (amiodarone, sotalol, dofetilide) predominantly block potassium channels and inhibit the outward potassium currents during phase 3 of the cardiac action potential, thereby prolonging repolarization and total action potential duration.

Pharmacology	Cardiovascular System	Antiarrhythmic drugs
Subject	System	Topic

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A 38-year-old woman comes to the office due to worsening shortness of breath over the past 4 weeks. She has no chest pain or lightheadedness but has noticed some ankle puffiness. The patient can hardly do chores without having to stop to catch her breath. Medical history is unremarkable. She delivered a healthy child 2 months ago without complications. The patient is a lifetime nonsmoker and does not use alcohol. She has no family history of early heart disease or sudden cardiac death. Blood pressure is 96/60 mm Hg and pulse is 92/min and regular. The apical impulse is palpated along the anterior axillary line, and S3 is heard at the apex. There are no heart murmurs. Which of the following best describes the left ventricular changes in this patient?

	Ejection fraction	Compliance
A.	Decreased	Increased
B.	Decreased	Normal
C.	Normal	Decreased
D.	Normal	Increased
E.	Normal	Normal

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	Ejection fraction	Compliance	
✓ A.	Decreased	Increased	(57%)
B.	Decreased	Normal	(18%)
C.	Normal	Decreased	(13%)
D.	Normal	Increased	(8%)
E.	Normal	Normal	(2%)

Correct

57% Answered correctly

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Explanation

Eccentric left ventricular enlargement & heart failure

LV systolic dysfunction (eg, ischemia, cardiomyopathy)

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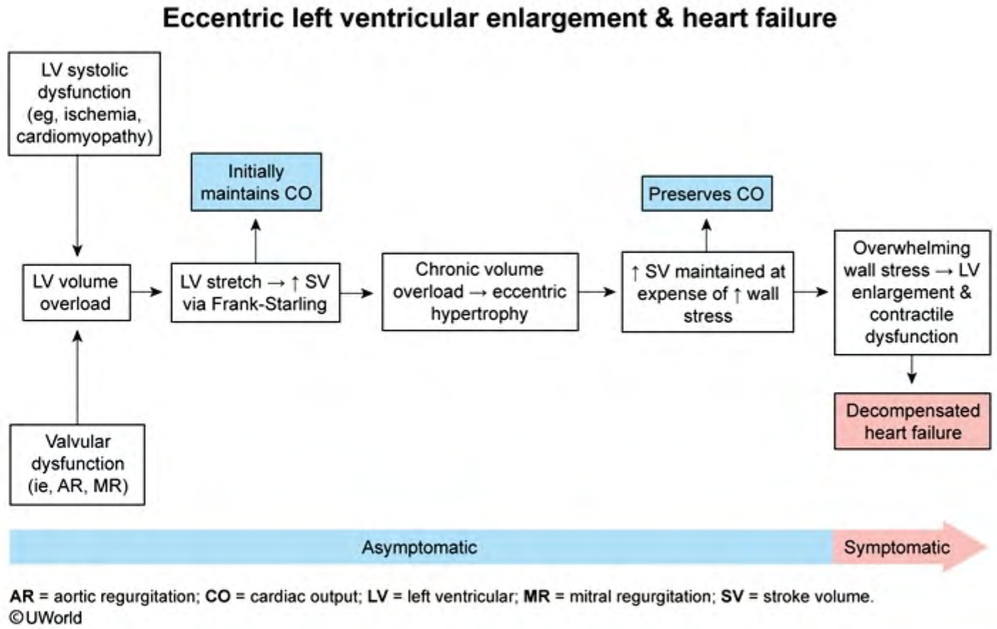

AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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Expl

(eg, ischemia, cardiomyopathy)

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This patient with progressive dyspnea and peripheral edema, as well as a laterally displaced point of maximal impulse and S3 on physical examination, likely has **heart failure** due to **peripartum cardiomyopathy**. Peripartum cardiomyopathy is relatively uncommon and manifests as a **dilated cardiomyopathy** that occurs during the last month of pregnancy or within 5 months after delivery. The pathogenesis of the condition is poorly understood, but it may be related to **impaired function of angiogenic growth factors** (eg, vascular endothelial growth factor) during the peripartum period. In addition, certain individuals may have genetic mutations affecting cardiac structural proteins that predispose to the development of peripartum cardiomyopathy.

In dilated cardiomyopathy, myocardial dysfunction causes an increase in left ventricular end-diastolic volume, which is compensated for by eccentric hypertrophy to maintain cardiac output. The dilation of the left ventricle allows for **increased compliance** to accommodate the increase in end-diastolic volume. Eventually, however, overwhelming wall stress leads to left ventricular failure with **reduced ejection fraction** and symptomatic heart failure.

(Choice B) Normal compliance with decreased ejection fraction may occur in neurogenic shock due to impaired sympathetic stimulation resulting in decreased myocardial contractility.

(Choice C) Decreased compliance with normal (or increased) ejection fraction (diastolic heart failure) is seen with left ventricular hypertrophy due to long-standing hypertension.

(Choice D) Increased compliance with relatively normal ejection fraction occurs during the compensated period of eccentric hypertrophy, before overwhelming wall stress leads to systolic failure.

(Choice E) Normal compliance and ejection fraction describes a heart that is without underlying pathology.

Educational objective:
Peripartum cardiomyopathy is a relatively uncommon cause of dilated cardiomyopathy that may be related to impaired function of angiogenic growth factors. Dilated cardiomyopathy involves compensatory eccentric hypertrophy, which increases ventricular compliance and also allows for temporary maintenance of cardiac output.

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(Choice E) Normal compliance and ejection fraction describes a heart that is without underlying pathology.

Educational objective:
Peripartum cardiomyopathy is a relatively uncommon cause of dilated cardiomyopathy that may be related to impaired function of angiogenic growth factors. Dilated cardiomyopathy involves compensatory eccentric hypertrophy, which increases ventricular compliance and also allows for temporary maintenance of cardiac output. Over time, overwhelming wall stress leads to left ventricular failure with reduced ejection fraction and symptomatic heart failure.

References

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hypertrophy, which increases ventricular compliance and also allows for temporary maintenance of cardiac output.

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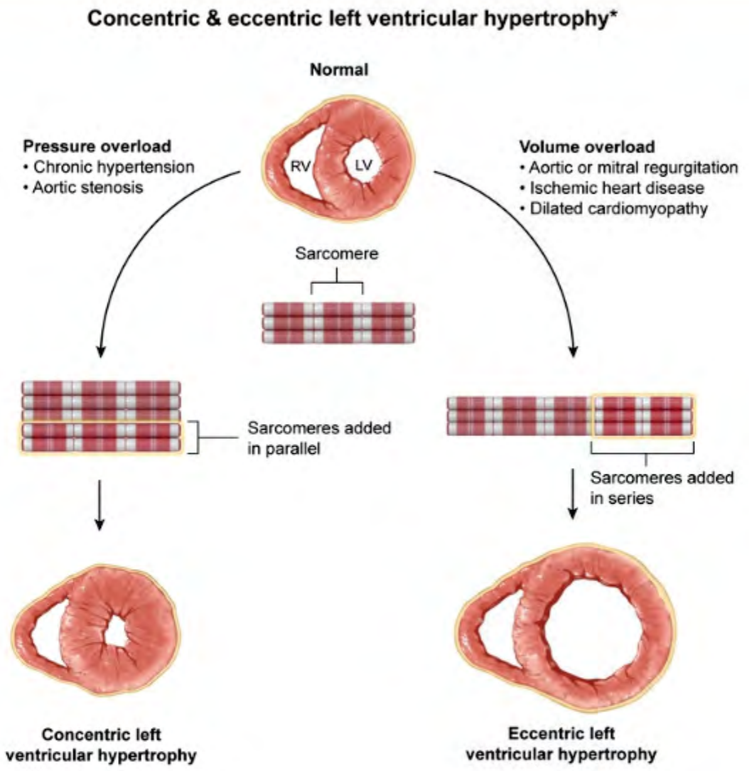

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A 64-year-old man dies suddenly while playing tennis. In the preceding months, the patient experienced fatigue and some exertional dyspnea. Autopsy reveals rupture of an unsuspected ascending aortic aneurysm. Heart examination shows a septal thickness of 1.1 cm (normal: ≤1.1), a posterior wall thickness of 1.1 cm (normal: ≤1.1), and an internal left ventricular diameter of 6.8 cm (normal: 3.5-5.9). No focal myocardial scarring is seen. Which of the following is the most likely cause of the cardiac findings seen in this individual?

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Hypertrophic cardiomyopathy
- D. Obstructive coronary artery disease
- E. Restrictive cardiomyopathy

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- ✓ A. Aortic regurgitation (68%)
- B. Aortic stenosis (16%)
- C. Hypertrophic cardiomyopathy (10%)
- D. Obstructive coronary artery disease (2%)
- E. Restrictive cardiomyopathy (2%)

Correct | 68% Answered correctly | 03 secs Time Spent | 2023 Version

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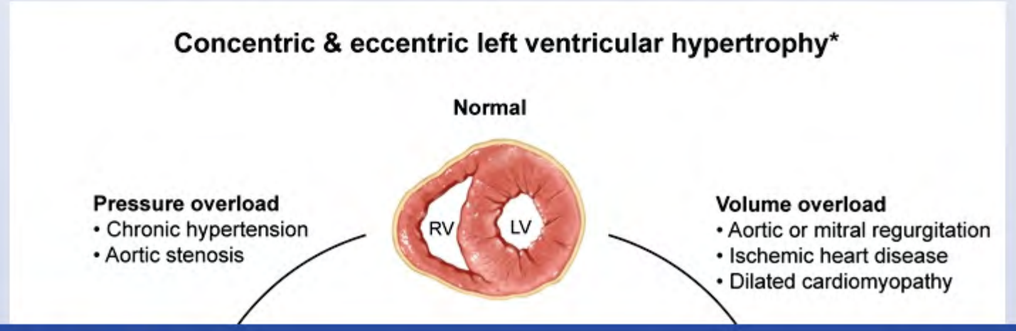

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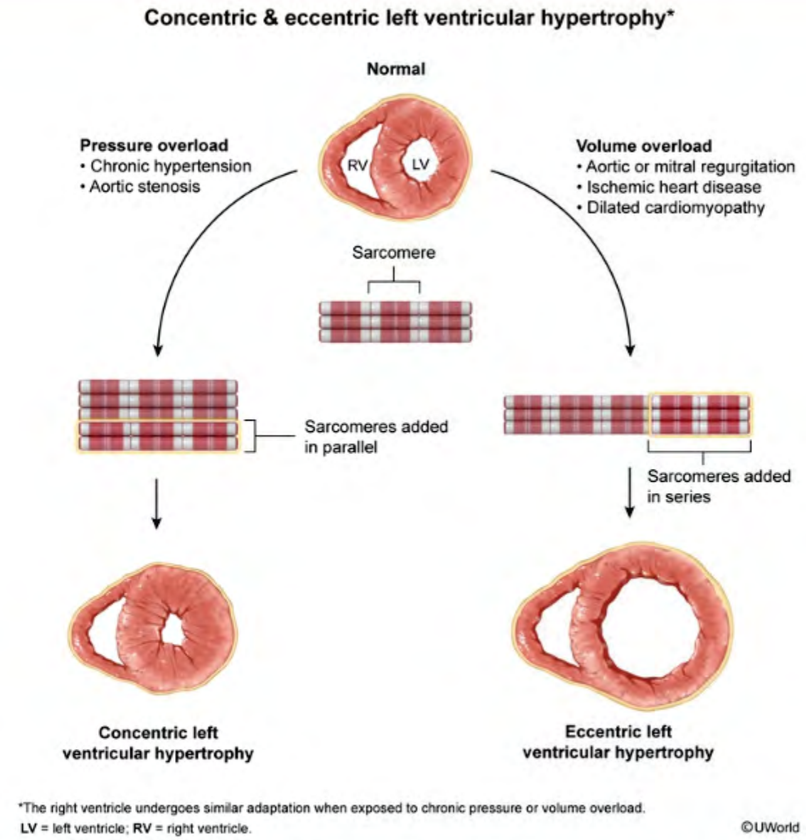

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• Dilated cardiomyopathy

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The right ventricle undergoes similar adaptation when exposed to chronic pressure or volume overload.
LV = left ventricle; **RV** = right ventricle.

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This individual's autopsy reveals a **dilated left ventricular (LV) cavity** with relatively thin ventricular walls, a finding consistent with **eccentric hypertrophy**. Eccentric hypertrophy results from the addition of myocardial contractile fibers in series in response to chronic **volume overload**. The adaptation allows the left ventricle to increase stroke volume and maintain cardiac output; however, LV wall stress increases, resulting in eventual decompensation and the development of heart failure. Common causes of eccentric hypertrophy include cardiomyopathy, ischemic heart disease, and chronic aortic or mitral valve regurgitation.

In developed countries, **aortic root dilation** is one of the most common causes of **chronic aortic regurgitation**. This individual with an ascending aortic aneurysm likely had aortic root dilation with consequent chronic aortic regurgitation, leading to LV volume overload and eccentric hypertrophy.

(Choice B) Aortic stenosis leads to pressure overload of the left ventricle and the development of concentric hypertrophy (thickening of the LV walls via the addition of myocardial contractile fibers in parallel). However, the ventricular cavity size is typically normal or reduced (not increased).

(Choice C) Hypertrophic cardiomyopathy is an inherited cardiomyopathy that involves localized thickening of the LV septal wall and resulting LV outflow tract obstruction. Although sudden cardiac death can occur (mostly in young athletes), the uniform thickness of this patient's ventricular walls and enlarged chamber size is more consistent with eccentric hypertrophy due to aortic regurgitation.

(Choice D) Obstructive coronary artery disease can lead to eccentric hypertrophy via myocardial contractile dysfunction and consequent LV volume overload (ie, ischemic heart disease). However, chronic aortic regurgitation is more likely in this individual with ascending aortic aneurysm and no evidence of myocardial scarring (scarring would suggest previous myocardial infarction).

(Choice E) Restrictive cardiomyopathy involves no change or a slight decrease, rather than an increase, in the LV cavity size.

Educational objective:

Eccentric ventricular hypertrophy results in a dilated cavity with relatively thin ventricular walls due to the addition

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contractile fibers in series in response to chronic **volume overload**. The adaptation allows the left ventricle to increase stroke volume and maintain cardiac output; however, LV wall stress increases, resulting in eventual decompensation and the development of heart failure. Common causes of eccentric hypertrophy include cardiomyopathy, ischemic heart disease, and chronic aortic or mitral valve regurgitation.

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Educational objective:
Eccentric ventricular hypertrophy results in a dilated cavity with relatively thin ventricular walls due to the addition of myocardial contractile fibers in series in response to chronic volume overload. Chronic aortic regurgitation can result from aortic root dilation and is a common cause of eccentric hypertrophy.

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contractile fibers in series in response to chronic **volume overload**. The adaptation allows the left ventricle to increa… decom… cardio…

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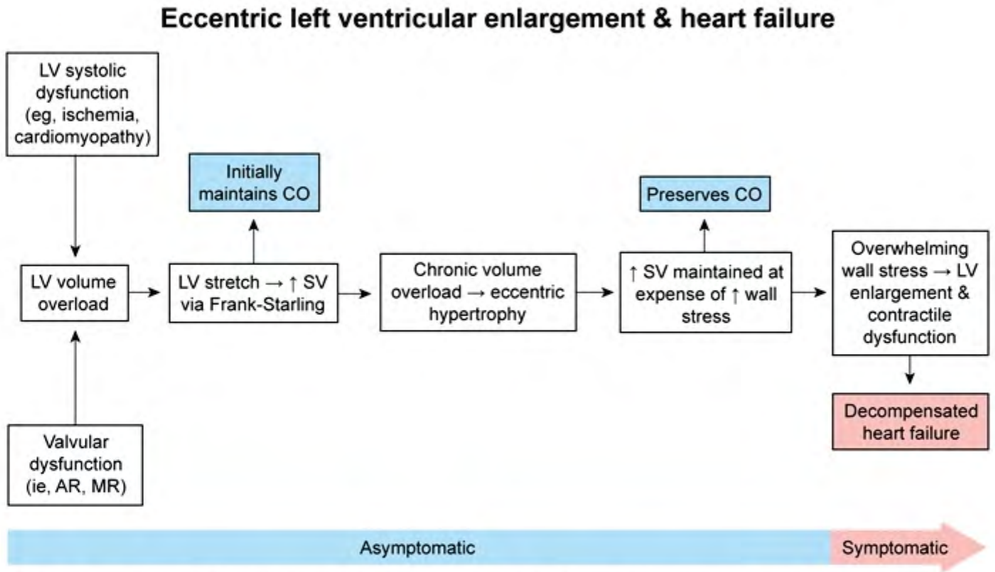

AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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A 6-month-old girl is brought to the office for her first visit after being adopted from another country a week ago. The parents note that she often seems to breathe hard and fast, particularly after feeding or crying. Her lips also sometimes turn blue. On examination, height and weight are at the 15th and 2nd percentiles, respectively. The patient has a 4/6, harsh systolic, crescendo-decrescendo murmur best heard at the left upper sternal border. The results of a complete blood count are as follows:

Hematocrit	52%
Platelets	240,000/mm^3
Leukocytes	11,000/mm^3

Which of the following processes is most likely contributing to this patient's elevated hematocrit?

- A. Decreased pulmonary dead space
- B. Impaired postnatal lung expansion
- C. Increased blood flow to the renal arteries
- D. Monoclonal proliferation of erythroid cells
- E. Reduced pulmonary arterial blood flow

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Hematocrit	52%
Platelets	240,000/mm^3
Leukocytes	11,000/mm^3

Which of the following processes is most likely contributing to this patient's elevated hematocrit?

- ✗ A. Decreased pulmonary dead space (1%)
- B. Impaired postnatal lung expansion (3%)
- C. Increased blood flow to the renal arteries (2%)
- D. Monoclonal proliferation of erythroid cells (15%)
- ✓ E. Reduced pulmonary arterial blood flow (75%)

Incorrect
Correct answer
E

75%
Answered correctly

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Explanation

Normal heart	Tetralogy of Fallot

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D. Monoclonal proliferation of erythroid cells (15%)

✓ E. Reduced pulmonary arterial blood flow (75%)

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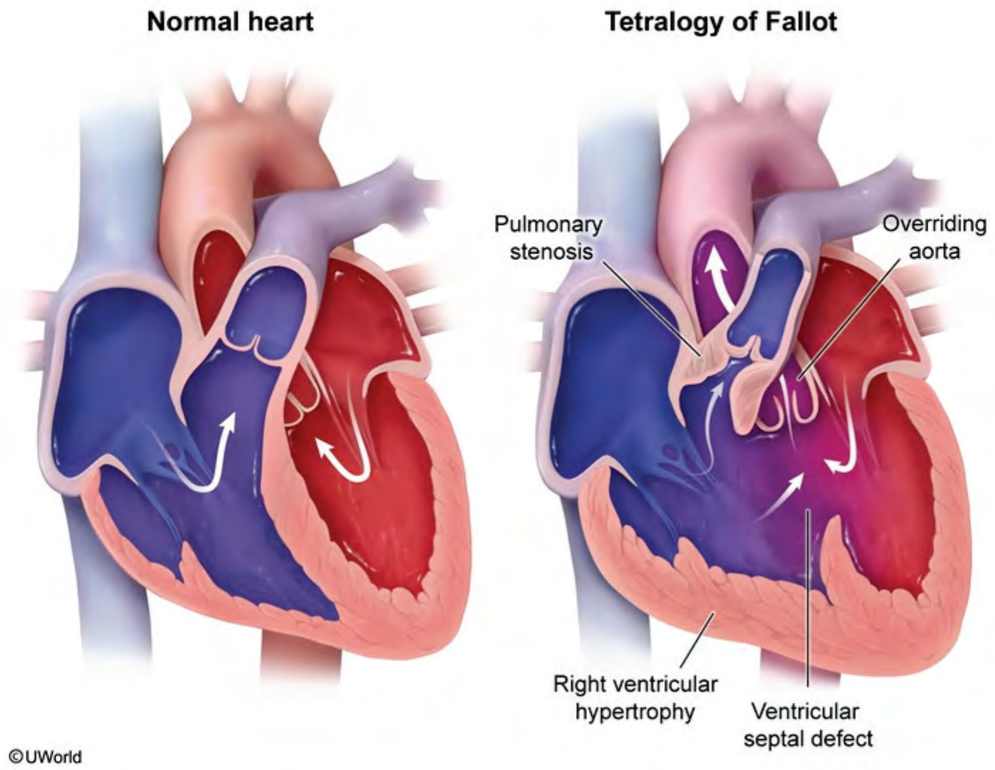

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This patient with **episodic cyanosis** and respiratory difficulty and a crescendo-decrescendo systolic murmur best heard at the left sternal border (suggestive of right ventricular outflow tract [RVOT] obstruction [ie, pulmonic stenosis]) most likely has **tetralogy of Fallot** (ToF).

In ToF, RVOT obstruction leads to right-to-left shunting through the large ventricular septal defect, with resulting **underperfusion of the pulmonary arteries** and hypoxemia. Because the degree of RVOT obstruction is dynamic (eg, can worsen during infundibular muscle spasm), patients can experience periodic episodes of worsening hypoxemia and cyanosis often triggered by feeding or crying (ie, "tet" episodes). **Poor weight gain** is common and likely related to reduced caloric intake and/or higher metabolic needs.

In response to **chronic hypoxemia**, the body increases red blood cell (RBC) production to increase oxygen-carrying capacity. Tissue hypoxia in the inner cortex and medulla of the kidney triggers **increased** renal production of **erythropoietin**, which then stimulates erythrocyte precursor cells in the bone marrow to produce more RBCs, resulting in an increased hemoglobin concentration and hematocrit. This physiologic type of **secondary polycythemia** is the most common overall mechanism of polycythemia.

(Choice A) Pulmonary dead space refers to portions of the lung that are ventilated but not perfused. Decreased pulmonary dead space tends to increase alveolar air exchange and would not contribute to development of hypoxemia or secondary polycythemia.

(Choice B) Impaired postnatal lung expansion (eg, pulmonary hypoplasia, diaphragmatic hernia), typically causes acute hypoxemia in the immediate newborn period. This patient's pathologic murmur makes cardiac pathology more likely.

(Choice C) Decreased, not increased, renal blood flow (as in renal artery stenosis) can sometimes cause secondary polycythemia due to reduced oxygen delivery to the renal interstitium.

(Choice D) Monoclonal proliferation of erythroid cells (ie, originating from a single mutant cell) is seen in polycythemia vera, a form of blood cancer that involves autonomous RBC production independent of erythropoietin. It is the most common cause of primary polycythemia, which is usually seen in adults and often accompanied by thrombocytosis and leukocytosis (not seen in this patient). In contrast, secondary polycythemia

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hypoxemia and cyanosis often triggered by feeding or crying (ie, "tet" episodes). **Poor weight gain** is common and likely related to reduced caloric intake and/or higher metabolic needs.

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Educational objective:
Tetralogy of Fallot can present in infancy with episodic cyanosis and poor weight gain. Right ventricular outflow tract obstruction (reduced perfusion of the pulmonary arteries) results in chronic hypoxemia, leading to secondary polycythemia.

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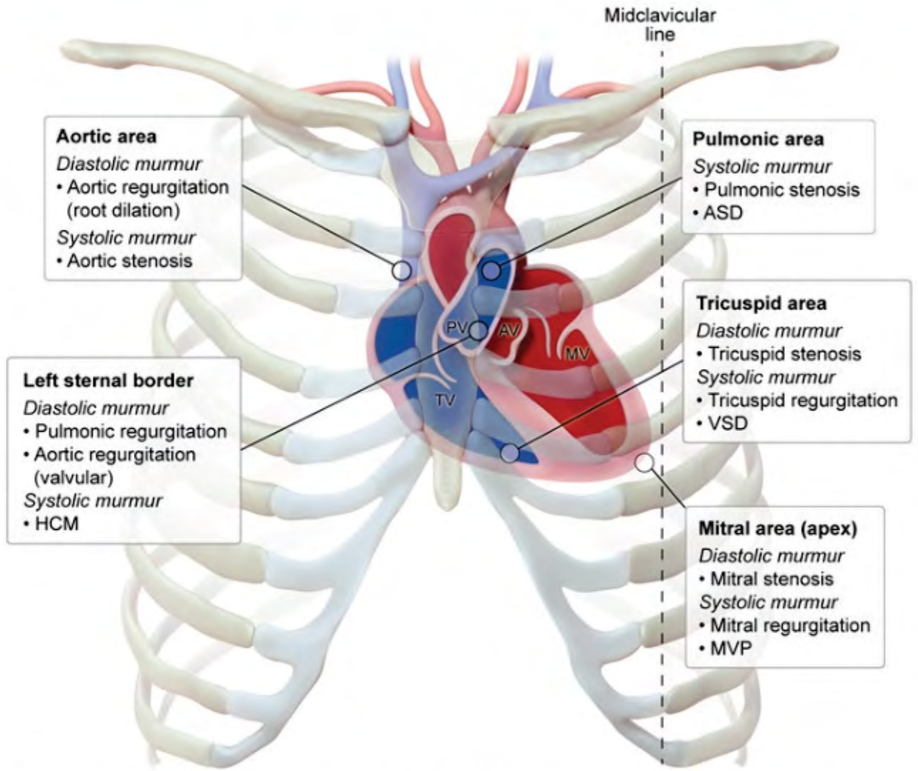

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Right ventricular

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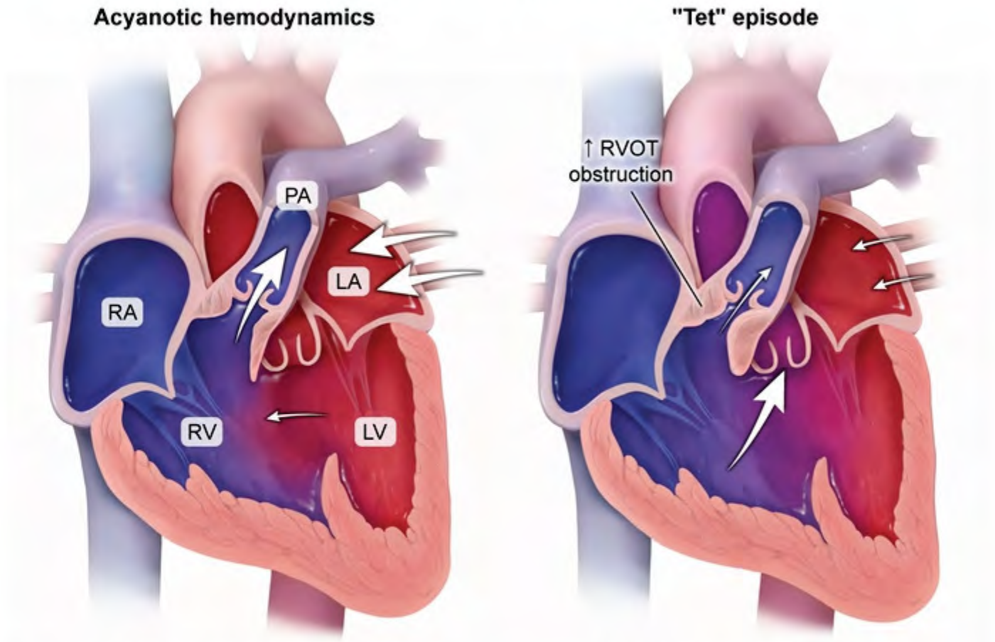
Pressure and flow changes during "tet" episode



Moderate flow through RVOT allows:
- Adequate pulmonary blood flow
- L-to-R or neutral shunting through VSD

↑ RVOT obstruction leads to:
- ↑ RV pressure
- ↓ PA & LA pressures
- R-to-L shunting through VSD

LA = left atrium; **L-to-R** = left to right; **LV** = left ventricle; **PA** = pulmonary artery; **RA** = right atrium; **R-to-L** = right to left; **RV** = right ventricle; **RVOT** = right ventricular outflow tract; **VSD** = ventricular septal defect.

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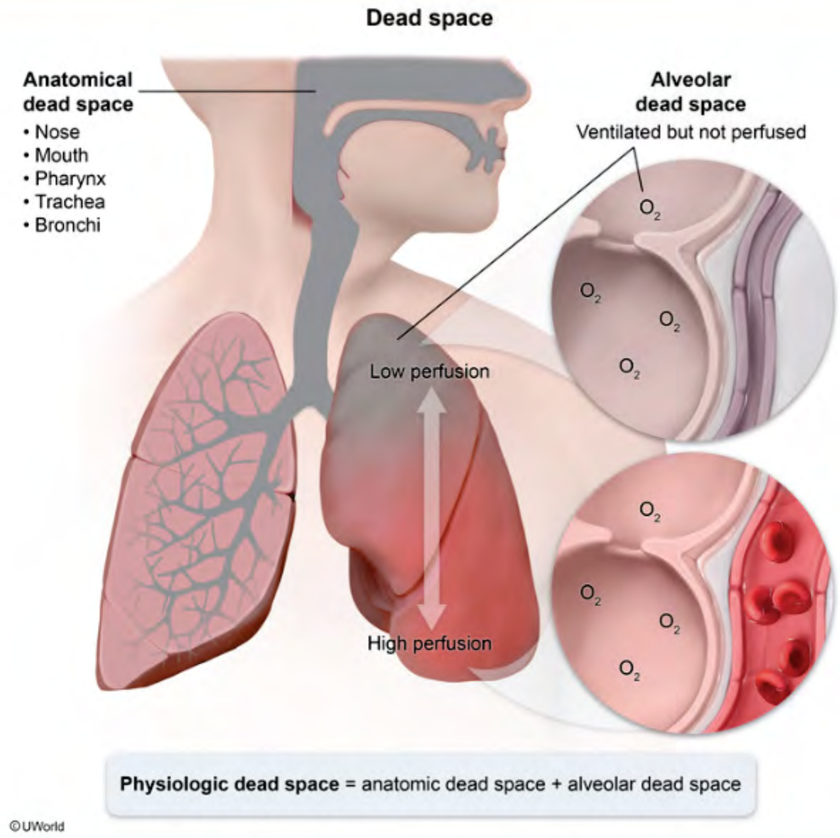

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A 44-year-old man is evaluated in the clinic for occasional chest discomfort that is not consistently related to exertion. The patient's past medical history is significant for hypertension and hyperlipidemia. His grandfather experienced a myocardial infarction at age 50. Coronary CT angiography reveals several nonobstructive atherosclerotic plaques in the coronary arteries. One plaque in the proximal left anterior descending artery appears extensive, has a large hypodense core, and occupies 40% of the lumen. No intervention is performed. One year later, the patient comes to the emergency department with acute severe chest pain and is found to have thrombotic occlusion of the proximal left anterior descending artery. High intraplaque activity of which of the following enzymes most likely resulted in this patient's myocardial infarction?

- A. Hydroxymethylglutaryl CoA reductase
- B. Lysyl oxidase
- C. Metalloproteinases
- D. Procollagen peptidases
- E. Prolyl hydroxylase

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- A. Hydroxymethylglutaryl CoA reductase (16%)
- ✗ B. Lysyl oxidase (10%)
- ✓ C. Metalloproteinases (59%)
- D. Procollagen peptidases (9%)
- E. Prolyl hydroxylase (4%)

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Correct answer
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59%
Answered correctly

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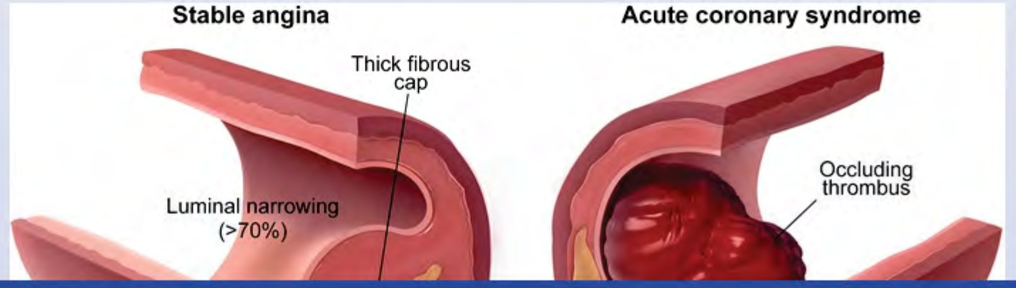

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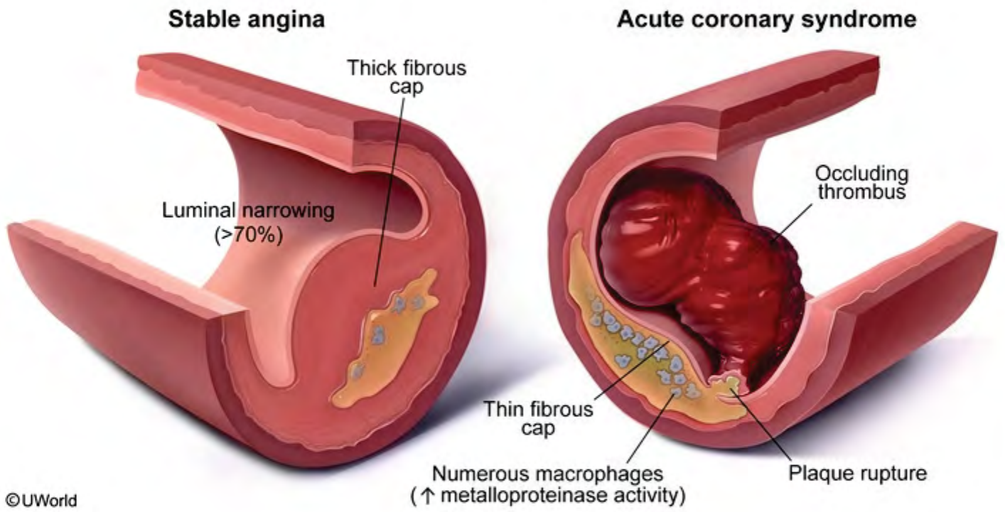

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(↑ metalloproteinase activity)

This patient with coronary atherosclerosis has likely developed plaque rupture with thrombotic occlusion of the left anterior descending artery, resulting in acute coronary syndrome (unstable angina, myocardial infarction).

Patients with coronary atherosclerosis can be asymptomatic but typically develop symptoms of stable angina (exertional chest pain relieved with rest/nitroglycerin) if progressive enlargement of the atherosclerotic plaque causes >70% luminal stenosis. **Acute coronary syndrome** usually occurs due to **plaque rupture**, which leads to superimposed **thrombosis** and vessel occlusion. The likelihood of plaque rupture is typically related to plaque stability rather than plaque size or degree of luminal narrowing. Plaque stability largely depends on the mechanical strength of the fibrous cap: Thin-cap fibroatheromas are generally unstable and more vulnerable to rupture.

During the chronic inflammatory progression of an atheroma, the fibrous cap is continually being remodeled. The balance of collagen synthesis and degradation determines the mechanical strength of the cap. **Thin-cap fibroatheromas** are characterized by a large necrotic core covered by a thin fibrous cap. Activated macrophages infiltrating the atheroma contribute to the breakdown of extracellular matrix proteins (eg, collagen) by secreting **metalloproteinases**. Ongoing intimal inflammation can destabilize the mechanical integrity of the plaque through release of these metalloproteinases, resulting in plaque rupture and consequent acute coronary syndrome.

(Choice A) Hydroxymethylglutaryl (HMG) CoA reductase is the rate-limiting enzyme in cholesterol synthesis and is mostly expressed in the liver, intestine, adrenal cortex, and reproductive tissues. Patients with hyperlipidemia are typically treated with statins (eg, atorvastatin), which competitively inhibit HMG CoA reductase.

(Choice B) Lysyl oxidase strengthens extracellular collagen fibers by mediating cross-link formation between lysine and hydroxylysine residues (requires copper). Therefore, a high activity of lysyl oxidase would favor atheroma stabilization by strengthening collagen in the fibrous cap.

(Choice D) Procollagen peptidase cleaves the terminal regions of procollagen molecules exocytosed by fibroblasts or smooth muscle cells, transforming procollagen into insoluble tropocollagen. Tropocollagen then aggregates to form collagen fibrils.

(Choice E) Prolyl hydroxylase is responsible for the hydroxylation of proline on procollagen chains (requires vitamin C), which is a necessary step in the formation of a stable collagen triple helix.

Educational objective:

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(Choice E) Prolyl hydroxylase is responsible for the hydroxylation of proline on procollagen chains (requires vitamin C), which is a necessary step in the formation of a stable collagen triple helix.

Educational objective:
The likelihood of plaque rupture is related to plaque stability rather than plaque size or the degree of luminal narrowing. Plaque stability largely depends on the mechanical strength of the fibrous cap. Inflammatory macrophages in the intima may reduce plaque stability by secreting metalloproteinases, which degrade extracellular matrix proteins (eg, collagen).

References

- Evidence for increased collagenolysis by interstitial collagenases-1 and -3 in vulnerable human atheromatous

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A 4-year-old boy is brought in by his parents for evaluation of a fever that has persisted for the past 5 days. He has also been more irritable than usual and had 2 or 3 episodes of vomiting. The patient has no prior medical problems and takes no medications. He has received all recommended vaccinations. He traveled to China last year to visit his grandparents and cousins but has not traveled outside of the country this year. Temperature is 38.9 C (102 F). Physical examination shows bilateral conjunctival injection with no exudates. His tongue is bright red and lips are cracked. Nonpitting edema is present on his hands and feet. Which of the following complications is this patient at greatest risk for developing?

- A. Aortic dissection
- B. Coronary artery aneurysm
- C. Monocular blindness
- D. Mononeuritis multiplex
- E. Proliferative glomerulonephritis
- F. Pulmonary cavitation

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- A. Aortic dissection (4%)
- ✓ B. Coronary artery aneurysm (70%)
- C. Monocular blindness (3%)
- D. Mononeuritis multiplex (3%)
- E. Proliferative glomerulonephritis (16%)
- F. Pulmonary cavitation (1%)

Correct

70%
Answered correctly

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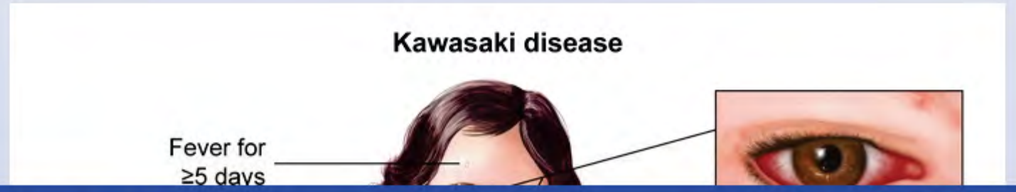
Explanation



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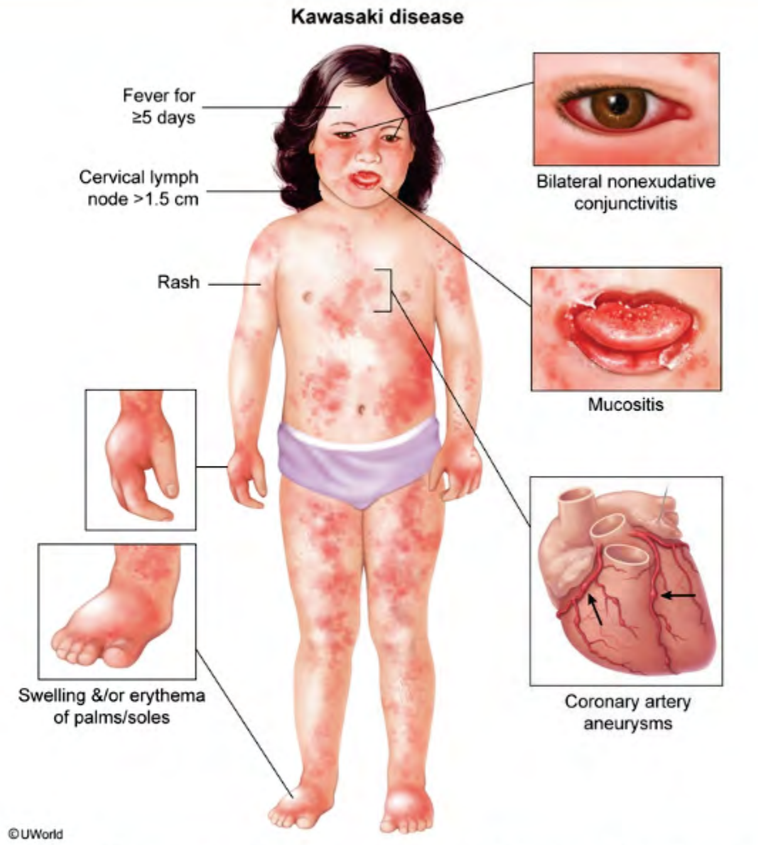

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Fever for ≥5 days

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Kawasaki disease is a vasculitis of **medium-sized arteries** that affects young children (usually age <5 years). It occurs most commonly in patients of **Asian** ethnicity. The diagnosis of Kawasaki disease is based on a **fever for ≥5 days** plus 4 of the following findings:

1. Bilateral non-exudative conjunctival injection (erythema)
2. Cervical lymphadenopathy
3. Mucositis: Erythema of the palatine mucosa, fissured erythematous lips, "strawberry" tongue
4. Extremity changes: Edema of hands and feet, erythema of palms and soles, desquamation of the fingertips (periungual)
5. Rash: Polymorphous (usually urticarial) erythematous rash on the extremities that spreads centripetally to the trunk

A serious complication of Kawasaki disease is coronary artery inflammation leading to the development of **coronary artery aneurysms**.

(Choice A) Dissecting aneurysms of the aorta are associated with long-standing hypertension and Marfan syndrome.

(Choice C) Monocular blindness is a complication of temporal arteritis.

(Choice D) Mononeuritis multiplex is a peripheral neuropathy of multiple individual nerves. It is associated with a variety of diseases, including granulomatosis with polyangiitis (Wegener's) and polyarteritis nodosa.

(Choice E) Acute proliferative glomerulonephritis can occur after group A streptococcal skin/throat infections. Although symptoms such as fever, "strawberry" tongue, and desquamation can occur in scarlet fever, this patient's bilateral nonexudative conjunctivitis, swelling of the hands and feet, and persistent high-grade fever are more suggestive of Kawasaki disease.

(Choice E) Pulmonary cavities can occur following resolution of pulmonary tuberculosis, histoplasmosis, or

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1. Bilateral non-exudative conjunctival injection (erythema)
2. Cervical lymphadenopathy
3. Mucositis: Erythema of the palatine mucosa, fissured erythematous lips, "strawberry" tongue
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(Choice F) Pulmonary cavities can occur following resolution of pulmonary tuberculosis, histoplasmosis, or sarcoidosis.

Educational objective:
Kawasaki disease is a vasculitis of medium-sized arteries that presents with persistent fever for ≥5 days, bilateral conjunctivitis, cervical lymphadenopathy, and mucocutaneous involvement. Coronary artery aneurysms are a serious complication of Kawasaki disease.

References

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occurs most commonly in patients of **Asian** ethnicity. The diagnosis of Kawasaki disease is based on a **fever for ≥5 day**

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Educational objective:
Kawasaki disease is a vasculitis of medium-sized arteries that presents with persistent fever for >5 days, bilateral

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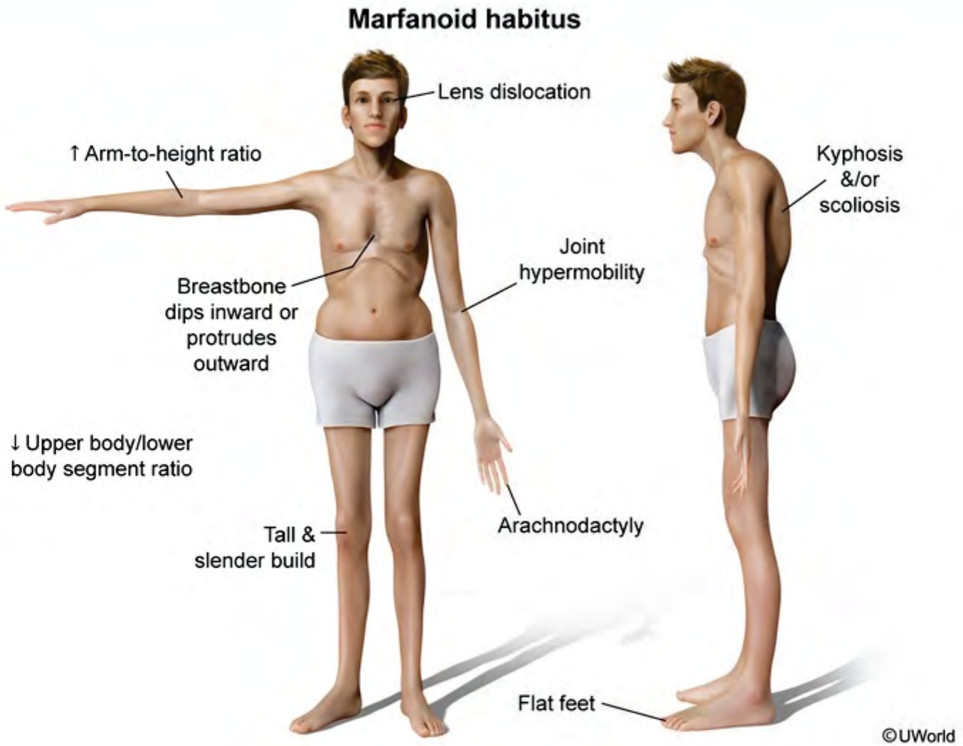

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A 64-year-old man comes to the office for follow-up of hypertension. At the last 2 visits, his blood pressure was persistently elevated. The patient has been treated with lisinopril for the past 5 years, but his other blood pressure medications have been changed several times due to various adverse effects. Medical history is significant for type 2 diabetes mellitus, coronary artery disease with stable angina, and moderately increased albuminuria. Blood pressure is 145/87 mm Hg and pulse is 70/min. BMI is 31 kg/m^2. Physical examination shows trace bilateral lower extremity edema. ECG is unremarkable. Serum laboratory results are as follows:

Sodium	138 mEq/L
Potassium	5.3 mEq/L
Chloride	104 mEq/L
Bicarbonate	24 mEq/L
Calcium	9.5 mg/dL
Creatinine	1.0 mg/dL

Review of the patient's medical records shows similar serum laboratory results 6 months ago. Which of the following medications should be avoided in this patient?

- A. Amlodipine
- B. Bumetanide
- C. Diltiazem
- D. Eplerenone
- E. Furosemide
- F. Metoprolol

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Review of the patient's medical records shows similar serum laboratory results 6 months ago. Which of the following medications should be avoided in this patient?

- ✗ A. Amlodipine (5%)
- B. Bumetanide (1%)
- C. Diltiazem (2%)
- ✓ D. Eplerenone (78%)
- E. Furosemide (8%)
- F. Metoprolol (3%)

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Correct answer
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78%
Answered correctly

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Common medications that cause hyperkalemia	
Medication	**Mechanism**
ACE inhibitor, ARB	Decreases aldosterone secretion (inhibition of AT II/AT II receptor) + inhibits ENaC
Cyclosporine	Blocks aldosterone activity
Digitalis	Inhibits Na^+/K^+-ATPase
Heparin	Blocks aldosterone production
Nonselective β-adrenergic blocker	Interferes with β_2-mediated intracellular potassium uptake
NSAID	Decreases renal perfusion → decreased potassium delivery to the collecting ducts
Potassium-sparing diuretic	Inhibits ENaC or aldosterone receptor
Succinylcholine	Causes extracellular leakage of potassium through acetylcholine receptors
Trimethoprim	Inhibits ENaC
ARB = angiotensin II receptor blocker; **AT** = angiotensin; **ENaC** = epithelial sodium channel; **Na^+/K^+-ATPase** = sodium-potassium pump; **NSAID** = nonsteroidal anti-inflammatory drug.	

This patient with diabetes complicated by albuminuria, coronary artery disease (CAD), and hypertension is being appropriately treated with an **ACE inhibitor** (eg, lisinopril). ACE inhibitors block the conversion of angiotensin I to angiotensin II, leading to decreased systemic vascular tone, reduced aldosterone secretion, and lowered blood

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This patient with diabetes complicated by albuminuria, coronary artery disease (CAD), and hypertension is being appropriately treated with an **ACE inhibitor** (eg, lisinopril). ACE inhibitors block the conversion of angiotensin I to angiotensin II, leading to decreased systemic vascular tone, reduced aldosterone secretion, and lowered blood pressure. Decreased aldosterone levels cause increased sodium excretion and **potassium retention**, which can lead to **hyperkalemia**. The risk is greatest in patients with renal insufficiency and those taking potassium-sparing diuretics or medications that decrease glomerular filtration (eg, nonsteroidal anti-inflammatory drugs).

Mineralocorticoid receptor blockers (eg, spironolactone, **eplerenone**), angiotensin II receptor blockers (eg, losartan), and epithelial sodium channel blockers (eg, amiloride, triamterene) are other antihypertensives that can raise potassium levels. Concurrent use with an ACE inhibitor can result in severe hyperkalemia and should be avoided in this patient with mild hyperkalemia at baseline.

(Choices A and C) Dihydropyridine calcium channel blockers (eg, amlodipine, nifedipine) inhibit the L-type calcium channel on vascular smooth muscle, leading to arterial vasodilation. Nondihydropyridine calcium channel blockers (eg, verapamil) reduce heart rate and contractility. Diltiazem causes both vasodilation and decreased heart rate and contractility. Calcium channel blockers do not cause significant electrolyte abnormalities and may be used in patients at risk for hyperkalemia. Although dihydropyridine calcium channel blockers may cause lower extremity edema, *trace pedal edema* is extremely common and *not a contraindication* for use.

(Choices B and E) Loop diuretics (eg, bumetanide, furosemide) inhibit the $Na^+/K^+/2Cl^-$ symporter in the ascending limb of the loop of Henle. They decrease intravascular volume, leading to an increase in serum aldosterone and a resultant increase in urinary potassium excretion, causing hypokalemia.

(Choice F) Metoprolol is a selective beta-1 blocker that decreases heart rate, cardiac contractility, and renin release, which reduces arterial blood pressure and myocardial oxygen demand. Although nonselective beta blockers (eg, carvedilol) can cause hyperkalemia by inhibiting beta-2–mediated intracellular potassium uptake, this does not typically occur with selective beta-1 blockers. Although beta blockers can mask hypoglycemia in *diabetic patients*, this is *not a contraindication* and they can still be used (cautiously) in patients with CAD to reduce myocardial oxygen demand.

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Educational objective:
ACE inhibitors block the conversion of angiotensin I to angiotensin II, thereby reducing vasoconstriction and aldosterone secretion. Decreased aldosterone leads to potassium retention, which can cause hyperkalemia, especially in patients with renal insufficiency and those taking other medications that raise potassium levels (eg, angiotensin II receptor blockers, mineralocorticoid receptor blockers).

References

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A 75-year-old man is brought to the emergency department by paramedics following a high-speed motor vehicle collision. Several attempts are made to resuscitate him but they are unsuccessful. Autopsy findings include heavy calcifications of the aortic valve. The patient's medical records indicate that he had no significant medical history and took no medications. He is described as being in good health and tolerant of moderate levels of physical activity. ECG from his last office visit shows no abnormalities. Which of the following most likely preceded the aortic valve changes observed in this patient?

- A. Chronic cell injury
- B. Extracellular amyloid deposition
- C. Hypercalcemia
- D. Intracellular hemosiderin accumulation
- E. Pathologic cell hypertrophy

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Explanation

Pathologic calcification		
	Dystrophic	**Metastatic**
Etiology	• Cellular injury	• Elevated calcium &/or phosphate levels (eg, 1° or 2° hyperparathyroidism)

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Pathologic calcification		
	Dystrophic	**Metastatic**
Etiology	• Cellular injury	• Elevated calcium &/or phosphate levels (eg, 1° or 2° hyperparathyroidism)
Tissue distribution	• Localized to injured/necrotic tissue	• Widespread, particularly alkaline tissues (eg, gastric mucosa, kidney, lung)
Serum calcium level	• Normal	• Elevated
Clinical examples	• Atheroma calcification • Calcific aortic valve disease • Fat saponification (eg, pancreatitis)	• Nephrocalcinosis

This patient's aortic valve calcifications most likely represent **dystrophic calcification** affecting an aging aortic valve. Dystrophic calcification is considered a hallmark of **cell injury** and death, occurring in all types of **necrosis** (eg, coagulative, fat, caseous, liquefactive) in the setting of **normal calcium levels**. On gross examination, dystrophic calcium deposits are seen as fine, gritty, white granules or clumps. On hematoxylin and eosin staining, these deposits typically appear as dark purple, sharp-edged aggregates; deposits that develop lamellated outer layers are described as psammoma bodies.

Dystrophic calcification in aged (or damaged) cardiac valves is thought to be the result of endothelial and fibroblast injury and death secondary to **chronic hemodynamic stress** (can be accentuated by valvular abnormalities) or **atherosclerotic inflammation**. Subsequent release of cellular degradation products, inflammatory mediators, and other proteins into the valvular interstitium promotes calcification and thickening of the valve leaflets and annulus. These changes are often asymptomatic in older adults (ie, **aortic sclerosis**); however, over time, progressive valvular stiffening can lead to outflow obstruction (ie, **calcific aortic stenosis**).

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	• Fat saponification (eg, pancreatitis)	

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(Choice B) Extracellular amyloid deposition occurs in patients with amyloidosis. Amyloidosis can cause restrictive cardiomyopathy and heart failure, but it is not directly associated with calcification of the aortic valve.

(Choice C) Hypercalcemia is associated with metastatic calcification, which affects normal tissues and organs. In this process, calcium deposition typically occurs in more alkaline tissues involved in acid excretion, such as kidneys, lungs, and gastric mucosa.

(Choice D) Intracellular hemosiderin accumulation is common in patients who have hemolytic anemia or who undergo frequent blood transfusions. It is not directly associated with calcification of the aortic valve.

(Choice E) Pathologic cell hypertrophy of left ventricular cardiomyocytes (ie, left ventricular hypertrophy) can occur as the result of severe aortic stenosis.

Educational objective:
Dystrophic calcification occurs in injured or necrotic tissue in the setting of normal calcium levels; metastatic

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Educational objective:
Dystrophic calcification occurs in injured or necrotic tissue in the setting of normal calcium levels; metastatic calcification occurs in normal tissue in the setting of hypercalcemia.

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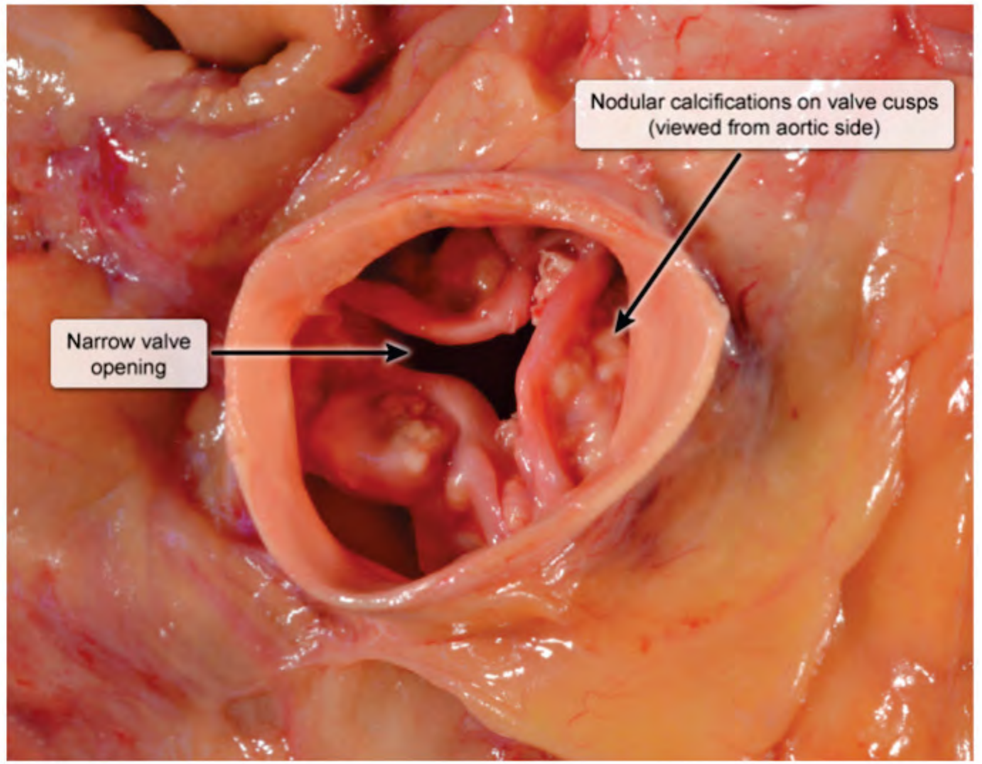
Calcific aortic stenosis



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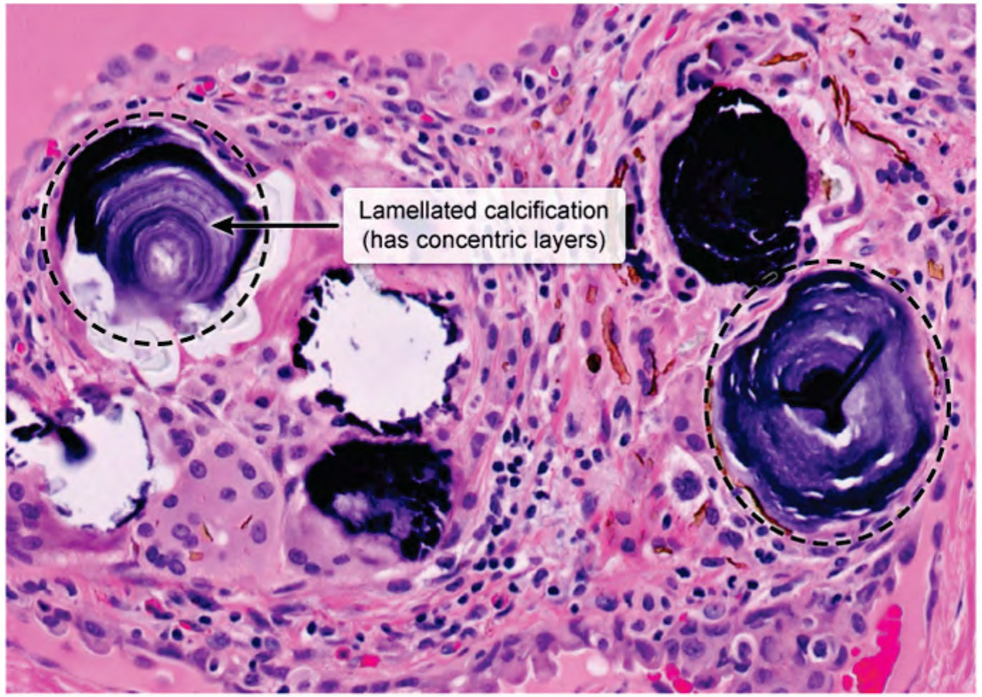

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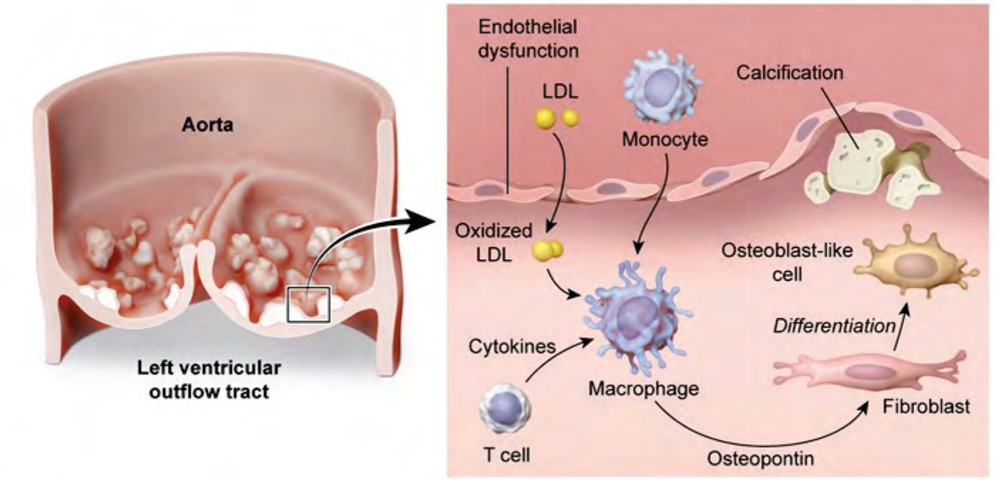

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Dystrophic calcification occurs in injured or necrotic tissue in the setting of normal calcium levels; metastatic

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A 43-year-old man is evaluated for occasional palpitations provoked by anxiety. The patient describes a sudden-onset pounding sensation in the chest followed by lightheadedness and shortness of breath. He has never had syncope. The patient has no history of heart disease, and family history is unremarkable. After initial evaluation, he is treated with verapamil and reports marked improvement in the frequency of palpitation episodes. The tracing below shows the electrical activity of a specific type of cardiac cell in the patient's heart.

Which of the following effects would verapamil administration most likely have on these cells?

- A. Decreased excitation and contraction coupling
- B. Increased action potential amplitude
- C. Lowered threshold potential
- D. Reduced refractory period
- E. Slowed spontaneous depolarization

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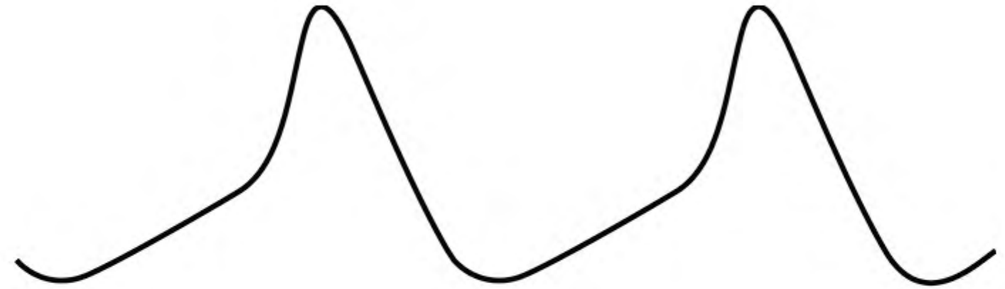

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Which of the following effects would verapamil administration most likely have on these cells?

- A. Decreased excitation and contraction coupling (38%)
- ✗ B. Increased action potential amplitude (3%)
- C. Lowered threshold potential (2%)
- D. Reduced refractory period (2%)
- ✓ E. Slowed spontaneous depolarization (53%)

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Correct answer
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53%
Answered correctly

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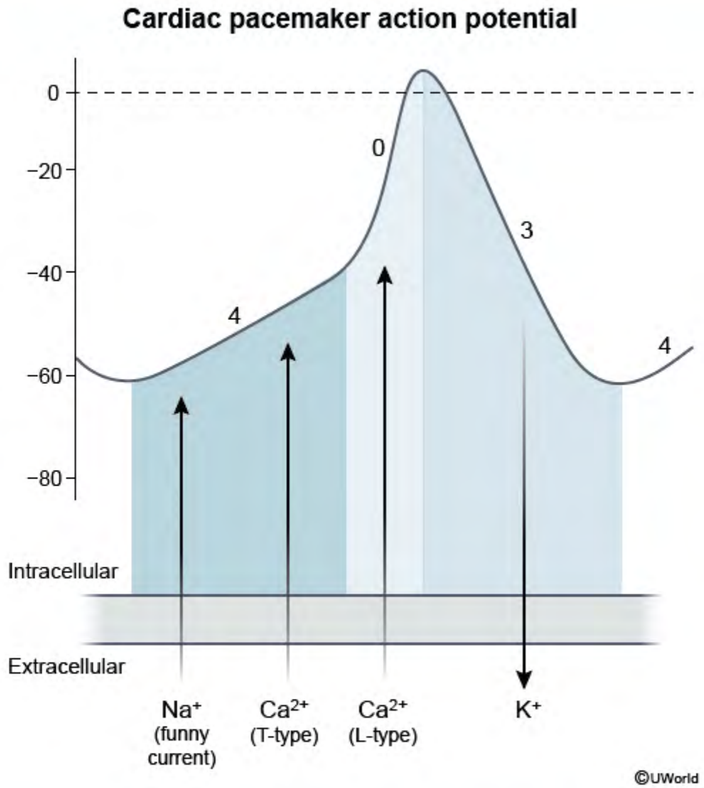

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The above tracing demonstrates the action potential of cardiac pacemaker cells in cardiac **slow-response tissues** such as the **sinoatrial** (SA) and **atrioventricular** (AV) nodes. The pacemaker action potential includes the following phases:

- Phase 4 (spontaneous depolarization) begins after hyperpolarization triggers the opening of HCN channels that allow slow influx of Na^+ ("funny current"). T-type (transient) Ca^{2+} channels then open once the membrane potential becomes more positive, allowing Ca^{2+} influx to contribute to depolarization. As the pacemaker cell approaches threshold, **L-type** (long-lasting) **Ca^{2+} channels begin to open**, which further increases Ca^{2+} influx and significantly decreases the time until threshold is reached.
- Phase 0 (upstroke) is characterized by continued opening of L-type Ca^{2+} channels. In cardiac slow-response tissues, the action potential upstroke is much slower and transitions gradually from phase 4 due to the relatively slow influx of Ca^{2+} into the cell.
- Phase 3 (repolarization) is characterized by the opening of K^+ channels and efflux of K^+ from the cell in conjunction with the closure of L-type Ca^{2+} channels.

This patient's anxiety-provoked episodes of sudden-onset palpitations associated with lightheadedness and shortness of breath are consistent with **paroxysmal supraventricular tachycardia**. Class IV antiarrhythmics (eg, **verapamil**, diltiazem) are commonly used to prevent recurrent nodal tachyarrhythmias such as PSVT. They work by **blocking L-type calcium channels** in cardiac slow-response tissues, which causes **slowing of phase 4 depolarization** and reduced conduction velocity in the SA and AV nodes.

(Choice A) Verapamil decreases the amount of intracellular calcium available for excitation-contraction coupling within cardiac myocytes. This reduces myocardial contractility, which can be harmful in patients with impaired ventricular function. However, the tracing shown in the question stem corresponds to pacemaker action potentials, not cardiac myocyte action potentials.

(Choices B and C) The threshold potential refers to the amount of depolarization required to initiate an action

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(Choices B and C) The threshold potential refers to the amount of depolarization required to initiate an action potential. Class I (sodium channel blockers) and class IV (calcium channel blockers) antiarrhythmics raise the threshold potential of cardiac fast- and slow-response tissues, respectively, by blocking the channels that trigger an action potential. This effect also results in reduced action potential amplitude, decreased upslope, and reduced impulse conduction speed within the respective tissues.

(Choice D) Verapamil's calcium blocking activity reduces the action potential duration in nodal tissues, but it also increases the refractory period by slowing the recovery of inactivated calcium channels. Class IA (eg, quinidine) and class III (eg, amiodarone) antiarrhythmics also increase the refractory period by blocking potassium channels and prolonging repolarization.

Educational objective:
Class IV antiarrhythmics (eg, verapamil, diltiazem) are commonly used to prevent recurrent nodal arrhythmias (eg, paroxysmal supraventricular tachycardia). They work by blocking calcium channels in slow-response cardiac tissues, slowing phase 4 (spontaneous depolarization) and phase 0 (upstroke). This reduces impulse conduction velocity in the sinoatrial and atrioventricular nodes.

References

- Class IV antiarrhythmic agents: new compounds using an old strategy.
- Antiarrhythmic and anti-ischemic properties of calcium-channel antagonists

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A group of researchers is studying population-based screening interventions to reduce mortality from abdominal aortic aneurysm. This disorder develops primarily in elderly patients and is often asymptomatic until an acute rupture event, which is frequently fatal. Screening of high-risk patients for abdominal aortic aneurysm is found to reduce mortality. Which of the following risk factor combinations would likely define the highest-risk group for screening purposes?

- A. Diabetes, smoking
- B. Female, hyperlipidemia
- C. Female, hypertension
- D. Hyperlipidemia, hypertension
- E. Male, diabetes
- F. Male, smoking

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- ✗ A. Diabetes, smoking (10%)
- B. Female, hyperlipidemia (0%)
- C. Female, hypertension (3%)
- D. Hyperlipidemia, hypertension (27%)
- E. Male, diabetes (0%)
- ✓ F. Male, smoking (57%)

Incorrect
Correct answer
F

57%
Answered correctly

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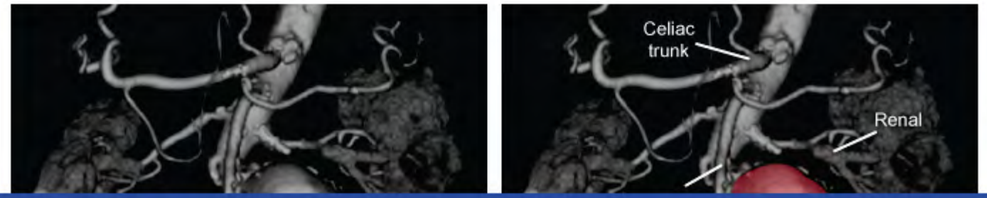
Explanation

Abdominal aortic aneurysm (CT angiography)



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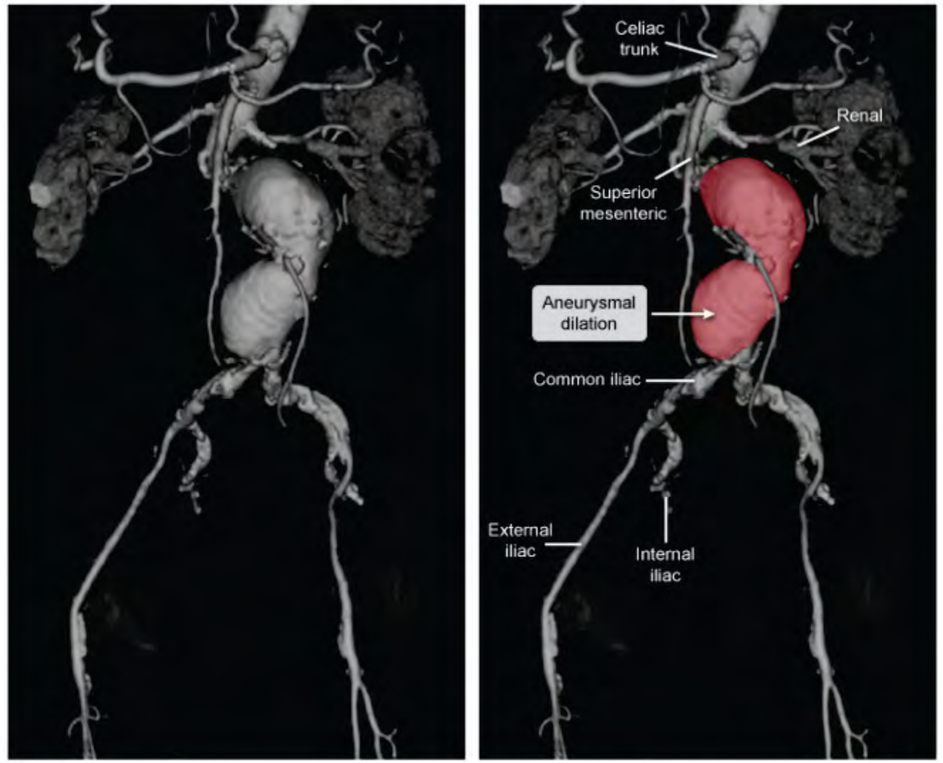

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Focal dilation of the abdominal aorta >50% above normal (or >3 cm in diameter) is termed **abdominal aortic aneurysm** (AAA). AAA formation involves **transmural inflammation** of the aortic wall with subsequent apoptosis

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Focal dilation of the abdominal aorta >50% above normal (or >3 cm in diameter) is termed **abdominal aortic aneurysm** (AAA). AAA formation involves **transmural inflammation** of the aortic wall with subsequent apoptosis of smooth muscle cells and degradation of matrix proteins (eg, elastin, collagen). This causes progressive thinning of the aortic wall with secondary expansion of the lumen due to chronic hemodynamic stress. AAA is generally asymptomatic until aneurysm rupture, which typically presents with acute abdominal pain and hypotension; mortality of AAA rupture is 75%-90%.

Major **risk factors** for development of AAA include:

- Age >65
- **Smoking** (up to 15-fold risk increase)
- **Male sex**

Smoking appears to be associated with increased inflammatory infiltrates and formation of reactive oxygen species (eg, superoxide anion) in the aortic wall. In light of this, screening for AAA with ultrasonography is recommended for men age 65-75 who have ever smoked. The risk of rupture increases with aneurysm diameter, and surgical or endovascular repair is considered for aneurysms ≥5.5 cm in diameter.

(Choices A and E) Although diabetes mellitus increases the risk for atherosclerotic cardiovascular disease, the risk of AAA is lower in patients with diabetes, possibly due to the effects of glycosylation of matrix proteins in the aortic wall.

(Choices B and C) Female sex is associated with a lower risk of AAA, and the benefit of screening in women (including in women who smoke) has not been shown to outweigh the risks of overdiagnosis and potential treatment complications.

(Choice D) Hypertension is associated with an increased risk of aneurysm rupture, but the relation to aneurysm incidence and expansion is less clear. Although there is a broad coincidence of AAA and atherosclerosis, studies on an epidemiologic link between hyperlipidemia and AAA are mixed.

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Educational objective:
Abdominal aortic aneurysm is focal dilation of the abdominal aorta >50% above normal (or >3 cm in diameter). It is generally asymptomatic until aneurysm rupture, which is frequently fatal. Risk factors include age >65, smoking, and male sex.

Pathophysiology	Cardiovascular System	Aortic aneurysm
Subject	System	Topic

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A 62-year-old man dies suddenly in his sleep. The patient had a history of type 2 diabetes mellitus and hypertension, and he smoked a pack of cigarettes daily for 25 years. His adherence to medical care was poor. Autopsy is performed, and a histologic section of his left ventricular myocardium is shown below:

Which of the following directly promotes the formation of the tissue indicated by the arrow?

- A. Endothelium-derived relaxing factor
- B. Interferon-beta
- C. Interleukin-8
- D. Transforming growth factor-beta
- E. Tumor necrosis factor-alpha

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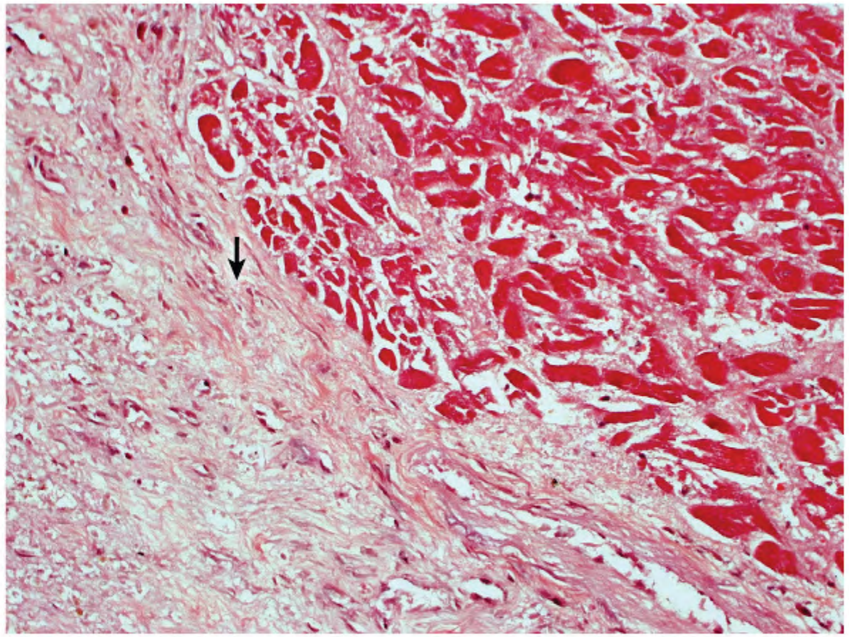

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Which

D. Transforming growth factor-beta
E. Tumor necrosis factor-alpha

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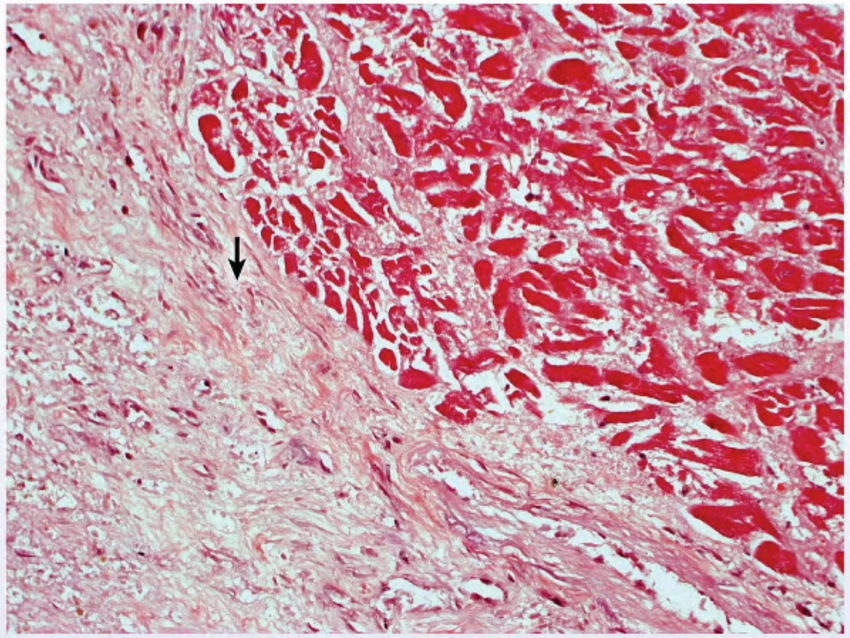

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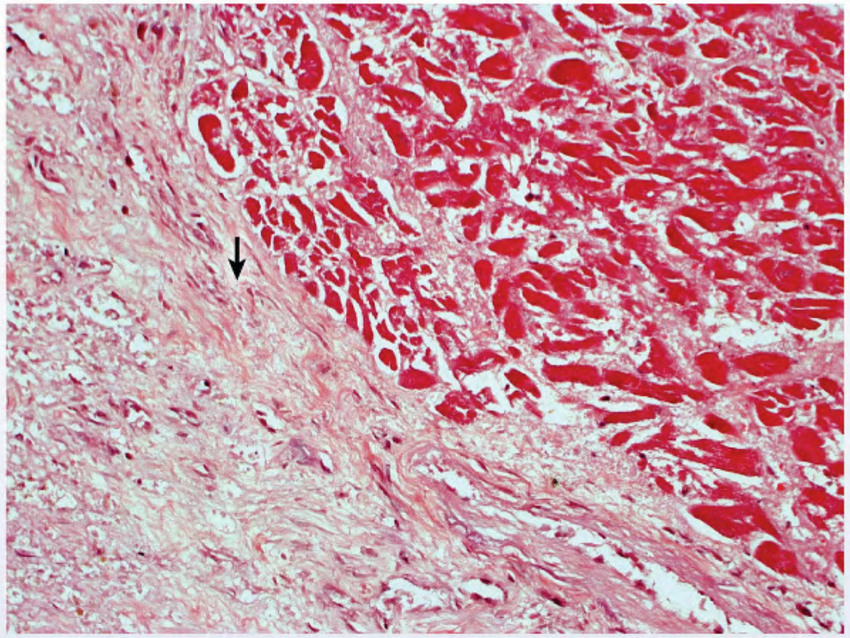

Which of the following directly promotes the formation of the tissue indicated by the arrow?

- A. Endothelium-derived relaxing factor (3%)
- B. Interferon-beta (1%)
- ✗ C. Interleukin-8 (2%)
- ✓ D. Transforming growth factor-beta (81%)
- E. Tumor necrosis factor-alpha (10%)

Incorrect
Correct answer
D

81%
Answered correctly

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C. Interleukin-8 (2%)

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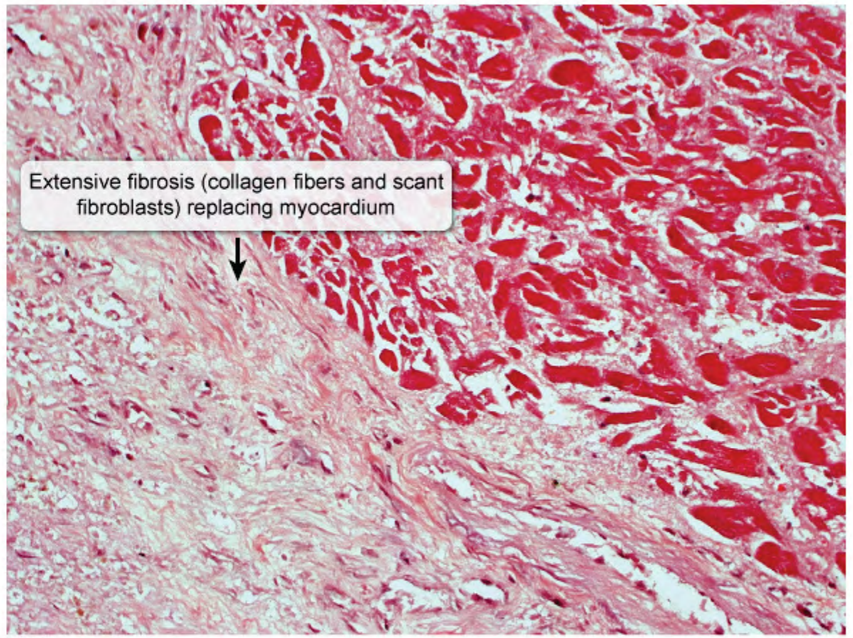
Resolved myocardial infarcts



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This patient's autopsy shows a large area of **fibrosis** replacing myocardium, consistent with scar tissue from a resolved **myocardial infarction** (MI). Myocardial tissue damage due to ischemia initiates a series of changes that are analogous to cutaneous wound healing:

- Inflammation: Hours to days post-MI, neutrophils and macrophages remove necrotic tissue and release cytokines and growth factors that initiate tissue proliferation.
- Proliferation: Days to weeks post-MI, **transforming growth factor-beta (TGF-β)** and other anti-inflammatory cytokines downregulate the inflammatory response and stimulate **fibroblast** migration and proliferation, causing extensive type I and III collagen deposition.
- Remodeling: Weeks to months post-MI, TGF-β also stimulates production of matrix metalloproteinases (MMPs) that facilitate collagen remodeling (ie, fiber rearrangement) and crosslinking to form dense **scar tissue**.

Because cardiac myocytes are terminally differentiated and do not proliferate to a significant degree in response to injury, areas of extensive myocardial fibrosis do not regain the electrical properties of normal myocardium. Therefore, patients with large scars from previous MIs are at increased risk of **sudden death** due to arrhythmia.

(Choice A) Endothelium-derived relaxing factor (ie, nitric oxide), causes vasodilation via stimulation of vascular smooth muscle relaxation. Although local synthesis of nitric oxide is increased post-MI, it contributes to reperfusion rather than fibrosis at the site of injury.

(Choice B) Interferon-beta (IFN-β) is released by a variety of cells, including macrophages, and functions as an anti-inflammatory cytokine that reduces the formation of fibrotic tissue.

(Choices C and E) Interleukin-8 and tumor necrosis factor-alpha (TNF-α) are proinflammatory cytokines that stimulate the recruitment and activation of other immune cells (eg, neutrophils). However, neither directly promotes fibrosis in a healing wound.

Educational objective:

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Educational objective:
After myocardial infarction, transforming growth factor-beta (TGF-β) reduces inflammation and promotes tissue remodeling due to fibroblast proliferation and collagen deposition, resulting in fibrosis in the area damaged by ischemia. Abnormal myocardium at the scar border can predispose patients to sudden death as a result of arrhythmia.

Pathology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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Time after myocardial infarction	Predominant light microscopic changes
0-4 hours	• No visible change
4-12 hours	• Wavy fibers with narrow, elongated myocytes
12-24 hours	• Myocyte hypereosinophilia with pyknotic (shrunken) nuclei
1-3 days	• Coagulation necrosis (loss of nuclei & striations) • Prominent neutrophilic infiltrate
3-7 days	• Disintegration of dead neutrophils & myofibers • Macrophage infiltration at border areas
7-10 days	• Robust phagocytosis of dead cells by macrophages • Beginning formation of granulation tissue at margins
10-14 days	• Well-developed granulation tissue with neovascularization
2-8 weeks	• Progressive collagen deposition & scar formation

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(Choices C and E) Interleukin-8 and tumor necrosis factor-alpha (TNF-α) are proinflammatory cytokines that

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A 4-week-old boy undergoes cardiac catheterization for closure of a patent ductus arteriosus. The patient was born at 26 weeks gestation due to placental abruption. He has been cared for in the neonatal intensive care unit since delivery. Pulse oximetry shows 99% in the right hand and left foot. Echocardiography shows a moderately sized patent ductus arteriosus. After catheterization but before closure, blood gases are obtained from several locations. Compared with the partial pressure of oxygen (PaO_2) of a normal infant, this patient would be expected to have a higher PaO_2 in which of the following locations?

- A. Ascending aorta
- B. Descending aorta
- C. Left atrium
- D. Pulmonary artery
- E. Right atrium

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- ✗ A. Ascending aorta (5%)
- B. Descending aorta (5%)
- C. Left atrium (8%)
- ✓ D. Pulmonary artery (70%)
- E. Right atrium (9%)

Incorrect
Correct answer
D

70%
Answered correctly

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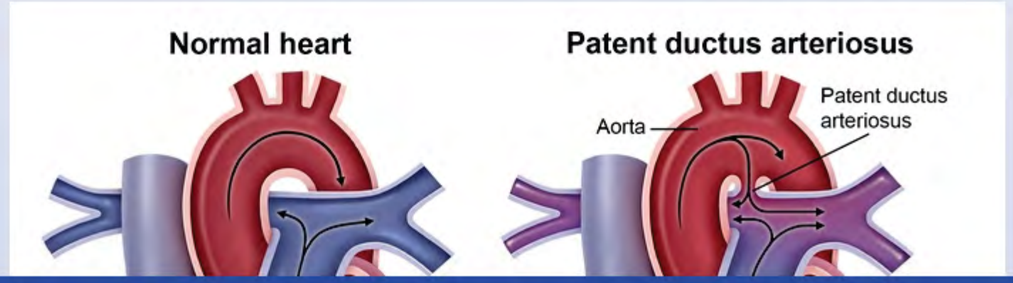
Explanation



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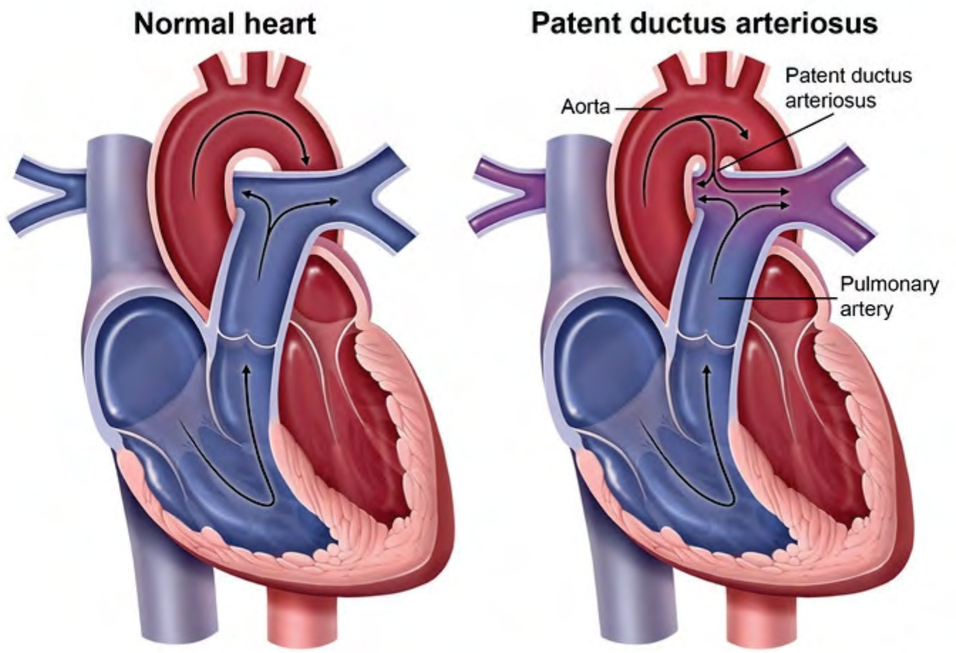

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A **patent ductus arteriosus** (PDA) is a **left-to-right shunt** that allows continuous flow of oxygenated blood from the aorta into the pulmonary artery. In the pulmonary artery, the oxygenated blood mixes with deoxygenated blood coming from the right ventricle. This leads to a **higher** partial pressure of oxygen (**PaO2**) in the **pulmonary artery** than would normally be seen.

(Choices A and B) In patients with a left-to-right shunt (eg, atrial or ventricular septal defect, PDA), the PaO_2 in the aorta remains the same. In contrast, if the shunt becomes right-to-left due to increased right-sided pressures (ie, Eisenmenger physiology) the oxygen concentration in the aorta can be lower than expected. However, patients with Eisenmenger syndrome have cyanosis, which is not seen in this patient.

(Choice C) The left atrium receives oxygenated blood from the pulmonary circulation, specifically from the alveolar capillaries via the pulmonary veins. Although the volume of blood received by the left atrium is increased with left-to-right PDA shunts, the PaO_2 in the left atrium is unchanged as all the blood passes through the lungs.

(Choice E) The right atrium receives deoxygenated blood from the systemic circulation, primarily the superior and inferior vena cava. The left-to-right shunt of a PDA is "downstream" of the right atrium (ie, in the pulmonary artery), so the PaO_2 of right atrial blood is unchanged.

Educational objective:
In patients with patent ductus arteriosus, left-to-right shunting of oxygenated blood from the aorta into the pulmonary artery results in a higher-than-expected oxygen concentration in the pulmonary artery. Oxygen saturation in the left and right heart chambers is unchanged.

Physiology	Cardiovascular System	Patent ductus arteriosus
Subject	System	Topic

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A 64-year-old man comes to the emergency department due to chest discomfort and shortness of breath. Medical history includes hypertension, type 2 diabetes mellitus, and severe osteoarthritis of the left knee. The patient is an active smoker with a 20-pack-year smoking history. He undergoes dobutamine infusion with simultaneous cardiac imaging. The following is seen:

- Before infusion: normal left ventricular contractility, ejection fraction 60%
- During infusion: decreased apical contractility, ejection fraction 45%
- Five minutes after infusion: normal left ventricular contractility, ejection fraction 60%

Which of the following best explains the observed findings?

A. Coronary artery occlusion due to plaque thrombosis
B. Coronary artery vasospasm
C. Focal myocardial fibrosis
D. Normal myocardial contractile response
E. Supply-demand mismatch due to fixed coronary stenosis

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Which of the following best explains the observed findings?

A. Coronary artery occlusion due to plaque thrombosis (9%)
B. Coronary artery vasospasm (10%)
✗ C. Focal myocardial fibrosis (3%)
D. Normal myocardial contractile response (18%)
✓ E. Supply-demand mismatch due to fixed coronary stenosis (57%)

Incorrect
Correct answer
E

57%
Answered correctly

03 secs
Time Spent

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Explanation

Stable angina results from **fixed coronary artery stenosis** in the setting of atherosclerotic coronary artery disease (CAD). Such a stenosis limits blood supply to the downstream myocardium, which, during periods of increased myocardial oxygen demand (eg, exercise), can cause a **mismatch of oxygen supply and demand** that

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Stable angina results from **fixed coronary artery stenosis** in the setting of atherosclerotic coronary artery disease (CAD). Such a stenosis limits blood supply to the downstream myocardium, which, during periods of increased myocardial oxygen demand (eg, exercise), can cause a **mismatch of oxygen supply and demand** that manifests as anginal symptoms (eg, chest pain, shortness of breath).

Pharmacologic stress testing can be useful in diagnosing atherosclerotic CAD. Dobutamine, a beta-1 agonist, **increases heart rate and contractility** to mimic the increase in myocardial oxygen demand that occurs with exercise. Myocardium that is unable to obtain sufficient blood flow to meet the increased oxygen demand typically demonstrates a **transient decrease in contractility** (ie, wall motion defect), often leading to **reduced ejection fraction**. This patient with a transient wall motion defect localized to the cardiac apex most likely has a fixed atherosclerotic lesion that limits blood flow to that area of myocardium.

(Choice A) Coronary artery occlusion due to plaque rupture and thrombosis leads to acute myocardial infarction (MI). Because of total coronary artery occlusion and a completely blocked myocardial oxygen supply, a persistent (before, during, and after dobutamine infusion), rather than transient, wall motion defect would be expected.

(Choice B) Vasospastic angina is due to coronary artery vasospasm and can mimic stable angina that results from atherosclerotic CAD; however, vasospastic angina typically occurs in relatively young patients (age <50). In addition, dobutamine has only a small agonistic effect on alpha-1 receptors and is unlikely to trigger coronary vasospasm.

(Choice C) Focal myocardial fibrosis typically results from previous MI. Contractility is impaired in the fibrosed myocardium, leading to a persistent wall motion defect similar to that expected with acute MI.

(Choice D) In the normal state, dobutamine boosts myocardial contractility, and the rise in myocardial oxygen demand is met by increased myocardial oxygen supply via increased blood flow. Therefore, the normal response to dobutamine infusion is a transient increase in ejection fraction.

Educational objective:
Stable angina results from fixed coronary artery stenosis that limits blood flow to downstream myocardium, preventing the myocardial oxygen supply from increasing during exertion. Dobutamine mimics the effects of

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Educational objective:
Stable angina results from fixed coronary artery stenosis that limits blood flow to downstream myocardium, preventing the myocardial oxygen supply from increasing during exertion. Dobutamine mimics the effects of exercise and increases myocardial oxygen demand; it can be used during stress testing to provoke areas of ischemic myocardium, which can be recognized on imaging by a localized and transient decrease in contractility (ie, wall motion defect).

Pathophysiology	Cardiovascular System	Coronary artery disease
Subject	System	Topic

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A 22-year-old man comes to the office due to a 6-month history of difficulty breathing and chest discomfort on exertion. He also reports intermittent palpitations. The patient's only other medical condition is major depression. Temperature is 37 C (98.6 F), blood pressure is 120/80 mm Hg, pulse is 82/min, and respirations are 15/min. Pulse oximetry on room air shows an oxygen saturation of 98%. BMI is 24 kg/m^2. The lungs are clear on auscultation. Cardiac examination reveals a prominent left ventricular impulse on palpation, an S4, and a faint systolic murmur at the left sternal border that accentuates during the strain phase of the Valsalva maneuver. Verapamil therapy would most likely improve this patient's dyspnea through which of the following mechanisms?

- A. Decrease in left ventricular afterload
- B. Decrease in left ventricular end-diastolic cavity size
- C. Increase in atrioventricular conduction velocity
- D. Increase in left ventricular diastolic filling time
- E. Increase in myocardial contraction velocity

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- A. Decrease in left ventricular afterload (18%)
- ✗ B. Decrease in left ventricular end-diastolic cavity size (4%)
- C. Increase in atrioventricular conduction velocity (3%)
- ✓ D. Increase in left ventricular diastolic filling time (69%)
- E. Increase in myocardial contraction velocity (4%)

Incorrect
Correct answer
D

69%
Answered correctly

03 secs
Time Spent

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Explanation

Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain

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Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain
Phenotypic manifestations	• Myocardial disarray & coronary microvascular dysfunction • Asymmetric septal hypertrophy with dynamic LVOT obstruction • LV stiffening with diastolic dysfunction • Increased risk of ventricular arrhythmias & SCD
Clinical presentation	• Often asymptomatic • Exertional dyspnea, angina, palpitations, light-headedness, syncope • Systolic murmur that increases in intensity with ↓ LV blood volume • Audible S4
LV = left ventricular; **LVOT** = left ventricular outflow tract; **SCD** = sudden cardiac death.	

A systolic murmur at the left sternal border that increases in intensity with maneuvers that decrease left ventricular (LV) blood volume is characteristic of **hypertrophic cardiomyopathy** (HCM). The murmur is caused by dynamic **LV outflow tract obstruction** that worsens with relatively low LV blood volume and improves with relatively high LV blood volume. Other common physical examination findings include an S4 (caused by LV wall-

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Pharmacologic management of HCM focuses on **maintaining relatively high LV blood volume** throughout the cardiac cycle, which minimizes LV outflow tract obstruction and associated symptoms. This is best accomplished via 2 mechanisms:

- **Negative chronotropy** (ie, reduced heart rate), which lengthens diastolic filling time to increase LV end-diastolic volume (ie, preload)
- **Negative inotropy** (ie, reduced LV contractility), which decreases the amount of blood ejected during ventricular systole (ie, increased end-systolic volume)

Beta blockers and nondihydropyridine calcium channel blockers (eg, verapamil) are used in HCM to provide negative chronotropy and inotropy and reduce symptoms related to outflow tract obstruction.

(Choice A) Decreased LV afterload is primarily caused by arterial dilators (eg, ACE inhibitors, dihydropyridine calcium channel blockers such as amlodipine) and is detrimental in HCM as it reduces LV blood volume to worsen LVOT obstruction. Verapamil has no significant vasodilatory effect on systemic vessels.

(Choices B and C) The negative chronotropy of verapamil decreases atrioventricular conduction velocity to lengthen diastolic filling time and increase LV end-diastolic cavity size. An increase in atrioventricular conduction velocity (eg, as occurs with exercise) tends to decrease LV end-diastolic cavity size and worsen LV outflow tract obstruction.

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cardiac cycle, which minimizes LV outflow tract obstruction and associated symptoms. This is best accomplished via 2 mechanisms:

- **Negative chronotropy** (ie, reduced heart rate), which lengthens diastolic filling time to increase LV end-diastolic volume (ie, preload)
- **Negative inotropy** (ie, reduced LV contractility), which decreases the amount of blood ejected during ventricular systole (ie, increased end-systolic volume)

Beta blockers and nondihydropyridine calcium channel blockers (eg, verapamil) are used in HCM to provide negative chronotropy and inotropy and reduce symptoms related to outflow tract obstruction.

(Choice A) Decreased LV afterload is primarily caused by arterial dilators (eg, ACE inhibitors, dihydropyridine calcium channel blockers such as amlodipine) and is detrimental in HCM as it reduces LV blood volume to worsen LVOT obstruction. Verapamil has no significant vasodilatory effect on systemic vessels.

(Choices B and C) The negative chronotropy of verapamil decreases atrioventricular conduction velocity to lengthen diastolic filling time and increase LV end-diastolic cavity size. An increase in atrioventricular conduction velocity (eg, as occurs with exercise) tends to decrease LV end-diastolic cavity size and worsen LV outflow tract obstruction.

(Choice E) The negative inotropy of verapamil decreases myocardial contraction velocity. Increased contraction velocity can worsen LV outflow tract obstruction by reducing end-systolic volume.

Educational objective:
Hypertrophic cardiomyopathy commonly involves dynamic left ventricular (LV) outflow tract obstruction that worsens with decreased LV blood volume and improves with increased LV blood volume. Nondihydropyridine calcium channel blockers (eg, verapamil) decrease heart rate (ie, negative chronotropy) and LV contractility (ie, negative inotropy) to increase LV blood volume, reduce LV outflow tract obstruction, and improve symptoms.

Pharmacology	Cardiovascular System	Hypertrophic cardiomyopathy
Subject	System	Topic

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A healthy 80-year-old man has participated in a longitudinal study over the past 30 years to study the effects of aging on overall cardiovascular status. He has undergone annual testing with echocardiography and his left ventricular ejection fraction has remained stable throughout the 30 years. Which of the following changes is most likely responsible for the preserved left ventricular ejection fraction in this patient?

- A. Cardiomyocyte hypertrophy
- B. Increased arterial reserve capacity
- C. Increased left ventricular compliance
- D. Increased left ventricular diastolic volume
- E. Stable ventricular myocyte number

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A healthy 80-year-old man has participated in a longitudinal study over the past 30 years to study the effects of aging on overall cardiovascular status. He has undergone annual testing with echocardiography and his left ventricular ejection fraction has remained stable throughout the 30 years. Which of the following changes is most likely responsible for the preserved left ventricular ejection fraction in this patient?

- ✓ A. Cardiomyocyte hypertrophy (60%)
- B. Increased arterial reserve capacity (2%)
- C. Increased left ventricular compliance (10%)
- D. Increased left ventricular diastolic volume (11%)
- E. Stable ventricular myocyte number (14%)

Correct

60% Answered correctly

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2023 Version

Explanation

Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
Mild concentric LVH*	• Response to cardiomyocyte dropout & ↑ afterload • Resting EF, SV & cardiac output maintained • ↓ Maximal cardiac output

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Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
Mild concentric LVH*	• Response to cardiomyocyte dropout & ↑ afterload • Resting EF, SV & cardiac output maintained • ↓ Maximal cardiac output
Conduction cell degeneration	• Slightly ↓ resting heart rate • ↓ Maximal heart rate
Reduced baroreceptor sensitivity & adrenergic responsiveness	• ↑ Orthostasis • ↓ Heart rate & contractility response • ↑ Circulating catecholamines
*A soft S4 is a normal finding in the elderly. **EF** = ejection fraction; **HTN** = hypertension; **LVH** = left ventricular hypertrophy; **SV** = stroke volume.	

The **cardiovascular system** undergoes an array of **age-related physiologic changes**. Over time, cardiomyocytes are lost to both apoptosis and necrosis (ie, cardiomyocyte dropout), resulting in an overall **reduced cardiomyocyte number**. The loss of ventricular cardiomyocytes **(Choice E)** weakens contractile strength. In addition, the aorta and other large arteries stiffen with age, resulting in reduced arterial compliance (ie, reduced arterial reserve capacity) **(Choice B)** and increased left ventricular (LV) afterload. To compensate for the cardiomyocyte loss and increased afterload, **cardiomyocyte hypertrophy** (ie, mild concentric LV hypertrophy) occurs, allowing for **maintenance of LV contractility** and ejection fraction.

Cardiomyocyte dropout also affects cardiac conduction cells, contributing to decreased maximal heart rate and

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Reduced baroreceptor sensitivity & adrenergic responsiveness	• ↓ Heart rate & contractility response • ↑ Circulating catecholamines

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EF = ejection fraction; **HTN** = hypertension; **LVH** = left ventricular hypertrophy; **SV** = stroke volume.

The **cardiovascular system** undergoes an array of **age-related physiologic changes**. Over time, cardiomyocytes are lost to both apoptosis and necrosis (ie, cardiomyocyte dropout), resulting in an overall **reduced cardiomyocyte number**. The loss of ventricular cardiomyocytes **(Choice E)** weakens contractile strength. In addition, the aorta and other large arteries stiffen with age, resulting in reduced arterial compliance (ie, reduced arterial reserve capacity) **(Choice B)** and increased left ventricular (LV) afterload. To compensate for the cardiomyocyte loss and increased afterload, **cardiomyocyte hypertrophy** (ie, mild concentric LV hypertrophy) occurs, allowing for **maintenance of LV contractility** and ejection fraction.

Cardiomyocyte dropout also affects cardiac conduction cells, contributing to decreased maximal heart rate and slightly decreased resting heart rate. Decreased responsiveness to adrenergic stimuli with age also contributes to reduced maximal heart rate.

(Choices C and D) The mild concentric LV hypertrophy that occurs with age results in reductions in LV compliance and LV end-diastolic volume. However, because these changes are relatively slight, stroke volume and cardiac output are mostly preserved.

Educational objective:
Cardiomyocyte hypertrophy (ie, concentric left ventricular hypertrophy) occurs with age to allow for maintenance of left ventricular contractility and ejection fraction despite cardiomyocyte dropout and increased left ventricular afterload (due to reduced aortic compliance).

Physiology	Cardiovascular System	Aging
Subject	System	Topic

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A 30-year longitudinal study that follows untreated individuals with no known cardiovascular disease from age 50 to 80 is performed to investigate normal age-related cardiovascular changes. Several parameters are measured annually. Patients age 80 are most likely to show which of the following changes compared to when they were age 50?

	Systolic blood pressure	Diastolic blood pressure	Pulse pressure
A.	Decreased	Decreased	Decreased
B.	Decreased	Decreased	Unchanged
C.	Unchanged	Decreased	Increased
D.	Increased	Decreased	Increased
E.	Increased	Increased	Increased

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A 30-year longitudinal study that follows untreated individuals with no known cardiovascular disease from age 50 to 80 is performed to investigate normal age-related cardiovascular changes. Several parameters are measured annually. Patients age 80 are most likely to show which of the following changes compared to when they were age 50?

	Systolic blood pressure	Diastolic blood pressure	Pulse pressure	
✗ A.	Decreased	Decreased	Decreased	(5%)
B.	Decreased	Decreased	Unchanged	(8%)
C.	Unchanged	Decreased	Increased	(5%)
✓ D.	Increased	Decreased	Increased	(53%)
E.	Increased	Increased	Increased	(27%)

Incorrect
Correct answer
D

53%
Answered correctly

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Time Spent

2023
Version

Explanation

Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
	• Response to cardiomyocyte dropout & ↑ afterload

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Physiologic age-related cardiovascular changes	
Aortic stiffening	• Elastin replacement with collagen • ↑ Pulse pressure (isolated systolic HTN)
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Reduced baroreceptor sensitivity & adrenergic responsiveness	• ↑ Orthostasis • ↓ Heart rate & contractility response • ↑ Circulating catecholamines
*A soft S4 is a normal finding in the elderly. **EF** = ejection fraction; **HTN** = hypertension; **LVH** = left ventricular hypertrophy; **SV** = stroke volume.	

Diastolic blood pressure is the baseline hydrostatic pressure in the arterial system and is directly related to systemic vascular resistance (SVR) and arterial blood volume. **Pulse pressure** is the amount that arterial pressure increases above diastolic pressure during left ventricular contraction; it is directly related to stroke volume and **inversely related to aortic compliance**. Systolic blood pressure is the summation of diastolic pressure and pulse pressure.

Age-related stiffening of the aorta is the primary driver of characteristic blood pressure changes that occur in those age >65. The reduced aortic compliance in the setting of unchanged stroke volume leads to **increased pulse pressure**. Reduced compliance also causes less blood volume to be retained in the arterial system (ie, blood is effectively displaced to the more compliant venous compartment), resulting in slightly **decreased diastolic**

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Age-related stiffening of the aorta is the primary driver of characteristic blood pressure changes that occur in those age >65. The reduced aortic compliance in the setting of unchanged stroke volume leads to **increased pulse pressure**. Reduced compliance also causes less blood volume to be retained in the arterial system (ie, blood is effectively displaced to the more compliant venous compartment), resulting in slightly **decreased diastolic pressure**. The increase in pulse pressure is greater than the decrease in diastolic pressure, resulting in **increased systolic pressure** and the characteristic isolated systolic hypertension commonly seen in the elderly population.

The systolic hypertension induced by aortic stiffening likely contributes to mild concentric left ventricular hypertrophy, another common finding in elderly individuals.

Educational objective:

Aortic compliance progressively decreases with age, especially in those age >65. This leads to an increase in pulse pressure, a slight decrease in diastolic pressure, and the characteristic isolated systolic hypertension commonly seen in the elderly population.

References

- Systolic hypertension.

Physiology	Cardiovascular System	Primary hypertension
Subject	System	Topic

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*A soft S4 is a normal finding in the elderly.

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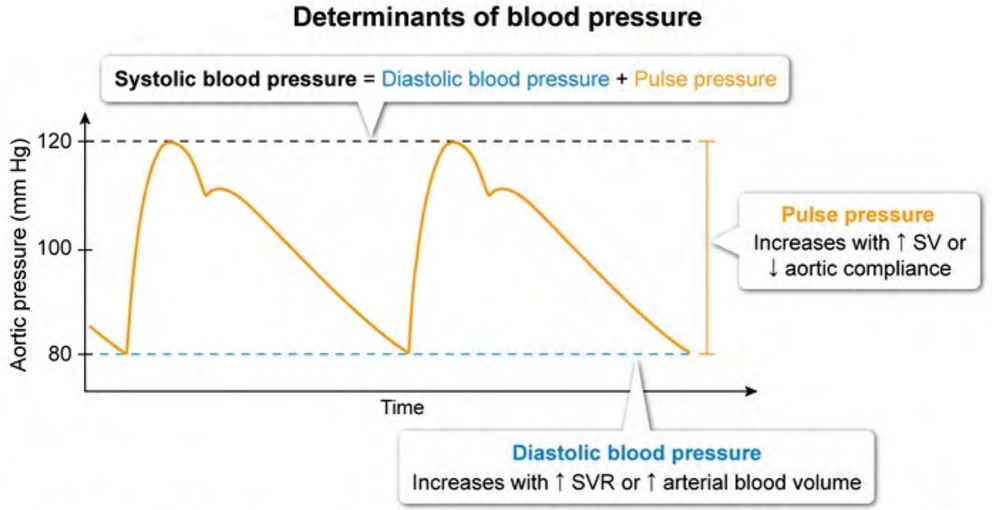

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A 6-year-old boy is evaluated in the office for difficulty hearing. The patient has no ear pain, discharge, or upper respiratory symptoms. Initial testing suggests that he has bilateral sensorineural hearing loss. He has no motor deficits or cerebellar signs. His paternal uncle died suddenly at age 12. Examination of the ears, nose, and throat is normal. ECG shows normal sinus rhythm with a prolonged QT interval (520 msec). Echocardiogram shows normal left and right ventricular function with no significant valvular disease. A genetic defect affecting which of the following is most likely present in this patient?

- A. Calcium channels
- B. Membrane anchoring protein
- C. Potassium channels
- D. Sodium channels
- E. Sodium-potassium ATPase

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- ✗ A. Calcium channels (8%)
- B. Membrane anchoring protein (5%)
- ✓ C. Potassium channels (70%)
- D. Sodium channels (8%)
- E. Sodium-potassium ATPase (6%)

Incorrect
Correct answer
C

70%
Answered correctly

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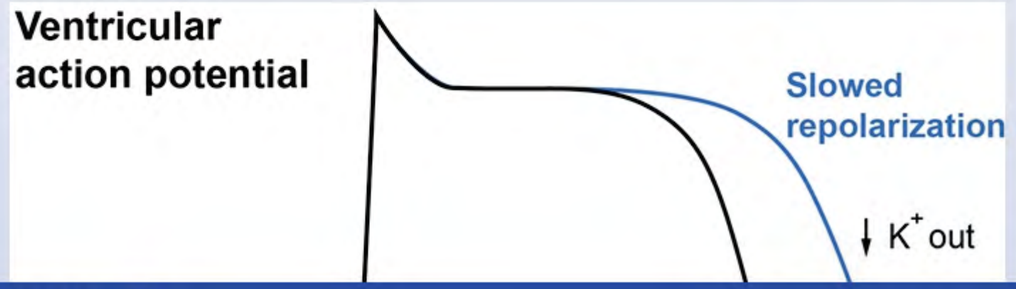
Explanation



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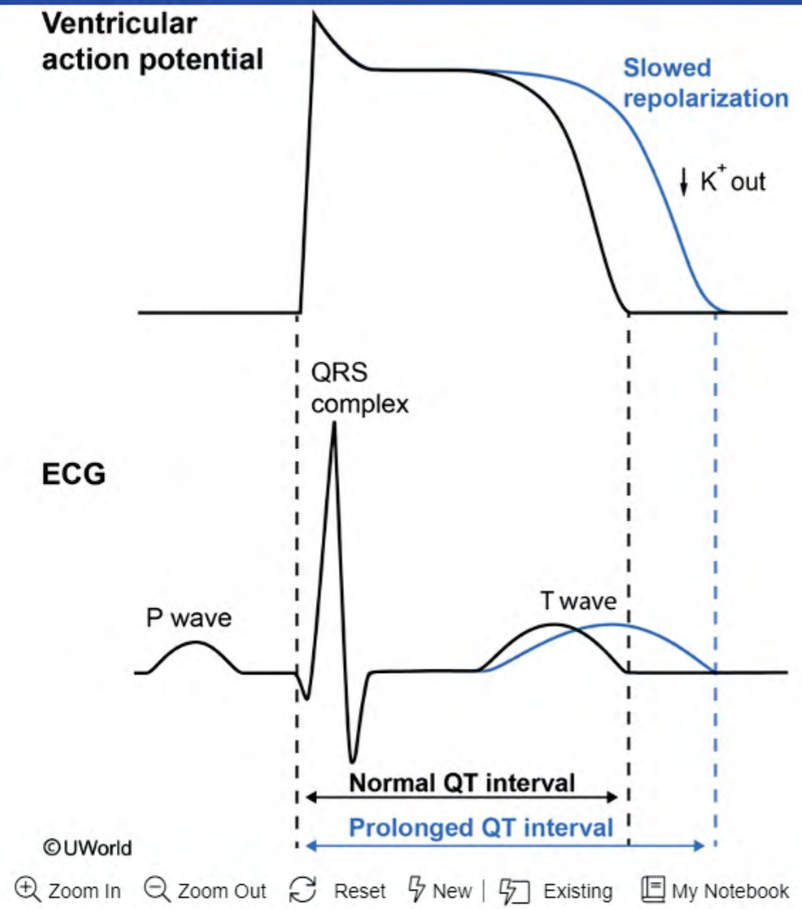

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This patient's clinical presentation is suggestive of **Jervell and Lange-Nielsen syndrome**, an autosomal recessive disorder characterized by profound bilateral **sensorineural hearing loss** and **congenital long QT syndrome**, which predisposes individuals to syncope and sudden cardiac death.

This syndrome occurs secondary to **mutations** in genes (eg, KCNQ1, KCNE1) that encode the alpha and beta subunits of **voltage-gated potassium channels**. These subunits contribute to the slow-acting component of the outward-rectifying potassium current, which is responsible for ventricular repolarization during phase 3 of the cardiac action potential. Mutations in the potassium channel lead to a decrease in potassium current with prolongation of action potential duration and the QT interval. QT interval prolongation predisposes to the development of life-threatening **ventricular arrhythmias**, such as torsades de pointes and ventricular fibrillation.

(Choices A and D) Brugada syndrome is an autosomal dominant condition that can be associated with mutations in cardiac sodium or L-type calcium channels, leading to characteristic ECG changes (eg, pseudo right bundle branch block, ST-segment elevation in leads V1-V3) and an increased risk of ventricular tachyarrhythmias and sudden cardiac death.

(Choice B) Duchenne muscular dystrophy is an X-linked disorder caused by mutations in dystrophin, a structural membrane protein that stabilizes the plasma membrane of myocytes. Patients typically present with progressive proximal muscle degeneration and weakness. Additional features may include cardiomyopathy and conduction abnormalities.

(Choice E) Genetic defects in the sodium-potassium ATPase pump typically are not associated with long QT syndrome. Cardiac glycosides (eg, digoxin) inhibit the sodium-potassium ATPase in myocardial cells, resulting in a mild increase in intracellular sodium and subsequent inhibition of the sodium-calcium exchanger. This leads to increased intracellular calcium and cardiac contractility, which can help reduce symptoms in patients with advanced systolic heart failure.

Educational objective:
Jervell and Lange-Nielsen syndrome is an autosomal recessive disorder characterized by profound bilateral sensorineural hearing loss and congenital long QT syndrome, which predisposes to ventricular arrhythmias and

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Educational objective:
Jervell and Lange-Nielsen syndrome is an autosomal recessive disorder characterized by profound bilateral sensorineural hearing loss and congenital long QT syndrome, which predisposes to ventricular arrhythmias and sudden cardiac death. This condition occurs secondary to mutations in genes that encode voltage-gated potassium channels.

References

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Normal QT interval

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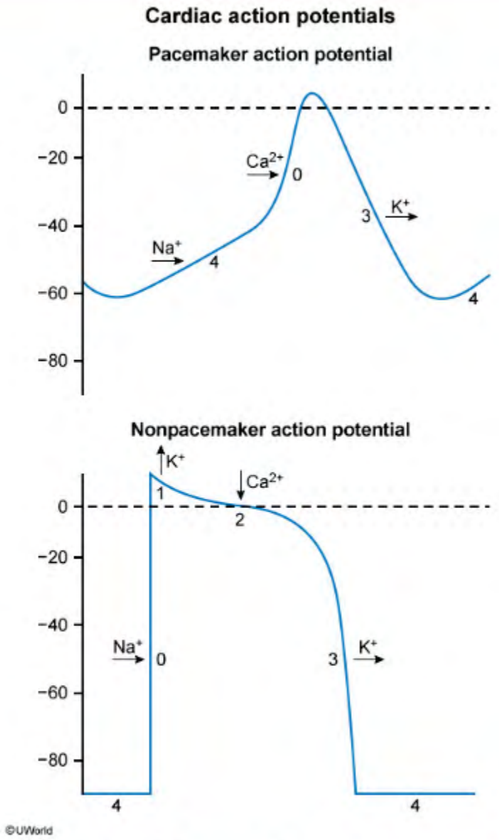

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increased intracellular calcium and cardiac contractility, which can help reduce symptoms in patients with advanced systolic heart failure.

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A 1-hour-old neonate with prenatally diagnosed tetralogy of Fallot is admitted to the neonatal intensive care unit with cyanosis. The mother is a 28-year-old gravida 3 para 2 who received routine prenatal care. The patient was born via spontaneous vaginal delivery at 35 weeks gestation. Initial Apgar scores are 5 and 7 at 1 and 5 minutes after birth, respectively. The neonate's lips, fingers, and toes appear cyanotic. This patient should be immediately treated with a medication producing which of the following effects?

- A. Decreased left ventricular end diastolic pressure
- B. Increased pulmonary surfactant levels
- C. Increased pulmonary vascular resistance
- D. Maintenance of ductus arteriosus patency
- E. Stimulation of fetal hemoglobin production

Submit

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- ✗ A. Decreased left ventricular end diastolic pressure (7%)
- B. Increased pulmonary surfactant levels (7%)
- C. Increased pulmonary vascular resistance (5%)
- ✓ D. Maintenance of ductus arteriosus patency (78%)
- E. Stimulation of fetal hemoglobin production (1%)

Incorrect
Correct answer
D

78%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation

Ductal-dependent congenital heart defects		
Pathophysiology	**Function of ductus arteriosus**	**Type of defect**
Right heart obstruction	Allows blood flow from aorta to pulmonary circulation	• ToF with severe RVOT • Tricuspid atresia • Pulmonary atresia

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Explanation

Ductal-dependent congenital heart defects		
Pathophysiology	**Function of ductus arteriosus**	**Type of defect**
Right heart obstruction	Allows blood flow from aorta to pulmonary circulation	• ToF with severe RVOT • Tricuspid atresia • Pulmonary atresia
Left heart obstruction	Allows blood flow from pulmonary artery to systemic circulation	• Hypoplastic left heart • Critical aortic stenosis • Critical aortic coarctation • Interrupted aortic arch
Parallel circulation	Allows mixing of oxygenated & deoxygenated blood	• Transposition of the great arteries
RVOT = right ventricular outflow tract obstruction; **ToF** = tetralogy of Fallot.		

Tetralogy of Fallot (TOF) is a cyanotic, congenital heart defect characterized by pulmonary stenosis, an overriding aorta, right ventricular hypertrophy, and a ventricular septal defect. When pulmonary stenosis is severe, TOF is a **ductal-dependent** lesion that relies on the patency of the **ductus arteriosus** (DA) for adequate oxygenation.

The DA is a fetal vascular structure that connects the main pulmonary artery and aorta. In utero, blood (which is oxygenated by the placenta) bypasses the lungs by flowing from the pulmonary artery through the DA to the aorta. **After birth**, the lungs fill with oxygen and pulmonary vascular resistance decreases, causing the **direction of flow** through the DA to reverse (ie, from **aorta to pulmonary artery**). DA patency is maintained by circulating prostaglandin released by the placenta; after birth, prostaglandin production stops, and the **DA starts closing**.

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RVOT = right ventricular outflow tract obstruction; **ToF** = tetralogy of Fallot.

Tetralogy of Fallot (TOF) is a cyanotic, congenital heart defect characterized by pulmonary stenosis, an overriding aorta, right ventricular hypertrophy, and a ventricular septal defect. When pulmonary stenosis is severe, TOF is a **ductal-dependent** lesion that relies on the patency of the **ductus arteriosus** (DA) for adequate oxygenation.

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In severe TOF, critical pulmonary stenosis or atresia limits blood flow from the right side of the heart to the pulmonary circulation for oxygenation. Instead, blood reaches the pulmonary vascular bed via retrograde flow from the aorta through the DA. Without flow through the DA, affected neonates are **unable to oxygenate** blood. These patients are **cyanotic** after birth, as in this neonate, and require immediate administration of **prostaglandin E1** to **maintain patency** of the DA.

(Choice A) Heart failure medications that reduce preload (eg, diuretics) decrease left ventricular end diastolic pressure. This decrease would not improve right ventricular outflow tract obstruction or increase retrograde blood flow through the DA in patients with TOF.

(Choice B) Administration of pulmonary surfactant can improve lung compliance in preterm neonates with respiratory distress syndrome. Because the primary pathology in TOF is cardiac rather than pulmonary, surfactant will not improve oxygenation.

(Choice C) Increased pulmonary vascular resistance would worsen the pathophysiology of TOF by decreasing blood flow from the aorta through the DA into the lungs.

(Choice E) Hydroxyurea increases fetal hemoglobin production, thereby improving the oxygen carrying capacity of blood in sickle cell anemia. In TOF, oxygenation is impaired due to inadequate pulmonary blood flow from right

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After birth, the lungs fill with oxygen and pulmonary vascular resistance decreases, causing the **direction of flow** through the DA to reverse (ie, from **aorta to pulmonary artery**). DA patency is maintained by circulating prostaglandin released by the placenta; after birth, prostaglandin production stops, and the **DA starts closing**.

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(Choice C) Increased pulmonary vascular resistance would worsen the pathophysiology of TOF by decreasing blood flow from the aorta through the DA into the lungs.

(Choice E) Hydroxyurea increases fetal hemoglobin production, thereby improving the oxygen carrying capacity of blood in sickle cell anemia. In TOF, oxygenation is impaired due to inadequate pulmonary blood flow from right ventricular outflow tract obstruction.

Educational objective:
Severe tetralogy of Fallot is a ductal-dependent lesion that requires blood flow through the ductus arteriosus (from the aorta to the pulmonary artery) for adequate oxygenation. Prostaglandin E1 prevents closure of the ductus arteriosus and should be immediately administered to all cyanotic neonates with suspected ductal-dependent lesions.

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- Hypoplastic left heart

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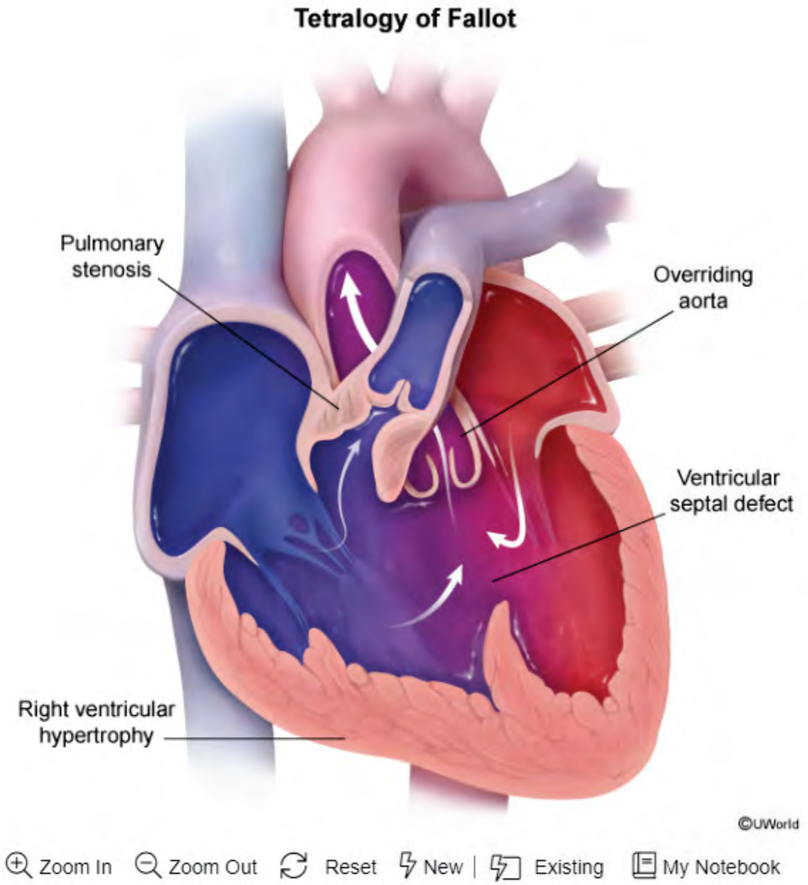

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(Choice B) Administration of pulmonary surfactant can improve lung compliance in preterm neonates with

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Left heart	Allows blood flow from pulmonary	• Hypoplastic left heart • Critical aortic stenosis

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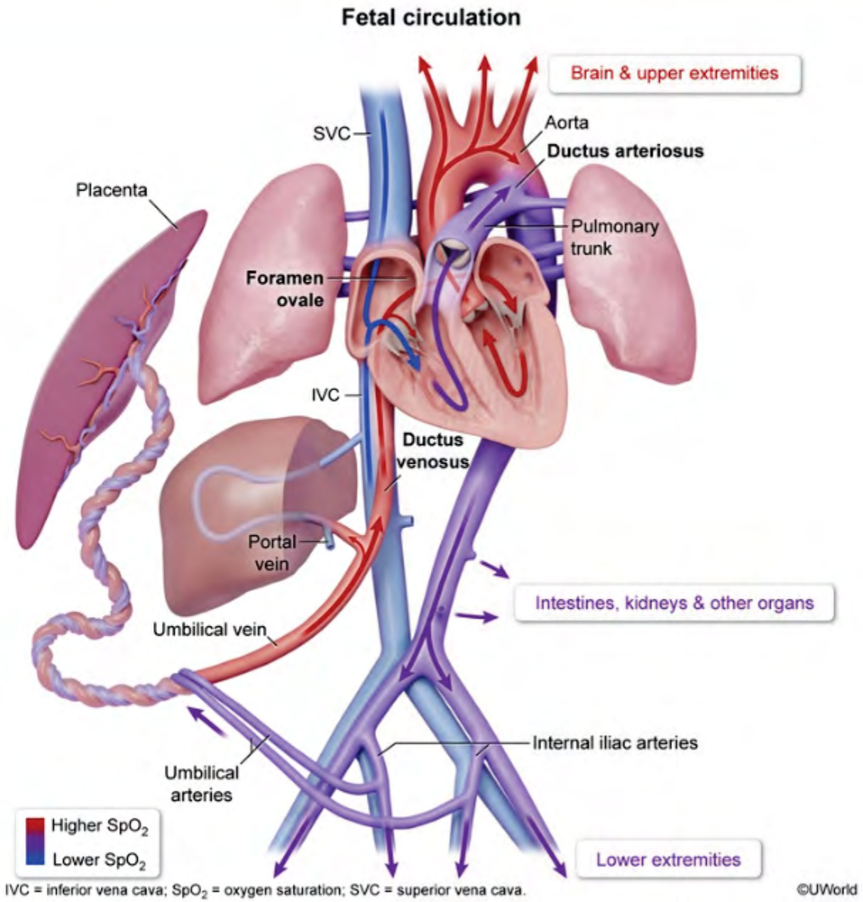

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- Hypoplastic left heart

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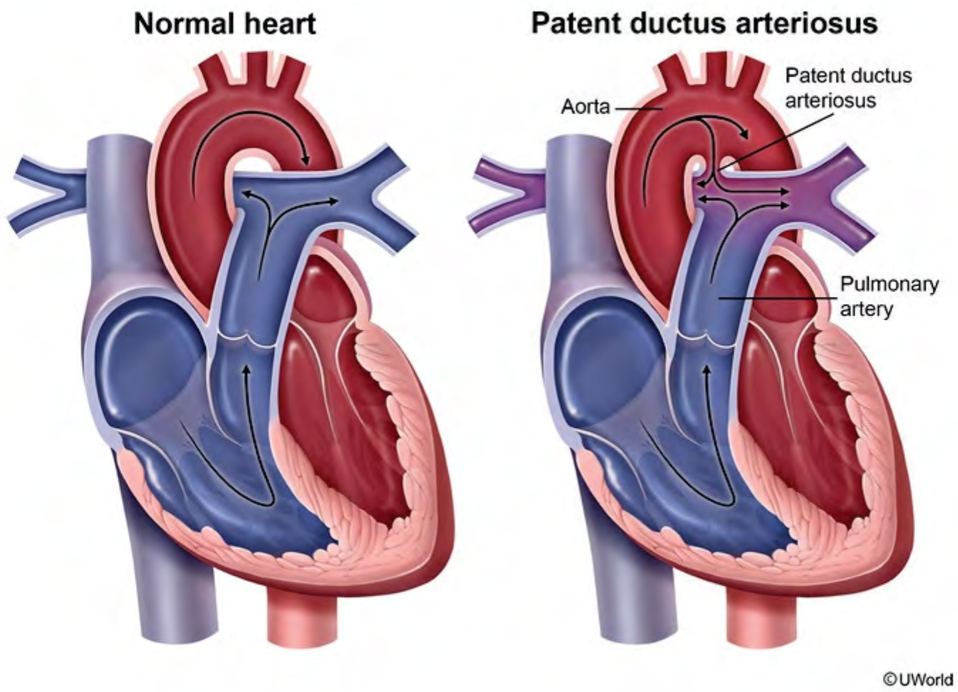

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(Choice B) Administration of pulmonary surfactant can improve lung compliance in preterm neonates with

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A 1-hour-old girl is evaluated in the delivery room. The infant was born at term via spontaneous vaginal delivery following an uncomplicated pregnancy. The mother is gravida 2 para 2 and has no significant medical history. Apgar scores were 9 and 9 at 1 and 5 minutes, respectively. The patient has been skin-to-skin with her mother and breastfed once without difficulty. Physical examination shows a pink, alert infant. Cardiac auscultation reveals a normal S1 and S2 with no murmurs, and femoral pulses are normal. Which of the following hemodynamic changes most likely occurred during the fetal-to-neonatal transition at birth?

	Systemic vascular resistance	Pulmonary vascular resistance	Left ventricular contribution to systemic blood flow
○ A.	↓	↓	↑
○ B.	↓	↑	↑
○ C.	↑	↓	↓
○ D.	↑	↓	↑
○ E.	↑	↑	↓

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A 1-hour-old girl is evaluated in the delivery room. The infant was born at term via spontaneous vaginal delivery following an uncomplicated pregnancy. The mother is gravida 2 para 2 and has no significant medical history. Apgar scores were 9 and 9 at 1 and 5 minutes, respectively. The patient has been skin-to-skin with her mother and breastfed once without difficulty. Physical examination shows a pink, alert infant. Cardiac auscultation reveals a normal S1 and S2 with no murmurs, and femoral pulses are normal. Which of the following hemodynamic changes most likely occurred during the fetal-to-neonatal transition at birth?

	Systemic vascular resistance	Pulmonary vascular resistance	Left ventricular contribution to systemic blood flow	
A.	↓	↓	↑	(11%)
B.	↓	↑	↑	(14%)
✗ C.	↑	↓	↓	(4%)
✓ D.	↑	↓	↑	(65%)
E.	↑	↑	↓	(2%)

Incorrect
Correct answer
D

65%
Answered correctly

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Explanation

Fetal circulation

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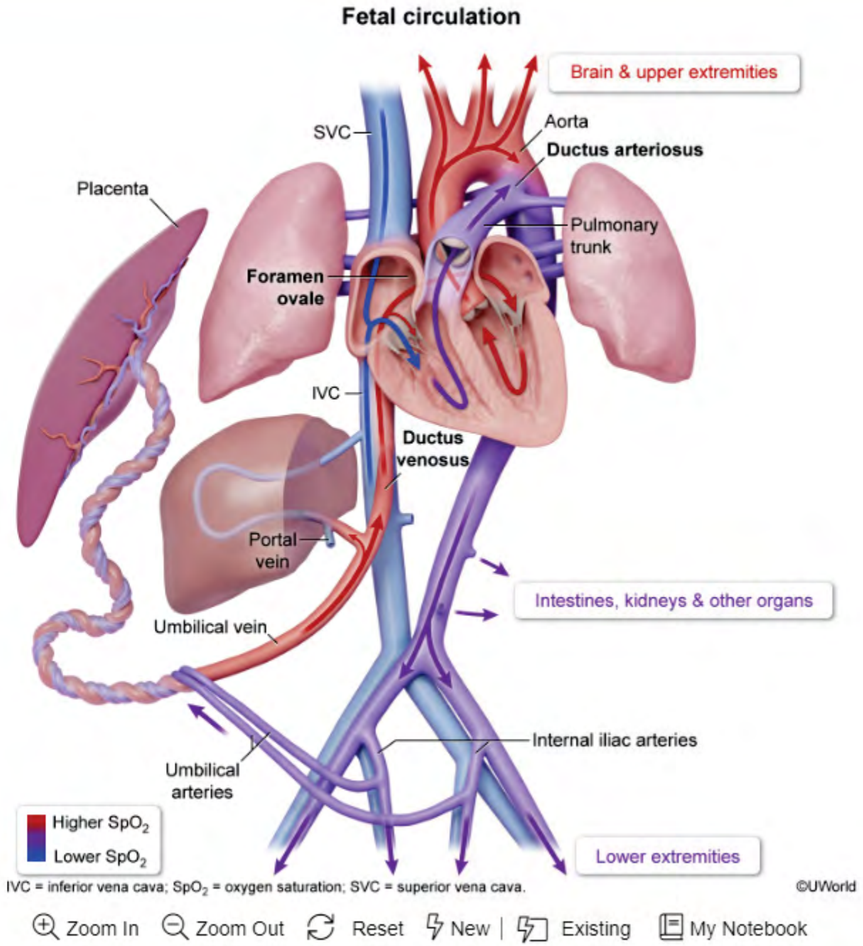

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Fetal circulation is characterized by:

- **High pulmonary vascular resistance** (PVR): In utero, the lungs are filled with amniotic fluid, which causes diffuse vasoconstriction of the pulmonary capillary beds (hypoxic vasoconstriction).
- **Low systemic vascular resistance** (SVR): The fetal systemic circulation is connected in parallel to the low-resistance placental vascular bed via the umbilical arteries (which deliver deoxygenated blood to the placenta) and umbilical vein (which delivers oxygenated blood to the fetus).

In the fetus, PVR is greater than SVR; the foramen ovale and ductus arteriosus allow blood to be shunted away from the high resistance pulmonary circulation into the low resistance systemic circulation (ie, right-to-left shunting), bypassing the lungs. Because the ductus arteriosus is a high-caliber shunt, the **right ventricle** contributes approximately **60 percent** of the fetal systemic blood flow (and the left ventricle contributes the remainder).

At birth, fetal circulation transitions to **neonatal circulation**, which is characterized by:

- **Decreased PVR**: Pulmonary expansion and aeration lead to diffuse pulmonary vasodilation.
- **Increased SVR**: Umbilical cord clamping abruptly removes the low-resistance placenta from neonatal circulation.

Because SVR is now greater than PVR, shunting through the ductus arteriosus reverses to left-to-right shunting (triggering eventual closure of the ductus). With the cessation of right-to-left shunting through the ductus arteriosus at birth, **only the left ventricle** contributes to systemic blood flow.

Educational objective:
Before birth, high pulmonary vascular resistance (PVR) and low systemic vascular resistance (SVR) facilitate right-to-left shunting through the ductus arteriosus, and both ventricles contribute to fetal systemic blood flow. At birth, decreased PVR (due to oxygenation and ventilation of the lungs) and increased SVR (due to umbilical cord clamping) reverse shunting through the ductus arteriosus (ie, left-to-right shunting), and only the left ventricle contributes to systemic blood flow.

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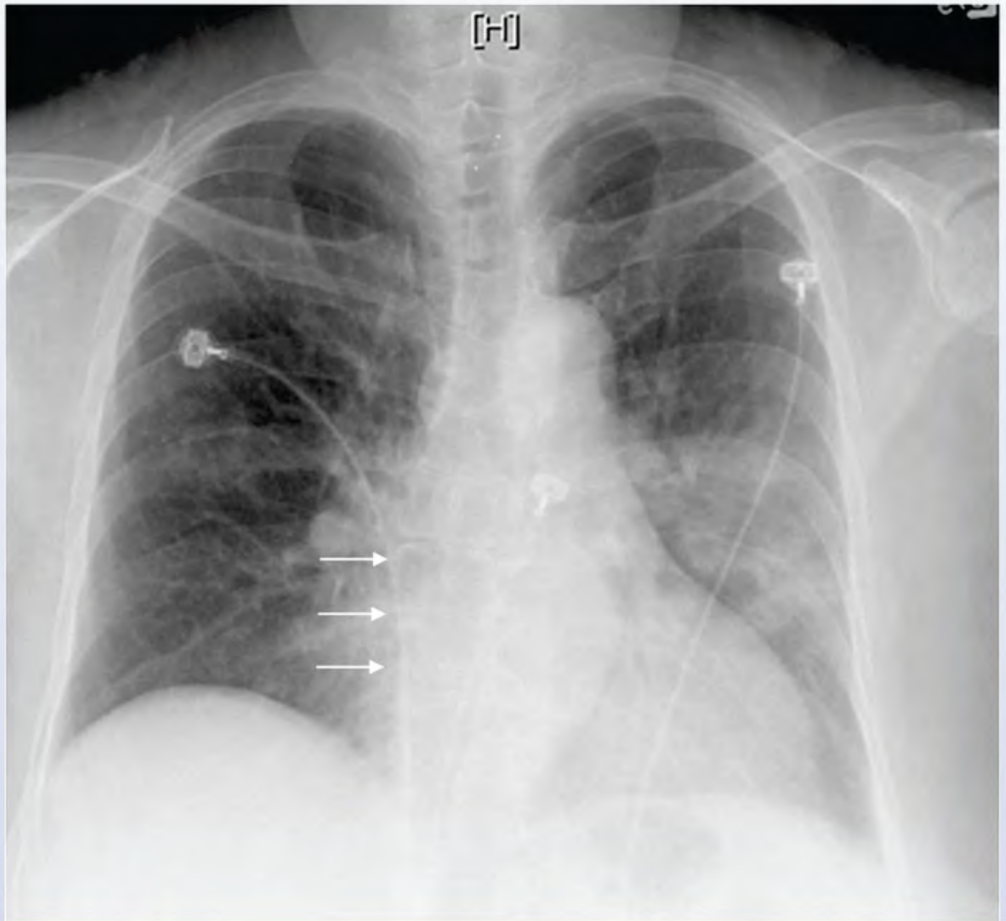
A 62-year-old man comes to the emergency department due to fever, productive cough, and dyspnea for the past several days. His symptoms are becoming progressively worse. The patient has underlying mild chronic obstructive pulmonary disease and has smoked a pack of cigarettes daily for the last 40 years. On examination, temperature is 38.3 C (100.9 F), pulse is 110/min, and respirations are 24/min. Auscultation of the right lung reveals bronchial breath sounds and crackles. Laboratory evaluation shows leukocytosis. Chest x-ray is shown below.



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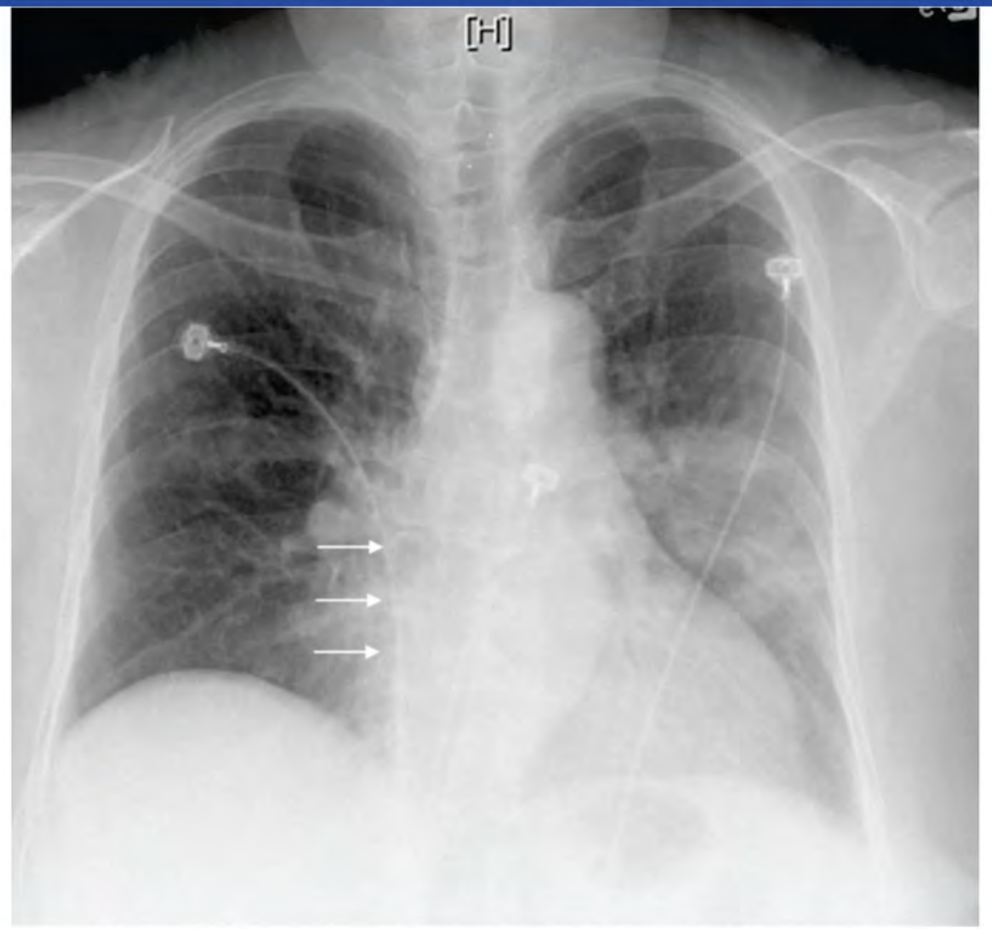

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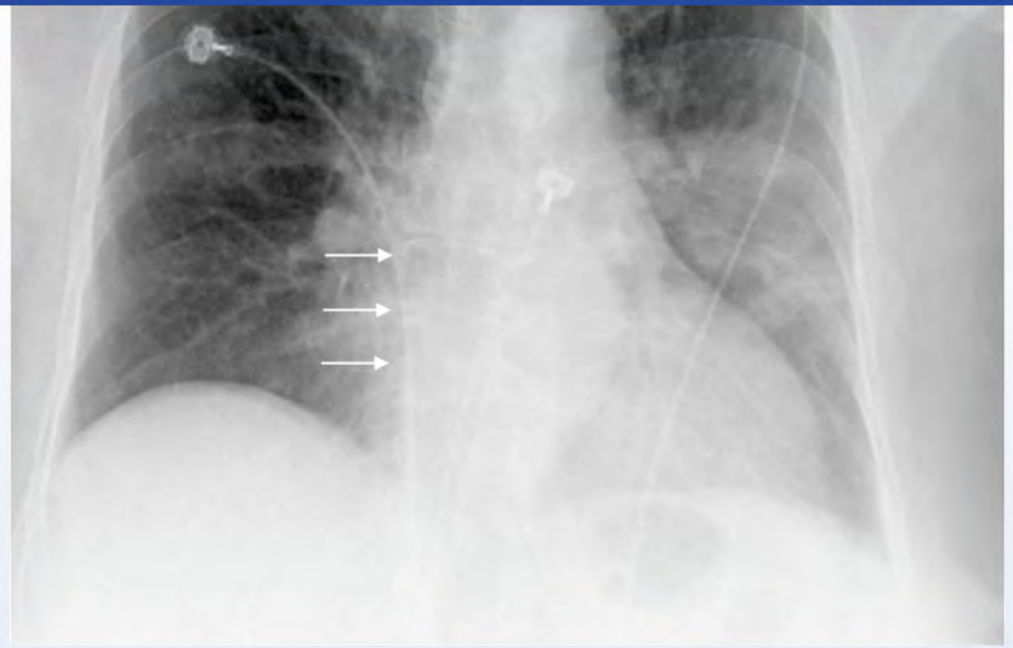

The mediastinal contour obscured by the lung consolidation on this patient's chest x-ray is primarily formed by which of the following structures?

- A. Coronary sinus
- B. Inferior vena cava
- C. Pulmonary artery
- D. Right atrium
- E. Right ventricle
- F. Superior vena cava

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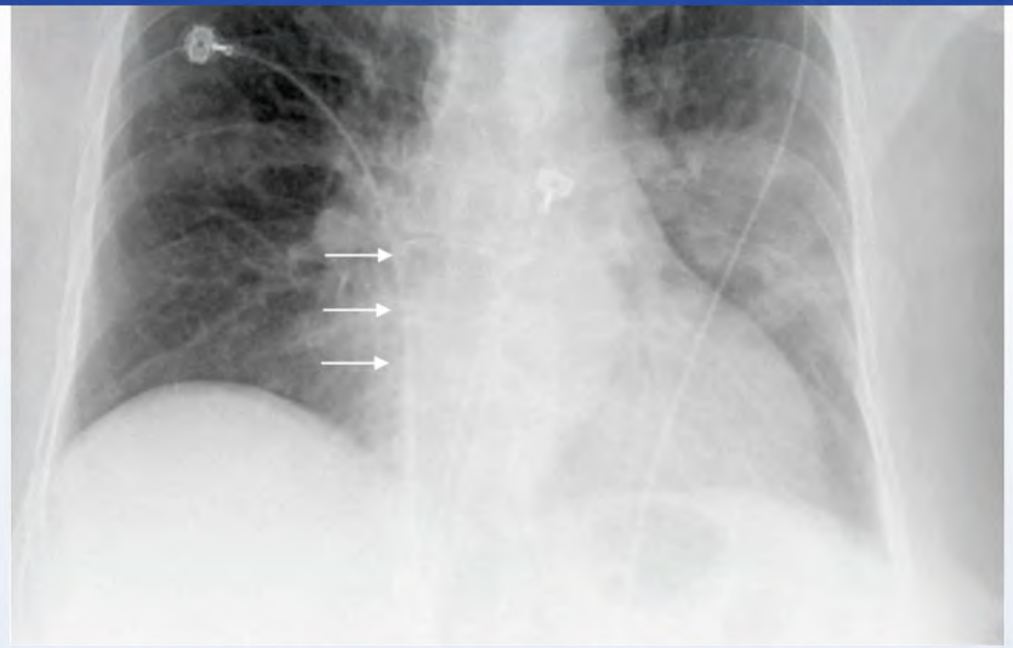

The mediastinal contour obscured by the lung consolidation on this patient's chest x-ray is primarily formed by which of the following structures?

- A. Coronary sinus (0%)
- B. Inferior vena cava (16%)
- C. Pulmonary artery (4%)
- ✓ D. Right atrium (56%)
- ✗ E. Right ventricle (16%)
- F. Superior vena cava (5%)

Incorrect
Correct answer
D

56%
Answered correctly

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E. Right ventricle (16%)

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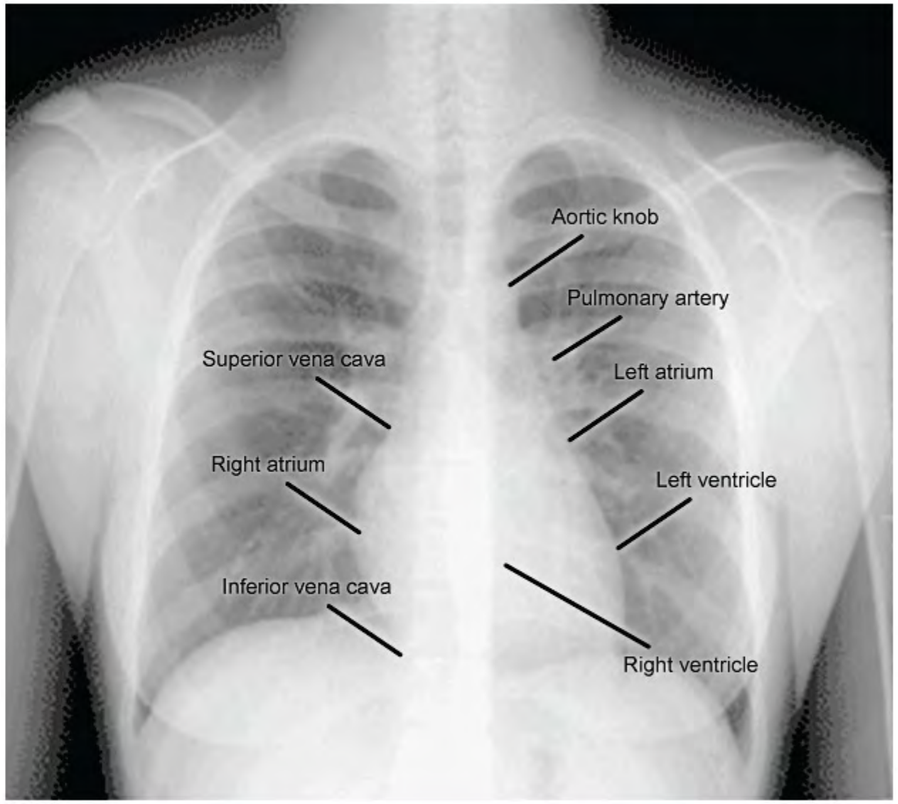
Normal cardiomediastinal contours on chest x-ray



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This patient has acute **lobar pneumonia** with consolidation involving the right middle lobe. The **right middle lobe** of the lung lies immediately adjacent to the **right border of the heart**, and any condition that increases radiographic density in the right middle lobe will obscure the adjacent cardiac silhouette. On posteroanterior (PA) chest x-ray projections, the **right atrium** composes most of the right side of the cardiac silhouette.

(Choice A) The coronary sinus receives most of the venous drainage from the cardiac circulation. It courses posteriorly around the heart in the coronary sulcus to empty directly into the right atrium. It is difficult to visualize on standard chest x-ray.

(Choice B) The inferior vena cava empties into the right atrium in the thorax. It composes only the most inferior edge of the right border of the cardiac silhouette.

(Choice C) The pulmonary artery can be seen on PA chest x-ray on the left side of the cardiomediastinal silhouette just below the aortic arch.

(Choice E) The right ventricle forms the anterior wall of the heart and is best seen on lateral chest x-ray.

(Choice F) The superior vena cava is formed behind the first costal cartilage by the confluence of the right and left brachiocephalic veins. On PA chest x-ray, it is the flattened opacity parallel to the vertebral column that terminates inferiorly at the right atrium.

Educational objective:
On posteroanterior chest x-ray, the right middle lobe is seen adjacent to the right border of the heart, which is primarily formed by the right atrium. Consolidation in the right middle lobe can obscure the x-ray silhouette of the right heart border.

Anatomy	Cardiovascular System	Community acquired pneumonia
Subject	System	Topic

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A 64-year-old man is brought to the emergency department due to severe chest pain, diaphoresis, and shortness of breath. Symptoms began suddenly on awakening and have worsened over the past 2 hours. Blood pressure is 150/90 mm Hg and pulse is 102/min. Physical examination reveals an S4. ECG shows ST-segment depression and T-wave inversion in the lateral leads. Coronary angiography reveals a ruptured atherosclerotic plaque, with a thrombus in the left circumflex artery that causes near-total occlusion. Normal endothelial cells surrounding the lesion have released large amounts of a chemical substance that help decrease thrombus propagation by inhibiting platelet aggregation. Which of the following substances was most likely secreted by these endothelial cells?

- A. Hageman factor
- B. Kallikrein
- C. Prostacyclin
- D. Protein C
- E. Serotonin
- F. Thromboxane A2

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- ✗ A. Hageman factor (5%)
- B. Kallikrein (6%)
- ✓ C. Prostacyclin (50%)
- D. Protein C (17%)
- E. Serotonin (1%)
- F. Thromboxane A2 (17%)

Incorrect
Correct answer
C

50%
Answered correctly

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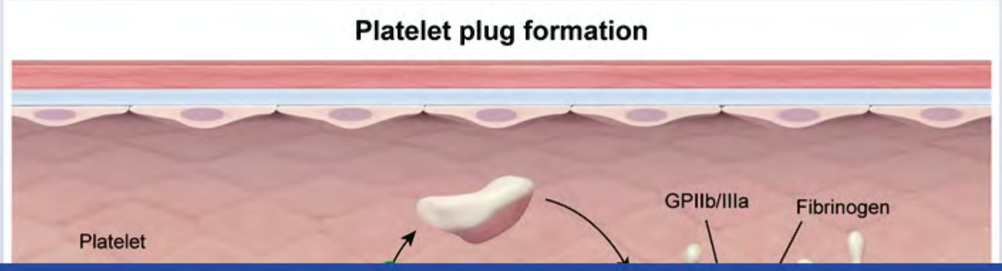
Explanation



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E. Serotonin (1%)

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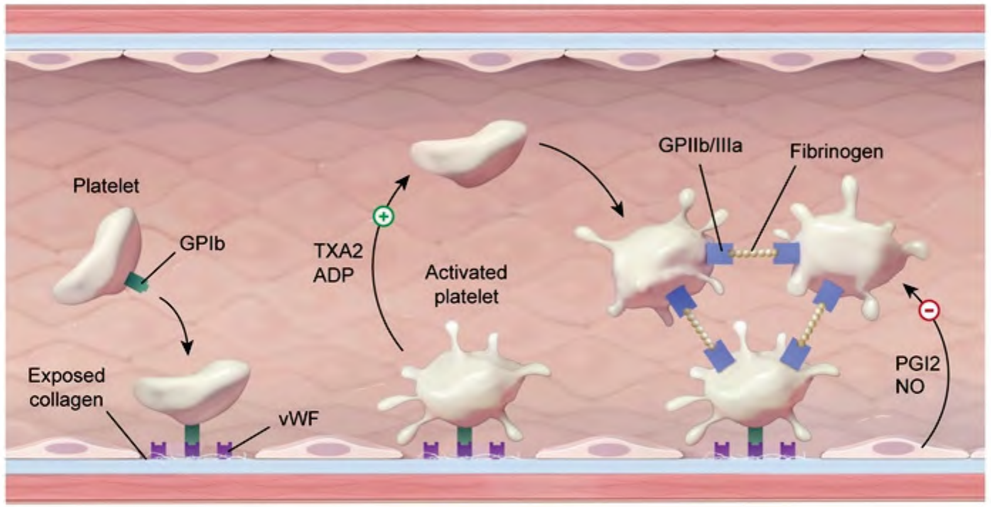
Platelet plug formation



Platelet adherence → Platelet activation & secretion → Platelet aggregation

GP = glycoprotein; **NO** = nitric oxide; **PGI2** = prostaglandin I2 (prostacyclin); **TXA2** = thromboxane A2; **vWF** = von Willebrand factor.

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endothelium; it occurs in 3 steps:

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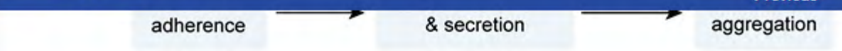

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The formation of a **platelet plug** (primary hemostasis) is essential for preventing bleeding after damage to vascular endothelium; it occurs in 3 steps:

- Platelet **adhesion** takes place via von Willebrand factor acting as a connector that binds platelets to underlying collagen
- Platelets become **activated** and secrete multiple substances, including adenosine diphosphate, ionized calcium, and fibrinogen, from their alpha and delta (dense) granules.
- Thromboxane A2 **(Choice F)** is released and acts as a vasoconstrictor and potent stimulator of platelet **aggregation**. Adenosine diphosphate also stimulates platelet aggregation.

Balance is required as excessive platelet plug formation can lead to a pathologic thrombus that restricts blood flow (eg, myocardial infarction). To oppose the functions of thromboxane A2, the endothelium secretes **prostacyclin** (prostaglandin I2), which is derived from arachidonic acid and synthesized from prostaglandin H2 by prostacyclin synthase. Once secreted, prostacyclin acts locally to **inhibit platelet aggregation** and adhesion to the vascular endothelium and to cause **vasodilation**. Nitric oxide aids in these functions as well. Atherosclerosis can impair the ability of endothelial cells to synthesize prostacyclin and nitric oxide, creating localized predisposition to excessive platelet thrombus formation.

A synthetic prostacyclin, epoprostenol, is used in the treatment of pulmonary hypertension, peripheral vascular disease, and Raynaud syndrome.

(Choice A) Hageman factor (factor XII) is synthesized by the liver and is activated by exposed collagen following damage to endothelial cells. It participates in secondary hemostasis by activating the intrinsic clotting pathway; it also activates fibrinolysis.

(Choice B) Kallikrein converts kininogen into bradykinin. It also may play a role in triggering the fibrinolytic pathway, but it does not inhibit platelet aggregation.

(Choice D) Protein C is a vitamin K–dependent factor synthesized by the liver. It regulates the coagulation cascade by inactivating factors Va and VIIIa.

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(Choice D) Protein C is a vitamin K–dependent factor synthesized by the liver. It regulates the coagulation cascade by inactivating factors Va and VIIIa.

(Choice E) Serotonin has a wide range of functions, including mediation of mood (in the central nervous system) and gastrointestinal motility. It is also released from the delta granules of activated platelets and facilitates localized vasoconstriction.

Educational objective:
Prostacyclin (prostaglandin I2) is synthesized from prostaglandin H2 by prostacyclin synthase in vascular endothelial cells. Once secreted, it inhibits platelet aggregation and causes vasodilation to oppose the functions of thromboxane A2 and help maintain vascular homeostasis.

References

- Continuing education course #2: current understanding of hemostasis

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- Platelets become **activated** and secrete multiple substances, including adenosine diphosphate, ionized ca
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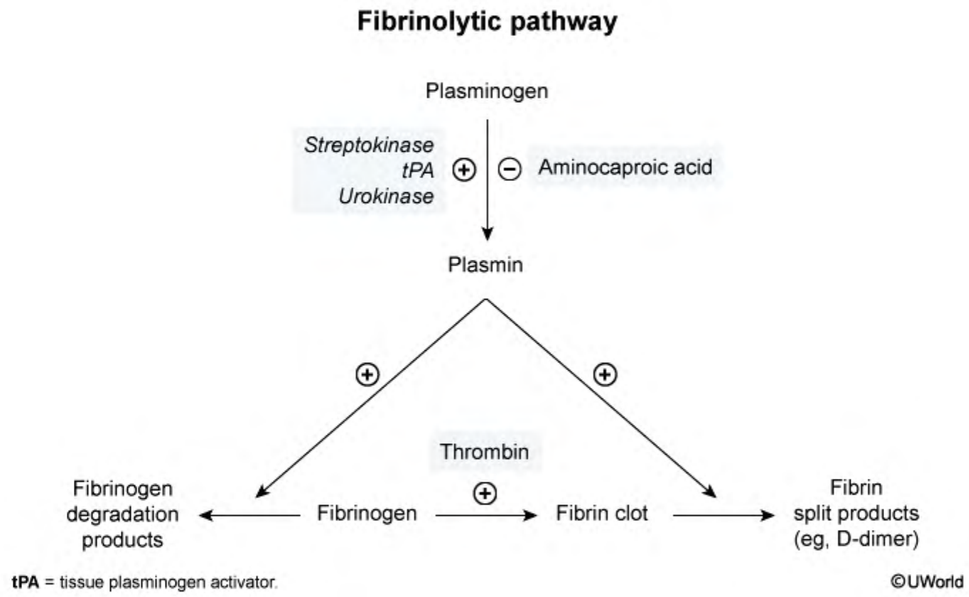

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A 72-year-old man comes to the emergency department due to left-sided weakness and speech difficulty upon awakening this morning. Medical history includes hypertension and persistent atrial fibrillation. His anticoagulant medication was stopped 6 months ago after an episode of gastrointestinal bleeding. Blood pressure is 160/90 mm Hg and pulse is 88/min and irregular. Examination shows mild dysarthria, left lower facial weakness, and left hemiparesis. CT scan of the head shows no evidence of intracranial bleeding. Transthoracic echocardiogram shows left atrial enlargement, normal left ventricular systolic function, and mild mitral regurgitation. The patient's symptoms are determined to be caused by a thromboembolic event. Which of the following is the most likely source of the thrombus?

- A. Crista terminalis
- B. Left atrial appendage
- C. Left ventricular apex
- D. Mitral valve
- E. Pulmonary veins
- F. Right atrial appendage
- G. Sinus of Valsalva

Submit

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- ✗ A. Crista terminalis (1%)
- ✓ B. Left atrial appendage (78%)
- C. Left ventricular apex (1%)
- D. Mitral valve (11%)
- E. Pulmonary veins (2%)
- F. Right atrial appendage (2%)
- G. Sinus of Valsalva (0%)

Incorrect
Correct answer
B

78%
Answered correctly

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Explanation

Atrial fibrillation with thrombus formation

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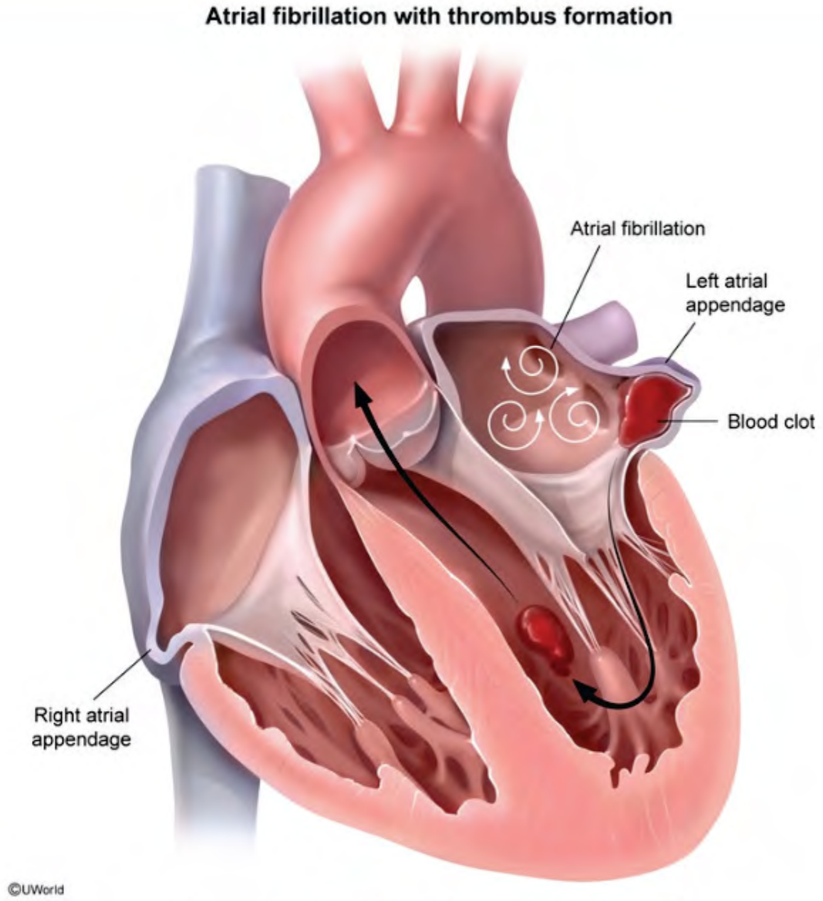

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Atrial fibrillation is associated with significant risk of **systemic thromboembolism**; therefore, long-term anticoagulation is often recommended in patients without any confounding contraindications (eg, recent gastrointestinal bleeding). Several factors contribute to thrombus development in atrial fibrillation, including left atrial enlargement, blood stasis due to ineffective atrial contraction, and atrial inflammation and fibrosis (exerts a procoagulant effect).

The **left atrial appendage** is a small saclike structure in the left atrium that is particularly susceptible to thrombus formation. Approximately 90% of left atrial thrombi are found within the left atrial appendage in patients with nonvalvular atrial fibrillation. These clots can then systemically embolize and lead to stroke (as in this patient), acute limb ischemia, or acute mesenteric ischemia.

(Choice A) The crista terminalis refers to a thick band of atrial muscle that separates the smooth sinus venosus from the right atrial appendage and atrium proper. It is the site of origin of the atrial pectinate muscles and is not a significant region of thrombus formation in atrial fibrillation.

(Choice C) A left ventricular (LV) apical thrombus poses risk of systemic embolization and stroke, but it typically only develops in patients with LV aneurysm or severe LV systolic dysfunction. It is not associated with atrial fibrillation.

(Choice D) Prosthetic valve thrombosis can occur with bioprosthetic or mechanical mitral valves in patients without adequate anticoagulation. Thrombus formation on a native mitral valve is extremely rare, especially when valve function is normal or near normal.

(Choices E and G) Thrombosis of the pulmonary veins or sinus of Valsalva (aortic sinus) is rare and not associated with atrial fibrillation.

(Choice F) Thrombus formation can occur in the right atrial appendage with atrial fibrillation, though less frequently than in the left atrial appendage. A right atrial thrombus poses risk of embolization to the pulmonary (rather than systemic) circulation, resulting in pulmonary embolism.

Educational objective:
Atrial fibrillation is associated with increased risk of systemic thromboembolism. The left atrial appendage is the most common site of thrombus formation in atrial fibrillation.

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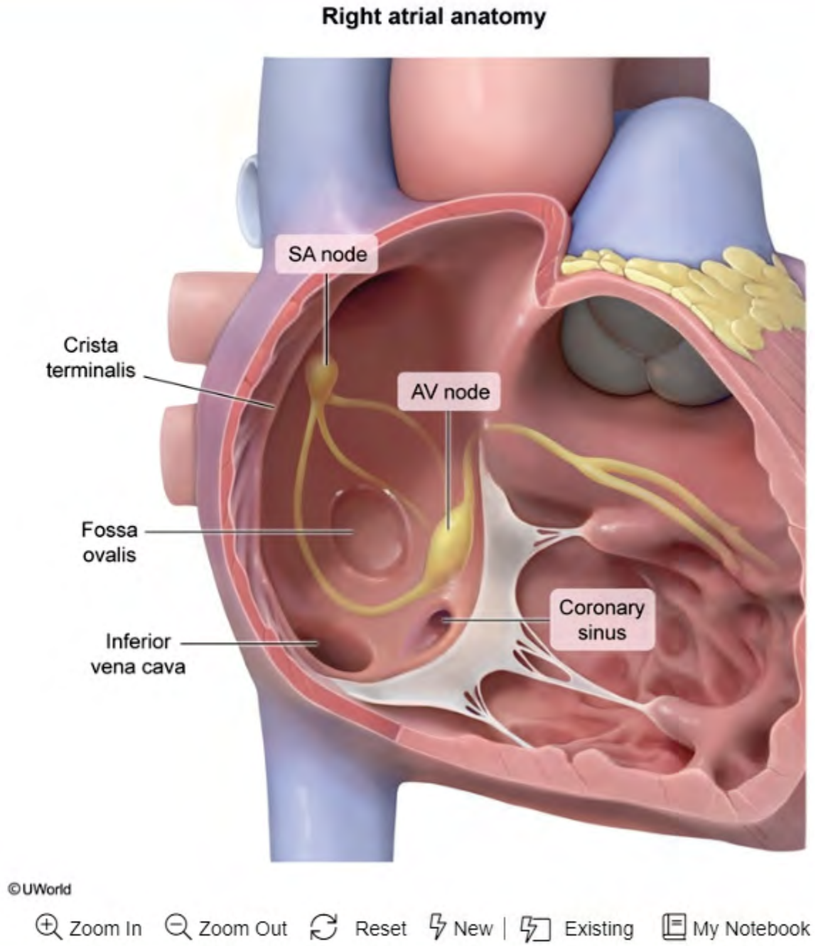

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Educational objective:
Atrial fibrillation is associated with increased risk of systemic thromboembolism. The left atrial appendage is the most common site of thrombus formation in atrial fibrillation.

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A 43-year-old man is being evaluated for occasional retrosternal chest pressure that develops with moderate exertion and sometimes occurs when resting. He does not use alcohol, tobacco, or illicit drugs. The patient has an extensive family history of coronary artery disease. His temperature is 36.7 C (98 F), blood pressure is 124/72 mm Hg, pulse is 81/min, and respirations are 14/min. Physical examination shows no abnormalities. Coronary angiography shows mild luminal irregularities but no significant obstructive lesions. Acetylcholine infusion during the procedure results in dilation of epicardial coronary vessels. A reaction involving which of the following amino acids is most likely responsible for the observed dilation?

- A. Arginine
- B. Aspartate
- C. Glutamate
- D. Tryptophan
- E. Tyrosine

Submit

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- ✓ A. Arginine (55%)
- B. Aspartate (4%)
- C. Glutamate (17%)
- D. Tryptophan (7%)
- E. Tyrosine (15%)

Correct

55% Answered correctly

03 secs Time Spent

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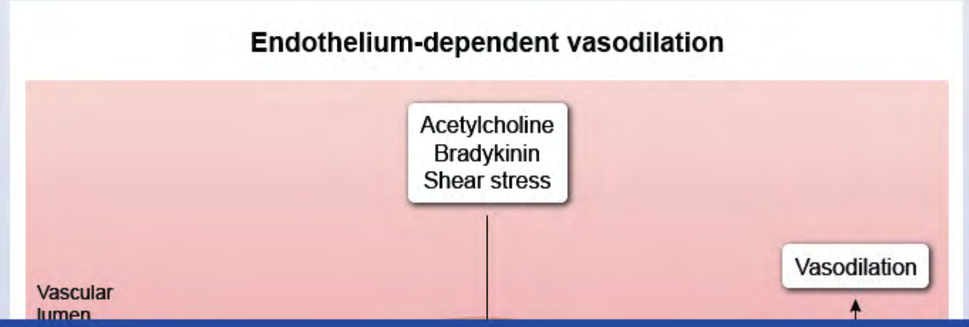
Explanation



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A 43-year-old man is being evaluated for occasional retrosternal chest pressure that develops with moderate exertio
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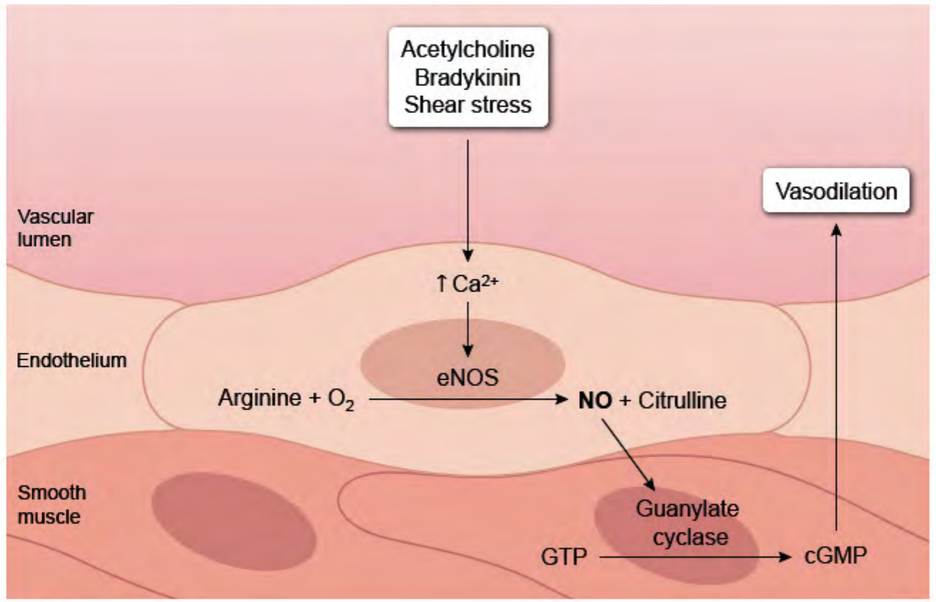
Endothelium-dependent vasodilation



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Vasodilation
Vascular lumen

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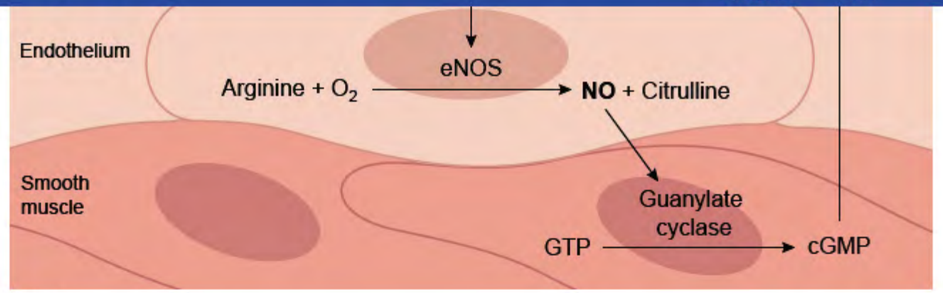

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The **vascular endothelium** plays an important role in vasodilation mediated by acetylcholine, bradykinin, serotonin, substance P, and shear forces. These stimuli activate specific membrane receptors present on endothelial cells, leading to an increase in cytosolic calcium levels. This causes activation of **endothelial nitric oxide synthase** (eNOS), which synthesizes **nitric oxide** from arginine, NADPH, and O_2. Nitric oxide then diffuses into the adjacent smooth muscle cells, where it activates guanylyl cyclase and increases formation of **cyclic GMP**. High levels of cyclic GMP activate protein kinase G, which causes a reduction in cytosolic calcium levels and **relaxation** of vascular smooth muscle cells.

The availability of arginine for synthesizing nitric oxide depends on several factors, including exogenous food intake, endogenous synthesis, intracellular storage and degradation, and the presence of asymmetrical dimethylarginine (an endogenous analog of arginine that works as a competitive inhibitor of eNOS).

Educational objective:
Nitric oxide is synthesized from arginine by nitric oxide synthase. As a precursor of nitric oxide, arginine supplementation may play an adjunct role in the treatment of conditions that improve with vasodilation, such as stable angina.

References

- Randomized trial of a medical food for the dietary management of chronic, stable angina.

Physiology	Cardiovascular System	Nitric oxide
Subject	System	Topic

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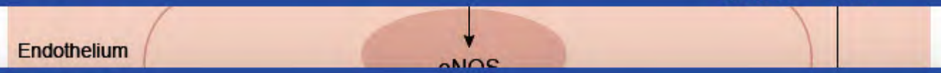

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Important amino acid derivatives

Precursor amino acids	Substance
Arginine	• Nitric oxide
Arginine + Aspartate	• Urea
Glycine + Succinyl CoA	• Heme
Glycine + Arginine + SAM	• Creatine
Glutamate	• GABA • Glutathione
Glutamine + Aspartate	• Pyrimidines
Glutamine + Aspartate + Glycine	• Purines
Histidine	• Histamine
Tyrosine	• Dopamine • Epinephrine • Norepinephrine • Thyroxine • Melanin
Tryptophan	• Serotonin • Melatonin • Niacin

GABA = gamma-aminobutyric acid; **SAM** = S-adenosylmethionine.

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A 17-year-old girl is evaluated due to decreased exercise tolerance and fatigue. She has no known medical conditions and takes no medications. Blood pressure is 110/60 mm Hg and pulse is 88/min. The lungs are clear on auscultation. Cardiac examination reveals a parasternal heave, and a widely split S2 is heard without variation throughout the respiratory cycle. A systolic ejection murmur is present at the left upper sternal border. Distal pulses are normal. Echocardiography of this patient is most likely to demonstrate which of the following findings?

	Right atrial cavity size	Right ventricular cavity size	Left atrial cavity size	Left ventricular cavity size
○ A.	Increased	Increased	Increased	Increased
○ B.	Increased	Increased	Normal	Normal
○ C.	Increased	Normal	Normal	Normal
○ D.	Normal	Normal	Increased	Increased
○ E.	Normal	Normal	Increased	Normal

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A 17-year-old girl is evaluated due to decreased exercise tolerance and fatigue. She has no known medical conditions and takes no medications. Blood pressure is 110/60 mm Hg and pulse is 88/min. The lungs are clear on auscultation. Cardiac examination reveals a parasternal heave, and a widely split S2 is heard without variation throughout the respiratory cycle. A systolic ejection murmur is present at the left upper sternal border. Distal pulses are normal. Echocardiography of this patient is most likely to demonstrate which of the following findings?

	Right atrial cavity size	Right ventricular cavity size	Left atrial cavity size	Left ventricular cavity size	
A.	Increased	Increased	Increased	Increased	(7%)
✓ B.	Increased	Increased	Normal	Normal	(67%)
C.	Increased	Normal	Normal	Normal	(10%)
D.	Normal	Normal	Increased	Increased	(7%)
E.	Normal	Normal	Increased	Normal	(4%)

Correct

67% Answered correctly

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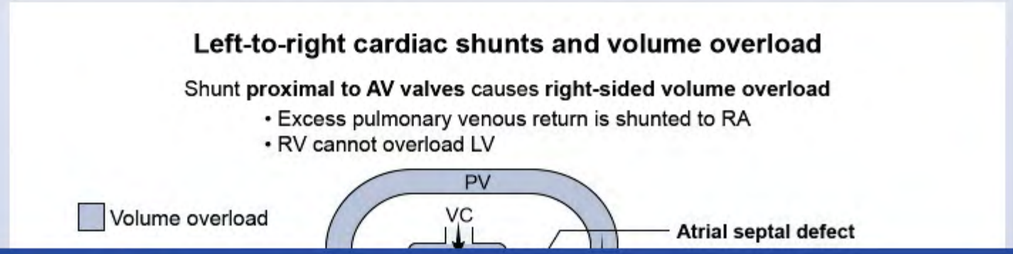

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A 17-year-old girl is evaluated due to decreased exercise tolerance and fatigue. She has no known medical condition on aus through pulses

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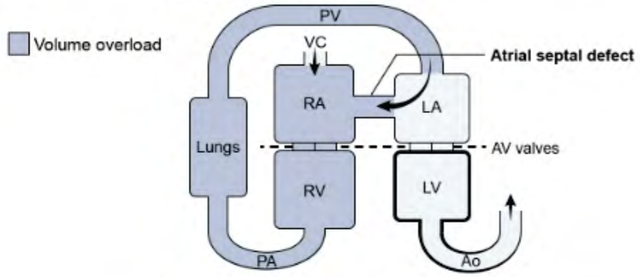
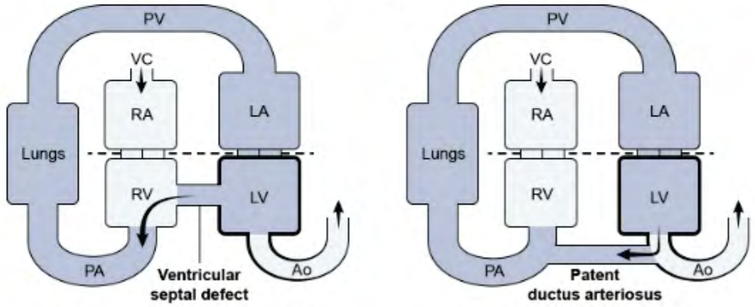
Left-to-right cardiac shunts and volume overload

Shunt **proximal to AV valves** causes **right-sided volume overload**
- Excess pulmonary venous return is shunted to RA
- RV cannot overload LV



Shunt **distal to AV valves** causes **left-sided volume overload**
- Excess pulmonary venous return must flow through LV
- LV can overload itself when given low-resistance pathway



Ao = aorta; **AV** = atrioventricular; **LA** = left atrium; **LV** = left ventricle; **PA** = pulmonary artery; **PV** = pulmonary vein; **RA** = right atrium; **RV** = right ventricle; **VC** = vena cava.

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• Excess pulmonary venous return is shunted to RA
• RV cannot overload LV
Volume overload
PV
VC
Atrial septal defect

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This patient's **widely split S2** that does not vary with the respiratory cycle (ie, **fixed**) and systolic ejection murmur at the left upper sternal border (ie, pulmonic flow murmur) are characteristic of an **atrial septal defect** (ASD). ASD creates left-to-right shunting of blood from the left atrium to the right atrium, increasing blood volume in the right atrium (ie, venous return from the vena cava plus blood delivered via the shunt) and causing right-sided volume overload. Over time, this leads to **right atrial** and **right ventricular enlargement**.

Pulmonary arterial blood flow is also increased with ASD, but **left-sided volume overload does not occur** because additional blood volume returning to the left atrium escapes back to the right atrium without affecting the LV. Most patients with ASD remain asymptomatic until adolescence or adulthood but can eventually have signs and symptoms of right-sided heart failure (eg, dyspnea, fatigue, parasternal heave). Severe pulmonary hypertension and Eisenmenger syndrome can also sometimes develop with ASD left unrepaired for many years.

In contrast to ASD, a left-to-right shunt distal to the atrioventricular valves (ie, ventricular septal defect, patent ductus arteriosus) can cause prominent pulmonary overcirculation that, due to the absence of ASD, must be accomodated by the left atrium and LV; as such, these shunts initially present with left-sided (rather than right-sided) volume overload. In addition, pulmonary pressures are often higher with VSD and PDA because these defects expose the pulmonary circulation to higher systemic-level arterial pressure. As such, they frequently cause symptoms much earlier in life compared to ASD, and the risk for severe pulmonary hypertension and Eisenmenger syndrome is greater than with ASD.

Educational objective:

Atrial septal defects cause left-to-right atrial shunting and right-sided volume overload with enlargement of the right atrium and right ventricle; left-sided volume overload does not occur as excess flow entering the left atrium cycles back into the right atrium through the septal defect. In contrast, left-to-right shunts distal to the atrioventricular valves (eg, ventricular septal defect, patent ductus arteriosus) initially cause left-sided volume overload.

References

- Atrial septal defect.
- Pathophysiology and natural history of atrial septal defect.

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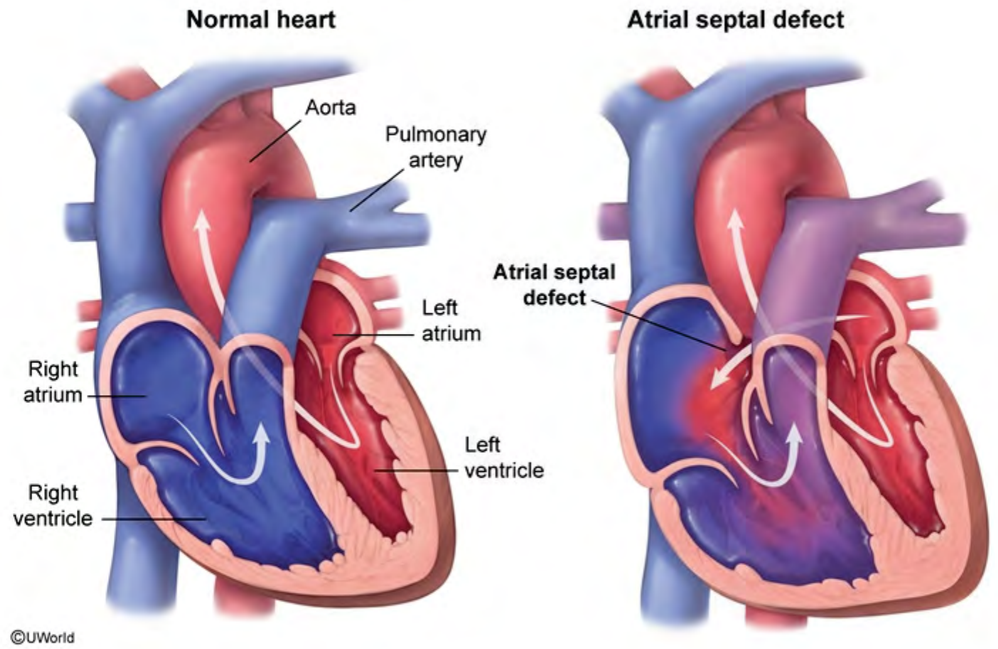

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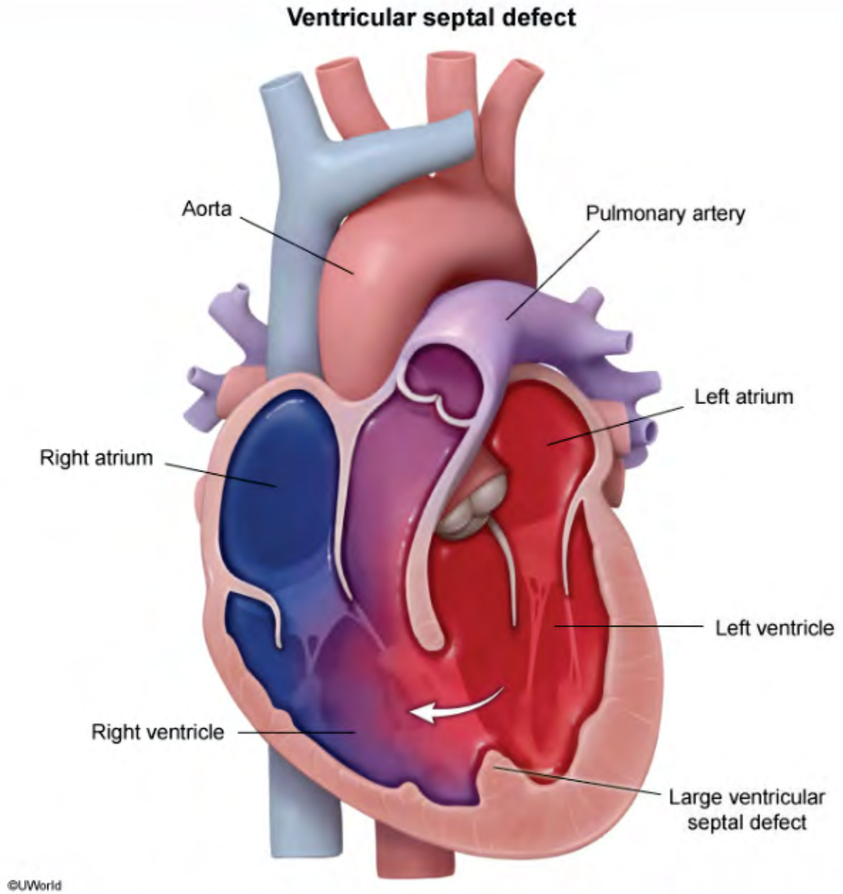

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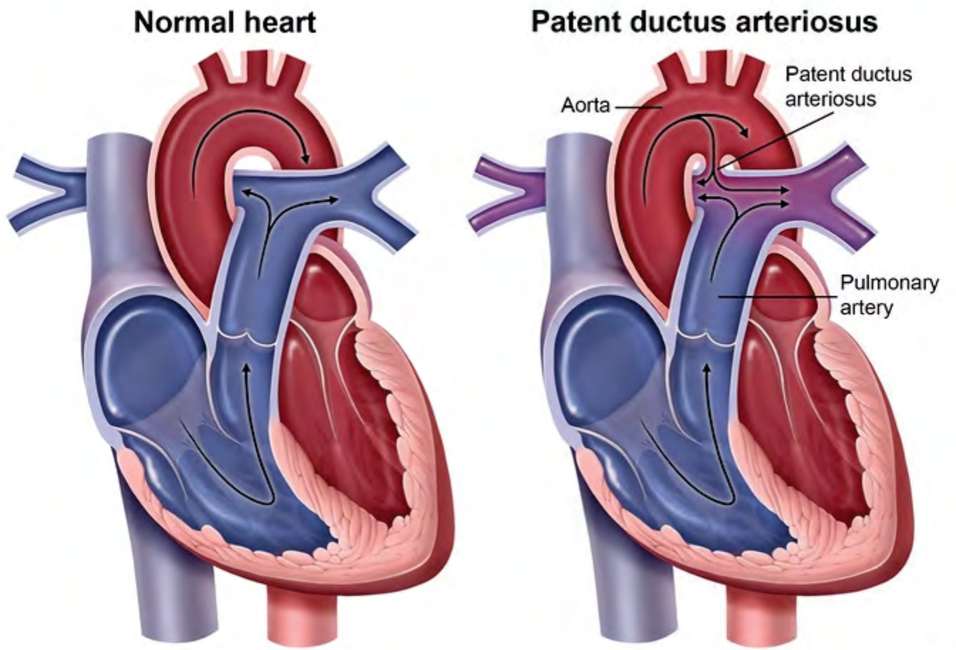

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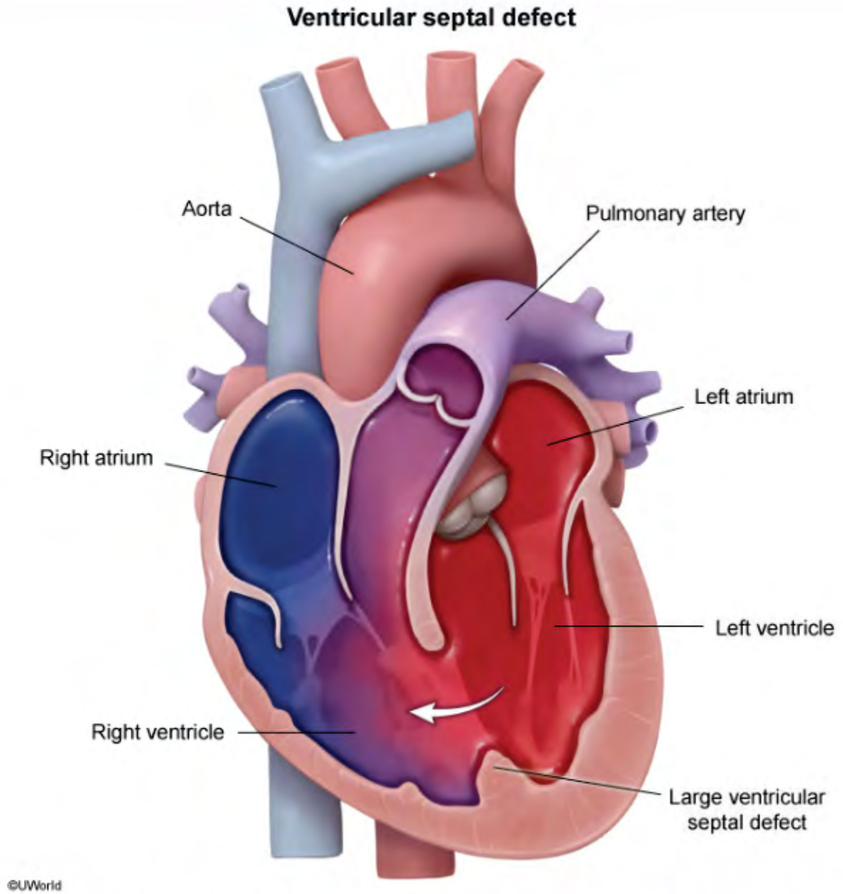

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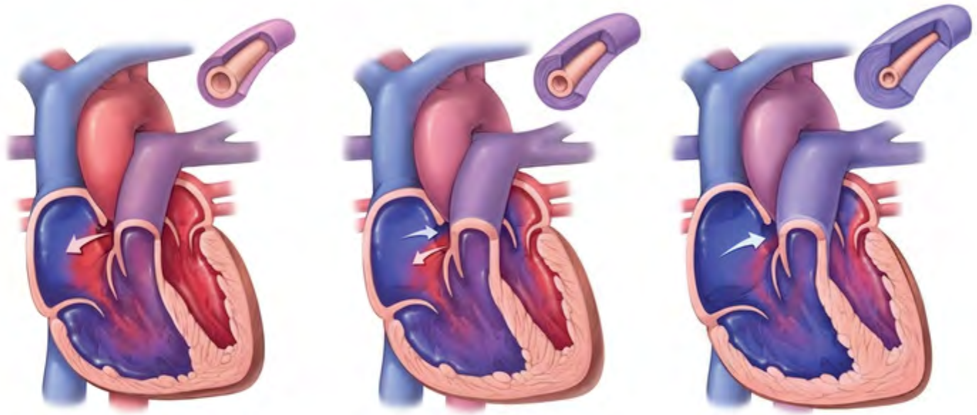
Eisenmenger syndrome



Congenital defect (eg, ASD) allows left-to-right shunt

Over time, ↑ PVR leads to bidirectional flow

PVR continues to increase with eventual continuous right-to-left shunt (Eisenmenger syndrome)

ASD = atrial septal defect; **PVR** = pulmonary vascular resistance.

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creates left-to-right shunting of blood from the left atrium to the right atrium, increasing blood volume in the right atrium

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Hemodynamic effects of common congenital heart defects		
	Overload produced	**Ventricle primarily affected**
Atrial septal defect	Volume	Right
Ventricular septal defect	Volume	Left
Patent ductus arteriosus	Volume	Left
Aortic coarctation	Pressure	Left

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A 23-year-old previously healthy man is brought to the emergency department after a stab injury. His friends report that they were "walking down the street minding our own business when a guy jumped out in front of us from behind a dumpster and stabbed him in the chest." They were able to disarm the man after this single attack, and the man then ran off into an alley. The patient is conscious but in distress. Physical examination shows a laterally directed anterior chest wall stab wound at the fifth intercostal space along the left midclavicular line. Which of the following structures is most likely to have been injured in this patient?

- A. Azygos vein
- B. Inferior vena cava
- C. Left atrium
- D. Left lung
- E. Right ventricle

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A 23-year-old previously healthy man is brought to the emergency department after a stab injury. His friends report that they were "walking down the street minding our own business when a guy jumped out in front of us from behind a dumpster and stabbed him in the chest." They were able to disarm the man after this single attack, and the man then ran off into an alley. The patient is conscious but in distress. Physical examination shows a laterally directed anterior chest wall stab wound at the fifth intercostal space along the left midclavicular line. Which of the following structures is most likely to have been injured in this patient?

- ✗ A. Azygos vein (1%)
- B. Inferior vena cava (0%)
- C. Left atrium (8%)
- ✓ D. Left lung (52%)
- E. Right ventricle (36%)

Incorrect
Correct answer
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52%
Answered correctly

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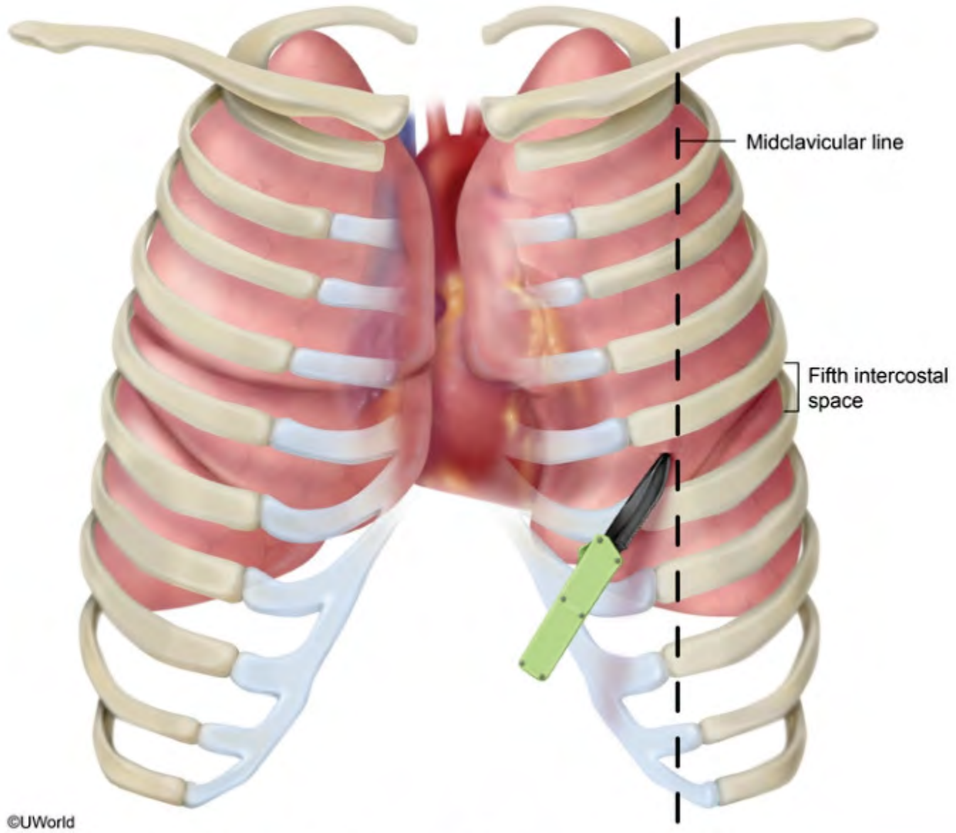
Chest anatomy & stab wound



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A penetrating, laterally directed stab wound (possibly from a left-handed assaulter) that involves the fifth intercostal space (ICS) at the left anterior midclavicular line (MCL) would most likely injure the **left lung**. The lungs occupy most of the volume of the thoracic cavity. The apex of each lung extends into the neck (3-4 cm above the first rib). The lung bases are in direct contact with the diaphragm, which separates the right lung from the right lobe of the liver and the left lung from the stomach, spleen, and (occasionally) left lobe of the liver. The mediastinal surface of each lung has a cardiac impression that accommodates the heart.

A deeper (and more medially directed) wound could have injured the left (not the right) ventricle. The heart is located behind the sternum, and its anterior surface is partially **covered** by the lungs. The cardiac apex is formed by the left ventricle. Depending on heart size, the point of **maximal** apical impulse can be felt at the fifth left ICS (between the fifth and sixth ribs) at the left MCL. All other heart chambers, including the right ventricle, lie **medial** to the left MCL and are unlikely to have been affected by the injury. The right ventricle forms the sternocostal (anterior) surface of the heart **(Choice E)**. The diaphragmatic (inferior) surface is formed by the left and right ventricles and is in contact with the central tendon of the diaphragm. The posterior surface of the heart is formed mainly by the left atrium **(Choice C)**.

(Choice A) The azygos vein lies in the posterior mediastinum immediately to the right of the midline. It drains blood from the posterior intercostal veins into the superior vena cava.

(Choice B) The inferior vena cava is located in the mediastinum, lies to the right of midline, and drains into the right atrium.

Educational objective:
The left ventricle forms the apex of the heart and can reach as far as the fifth intercostal space at the left midclavicular line (MCL). All other chambers of the heart lie medial to the left MCL. The lungs overlap much of the anterior surface of the heart.

Anatomy	Cardiovascular System	Penetrating thoracic trauma
Subject	System	Topic

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A 43-year-old man comes to the office due to occasional chest discomfort over the last 6 weeks. He thinks he is most likely experiencing musculoskeletal pain but is concerned due to family history of heart disease. The patient has no medical conditions and does not use tobacco. He leads an active lifestyle and exercises every day. He undergoes treadmill exercise stress testing. Baseline blood pressure is 122/75 mm Hg and pulse is 54/min. After 10 minutes of exercise, his blood pressure is 155/80 mm Hg and pulse is 150/min. He has no chest pain and ECG shows no abnormalities. Compared to pretest conditions, which of the following is the single most important limiting factor for left ventricular myocardial blood supply during the test?

- A. Contraction force
- B. Coronary vasoconstriction
- C. Diastolic aortic pressure
- D. Duration of diastole
- E. Intraventricular pressure
- F. Systolic ventricular wall stress

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A 43-year-old man comes to the office due to occasional chest discomfort over the last 6 weeks. He thinks he is most likely experiencing musculoskeletal pain but is concerned due to family history of heart disease. The patient has no medical conditions and does not use tobacco. He leads an active lifestyle and exercises every day. He undergoes treadmill exercise stress testing. Baseline blood pressure is 122/75 mm Hg and pulse is 54/min. After 10 minutes of exercise, his blood pressure is 155/80 mm Hg and pulse is 150/min. He has no chest pain and ECG shows no abnormalities. Compared to pretest conditions, which of the following is the single most important limiting factor for left ventricular myocardial blood supply during the test?

- ✗ A. Contraction force (6%)
- B. Coronary vasoconstriction (8%)
- C. Diastolic aortic pressure (4%)
- ✓ D. Duration of diastole (74%)
- E. Intraventricular pressure (1%)
- F. Systolic ventricular wall stress (5%)

Incorrect
Correct answer
D

74%
Answered correctly

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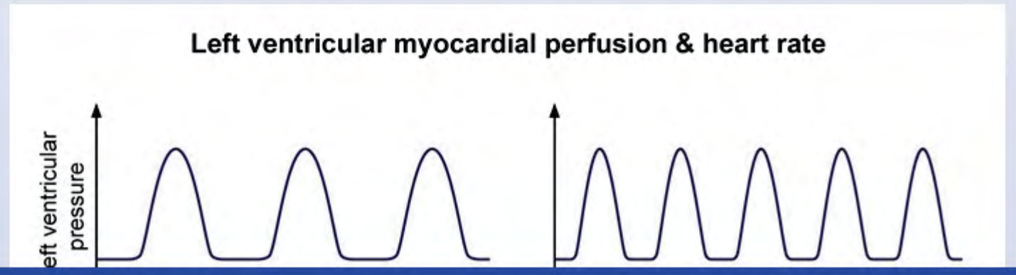

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E. Intraventricular pressure (1%)

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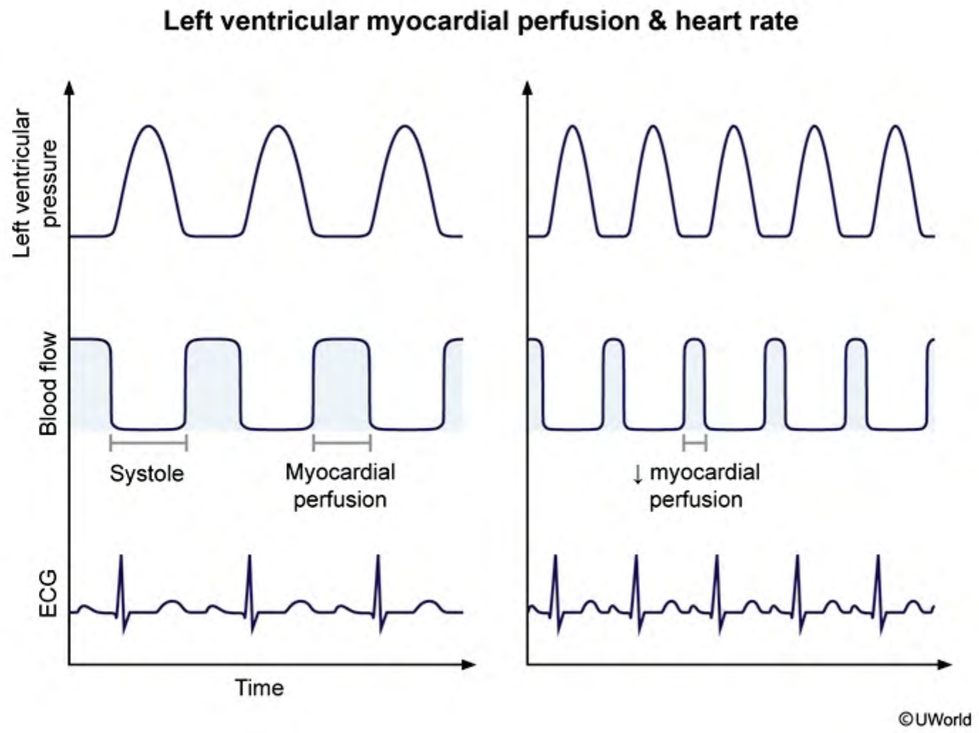

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Myocardial perfusion is provided by the right and left coronary arteries arising directly from the aortic root. In contrast to most other vascular beds in the body, **left ventricular myocardial perfusion** occurs mainly during

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Myocardial perfusion is provided by the right and left coronary arteries arising directly from the aortic root. In contrast to most other vascular beds in the body, **left ventricular myocardial perfusion** occurs mainly during **diastole**. During ventricular systole, intraventricular pressure and wall stress in the left ventricle exceed the aortic perfusion pressure (eg, 120 mm Hg), preventing effective coronary perfusion. Wall tension is highest in the subendocardial region, making this area of the myocardium particularly susceptible to ischemia. Relaxation of the left ventricle during diastole decreases intraventricular pressure to about 10 mm Hg (which is much lower than aortic diastolic pressure [~80 mm Hg]), providing a driving force that allows for adequate perfusion.

Increased heart rate shortens the time of ventricular relaxation (**duration of diastole**); therefore, the time available for maximal coronary blood flow decreases and consequently becomes the major limiting factor for coronary blood supply to the myocardium.

(Choice B) During exercise, mechanisms such as flow-mediated dilation and release of vasodilators such as adenosine and nitric oxide ensure adequate coronary blood supply.

(Choices A, E, and F) Left ventricular contraction force, intraventricular pressure, and ventricular wall stress all increase during exercise to provide adequate blood flow to actively contracting skeletal muscle. However, because there is already minimal left ventricular perfusion occurring during systole, the increase in these forces does not cause further limitation in left ventricular myocardial perfusion.

(Choice C) Diastolic aortic pressure is the driving force for coronary blood flow during diastole, and coronary blood flow is reduced in patients with low diastolic pressures. This patient's diastolic pressures are similar while at rest and during exercise and would not contribute to decreased coronary blood supply.

Educational objective:
The high systolic intraventricular pressure and wall stress of the left ventricle prevent myocardial perfusion during systole; therefore, the majority of left ventricular myocardial perfusion occurs during diastole. Shorter duration of diastole is the major limiting factor for coronary blood supply to the left ventricular myocardium during periods of tachycardia (eg, exercise).

Physiology	Cardiovascular System	Coronary blood flow
Subject	System	Topic

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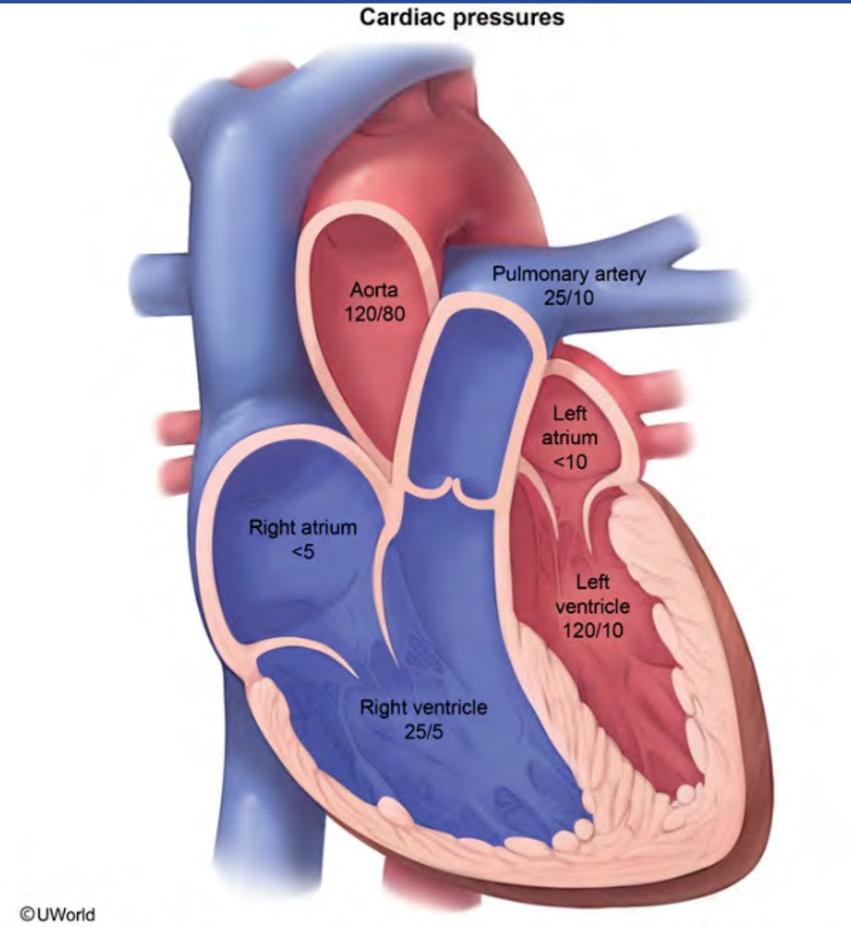

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Physiology (Subject) | Cardiovascular System (System) | Coronary blood flow (Topic)

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 52-year-old man comes to the physician because of increasing fatigue over the last 2 weeks. He has also been experiencing exertional dyspnea and occasional headaches. Cardiac auscultation findings at the left sternal border are shown below. Which of the following is most consistent with this patient's auscultatory findings?

Play Media

- A. Aortic regurgitation
- B. Hypertrophic cardiomyopathy
- C. Mitral valve prolapse
- D. Papillary muscle dysfunction
- E. Tricuspid regurgitation

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Play Media

- ✓ A. Aortic regurgitation (59%)
- B. Hypertrophic cardiomyopathy (4%)
- C. Mitral valve prolapse (9%)
- D. Papillary muscle dysfunction (5%)
- E. Tricuspid regurgitation (20%)

Correct

59%
Answered correctly

01 min, 35 secs
Time Spent

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Explanation

Cardiac murmurs

	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed

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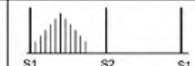
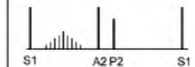
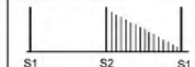
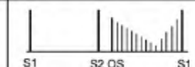
Exhibit Display

Cardiac murmurs

Systolic	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid–left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation*		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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This patient has an **aortic regurgitation** (AR) murmur. It is a high-pitched, blowing, **diastolic** murmur; the intensity is maximal right after A2 (sound of aortic valve closure) and proceeds in a decrescendo pattern for part or all of diastole. Higher overall intensity and longer duration of the murmur correlate with the severity of AR, and A2 may be absent in severe AR.

When AR is due to **aortic root dilation** (eg, in Marfan syndrome) the murmur is best heard at the **right upper sternal border** (classic aortic valve auscultatory location). However, for AR due to **valvular pathology** (eg, bicuspid aortic valve, rheumatic heart disease) the murmur is best heard at the **left third intercostal space**. In either scenario, the murmur is best appreciated at the end of expiration with the patient leaning forward (minimized lung volume with the heart closer to the chest wall enhances left-sided murmurs).

(Choice B) Hypertrophic cardiomyopathy causes left ventricular outflow obstruction and produces a crescendo-decrescendo systolic murmur similar to that of aortic stenosis. The murmurs can be differentiated by maneuvers that increase left ventricular blood volume (eg, squatting); the murmur of hypertrophic cardiomyopathy decreases in intensity with increased left ventricular blood volume (outflow obstruction is reduced), whereas the murmur of aortic stenosis increases in intensity.

(Choice C) Mitral valve prolapse is classically associated with a midsystolic click and a late systolic murmur best heard at the cardiac apex.

(Choice D) Papillary muscle dysfunction can lead to mitral regurgitation, which causes a holosystolic murmur best heard at the cardiac apex.

(Choice E) Tricuspid regurgitation causes a holosystolic murmur that is best heard at the left lower sternal border. The murmur can be differentiated from that of mitral regurgitation by auscultation during inspiration; the murmur of tricuspid regurgitation will increase in intensity with inspiration as right ventricular blood volume is increased.

Educational objective:

Aortic regurgitation (AR) causes a high-pitched, blowing, diastolic murmur with a decrescendo intensity pattern.

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intensity is maximal right after A2 (sound of aortic valve closure) and proceeds in a decrescendo pattern for part or all of diastole. Higher overall intensity and longer duration of the murmur correlate with the severity of AR, and A2 may be absent in severe AR.

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Educational objective:
Aortic regurgitation (AR) causes a high-pitched, blowing, diastolic murmur with a decrescendo intensity pattern. The murmur of AR due to aortic root dilation is best heard at the right upper sternal border, whereas the murmur of AR due to valvular pathology is best heard at the left third intercostal space.

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Continuous	Patent ductus		Machinery-like at LUSB or

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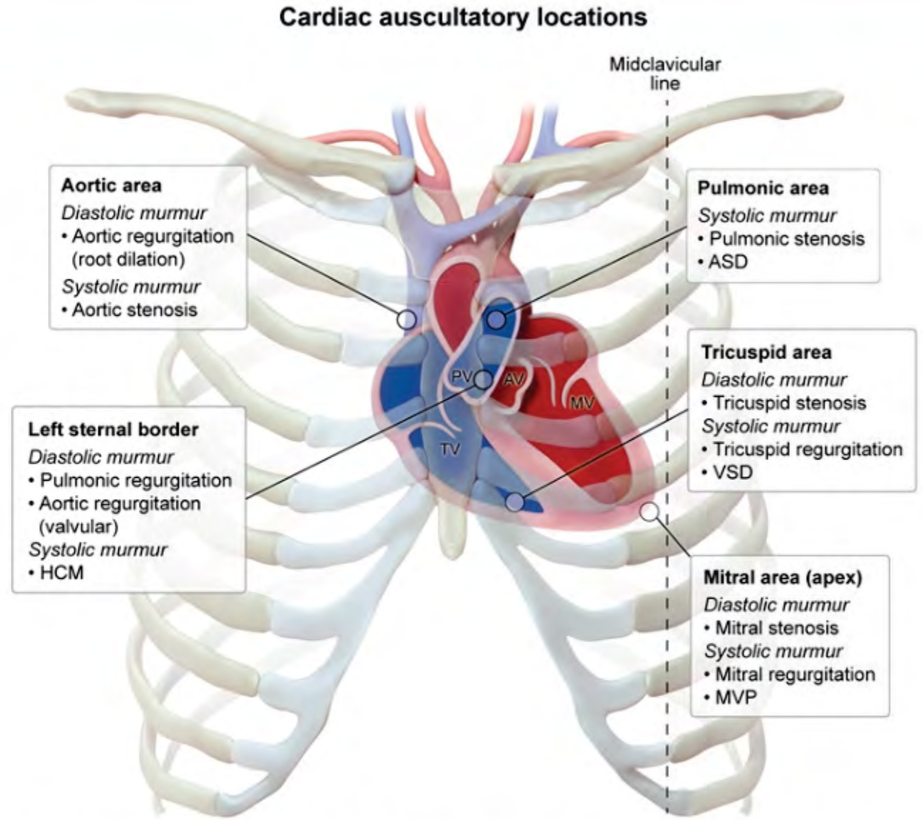

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A 78-year-old man with severe dementia is brought to the emergency department due to a daylong history of lethargy, fever, and vomiting. The patient lives in a nursing home, and his caregivers report that he has had poor oral intake over the last week. Medical history is significant for emphysema, heart failure with reduced ejection fraction, hypertension, and diabetes mellitus type 2. On physical examination, the patient is lethargic but arousable. Blood pressure is 60/30 mm Hg and pulse is 120/min and regular. The extremities are warm. Coarse rhonchi are heard over the right lower lung. After receiving several intravenous fluid boluses, the patient is given an intravenous infusion of an agent that increases peripheral vascular resistance, increases blood pressure, and decreases heart rate. Which of the following agents is most likely being described?

- A. Dobutamine
- B. High-dose dopamine
- C. Low-dose epinephrine
- D. Milrinone
- E. Phenylephrine

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A 78-year-old man with severe dementia is brought to the emergency department due to a daylong history of lethargy, fever, and vomiting. The patient lives in a nursing home, and his caregivers report that he has had poor oral intake over the last week. Medical history is significant for emphysema, heart failure with reduced ejection fraction, hypertension, and diabetes mellitus type 2. On physical examination, the patient is lethargic but arousable. Blood pressure is 60/30 mm Hg and pulse is 120/min and regular. The extremities are warm. Coarse rhonchi are heard over the right lower lung. After receiving several intravenous fluid boluses, the patient is given an intravenous infusion of an agent that increases peripheral vascular resistance, increases blood pressure, and decreases heart rate. Which of the following agents is most likely being described?

- A. Dobutamine (10%)
- B. High-dose dopamine (14%)
- C. Low-dose epinephrine (10%)
- ✗ D. Milrinone (3%)
- ✓ E. Phenylephrine (60%)

Incorrect
Correct answer
E

60%
Answered correctly

19 secs
Time Spent

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Version

Explanation

Cardiovascular effects of adrenergic agonists
• α_1: peripheral vasoconstriction, ↑ SVR
• β_1: ↑ heart rate & contractility, ↑ cardiac output
• β_2: peripheral vasodilation, ↓ SVR

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Explanation

Cardiovascular effects of adrenergic agonists

- α_1: peripheral vasoconstriction, ↑ SVR
- β_1: ↑ heart rate & contractility, ↑ cardiac output
- β_2: peripheral vasodilation, ↓ SVR
- D_1: renal arteriolar vasodilation

Agonist	Receptor binding	Blood pressure	Heart rate
Dobutamine	$\beta_1 > \beta_2$	–/↓	↑
Dopamine	$D_1 > \beta_1 > \alpha_1$ (low-dose)	–/↓	↑
	$\alpha_1 > \beta_1 > D_1$ (high-dose)	↑	–/↓*
Epinephrine	$\beta_1 > \beta_2 > \alpha_1$ (low-dose)	–/↓	↑
	$\alpha_1 > \beta_1 > \beta_2$ (high-dose)	↑	–/↓*
Isoproterenol	$\beta_1 = \beta_2$	–/↓	↑
Norepinephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	–/↓*
Phenylephrine	α_1	↑	↓*

*Reflex-mediated decrease.

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- α_1: peripheral vasoconstriction, ↑ SVR
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Agonist	Receptor binding	Blood pressure	Heart rate
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	$\alpha_1 > \beta_1 > D_1$ (high-dose)	↑	–/↓*
Epinephrine	$\beta_1 > \beta_2 > \alpha_1$ (low-dose)	–/↓	↑
	$\alpha_1 > \beta_1 > \beta_2$ (high-dose)	↑	–/↓*
Isoproterenol	$\beta_1 = \beta_2$	–/↓	↑
Norepinephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	–/↓*
Phenylephrine	α_1	↑	↓*

*Reflex-mediated decrease.
SVR = systemic vascular resistance.

Adrenergic agonists are often used to increase blood pressure in patients with shock. **Phenylephrine** is a **selective alpha-1 agonist** with no effect on alpha-2 receptors or beta receptors; it causes peripheral

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SVR = systemic vascular resistance.

Adrenergic agonists are often used to increase blood pressure in patients with shock. **Phenylephrine** is a **selective alpha-1 agonist** with no effect on alpha-2 receptors or beta receptors; it causes peripheral vasoconstriction, resulting in **increased systemic vascular resistance** (SVR) and **increased blood pressure**, but it has **no direct effect on the heart**. Drugs that increase blood pressure by increasing SVR are called **pressors** and are most useful in treating septic shock.

Phenylephrine has indirect effects on the heart mediated via the **baroreceptor reflex**. Increased blood pressure stimulates firing of aortic and carotid baroreceptors. In response, the CNS increases parasympathetic output and decreases sympathetic output, which causes **decreased heart rate** and myocardial contractility. These reflexive changes slightly attenuate the initial increase in blood pressure and lead to overall reduced cardiac output.

(Choices A and D) Dobutamine is primarily a beta-1 agonist with partial activity on beta-2 receptors and minimal effect on alpha receptors. It increases heart rate and myocardial contractility (via beta-1 stimulation) and causes mild vasodilation (via beta-2 stimulation) to decrease SVR. Milrinone is not an adrenergic agonist, but it inhibits phosphodiesterase 3 to increase myocardial contractility and cause peripheral vasodilation (decreases SVR). The combination of increased myocardial contractility (ie, inotropic effect) and reduced SVR induced by these drugs is most useful in treating cardiogenic shock.

(Choice B) Dopamine stimulates dopamine-1 (located primarily in the renal arterioles), beta-1, and alpha-1 receptors, with the relative effects being dose dependent. High-dose dopamine primarily stimulates alpha-1 receptors to increase SVR and blood pressure; ongoing beta-1 receptor stimulation offsets the reflex–mediated decrease in heart rate, leaving the heart rate relatively unchanged.

(Choice C) Epinephrine stimulates beta-1, beta-2, and alpha-1 receptors with dose-dependent effects. Low-dose epinephrine primarily stimulates beta-1 receptors, resulting in increased cardiac output due to increased heart rate and contractility. At low doses, the vasodilative beta-2–mediated effects are greater than the vasoconstrictive alpha-1–mediated effects, leading to a slight reduction in SVR.

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Phenylephrine has indirect effects on the heart mediated via the **baroreceptor reflex**. Increased blood pressure stimulates firing of aortic and carotid baroreceptors. In response, the CNS increases parasympathetic output and decreases sympathetic output, which causes **decreased heart rate** and myocardial contractility. These reflexive changes slightly attenuate the initial increase in blood pressure and lead to overall reduced cardiac output.

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Educational objective:
Phenylephrine is a selective alpha-1 agonist with no effect on alpha-2 or beta receptors. It causes peripheral vasoconstriction that increases systemic vascular resistance and blood pressure. It has no direct effect on the heart but stimulates a reflex–mediated decrease in heart rate and myocardial contractility.

Pharmacology	Cardiovascular System	Sepsis
Subject	System	Topic

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Norepinephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	−/↓
Phenylephrine	α_1	↑	↓*

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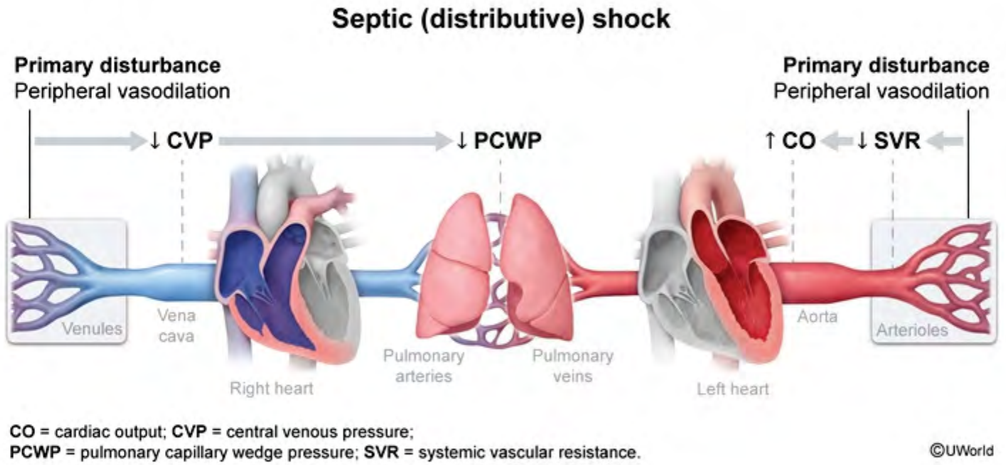

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Norepinephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	−/↓
Phenylephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	−/↓

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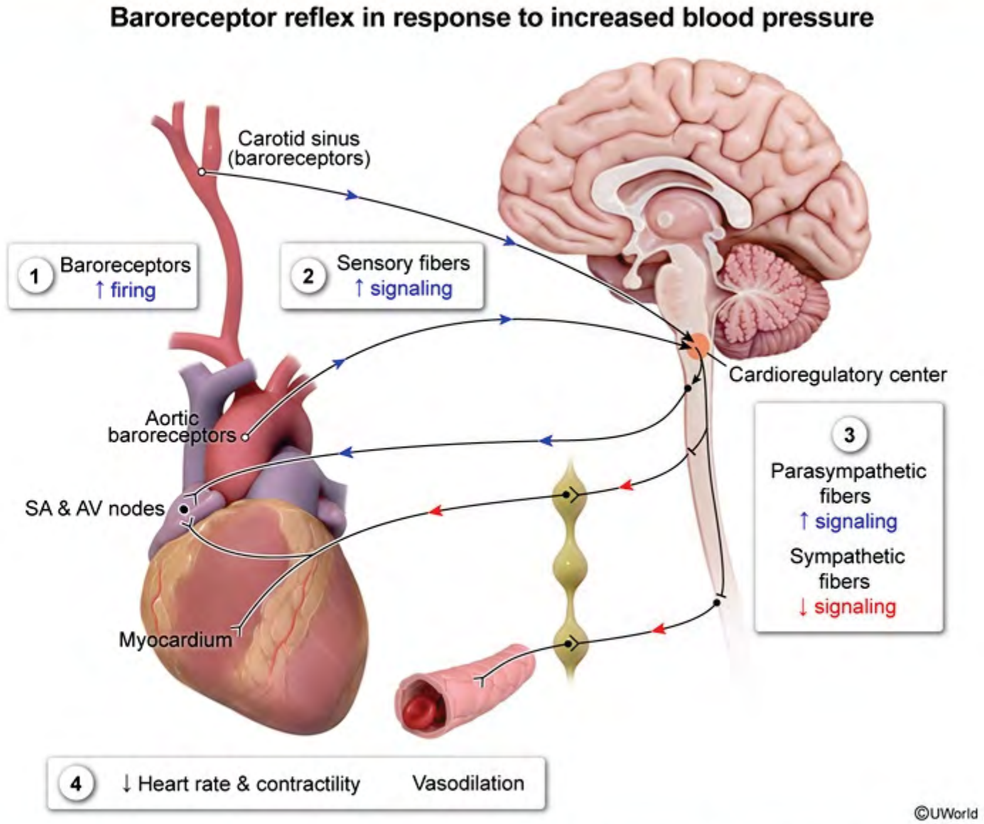

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(Choice C) Epinephrine stimulates beta-1, beta-2, and alpha-1 receptors with dose-dependent effects. Low-dose

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SVR = systemic vascular resistance.

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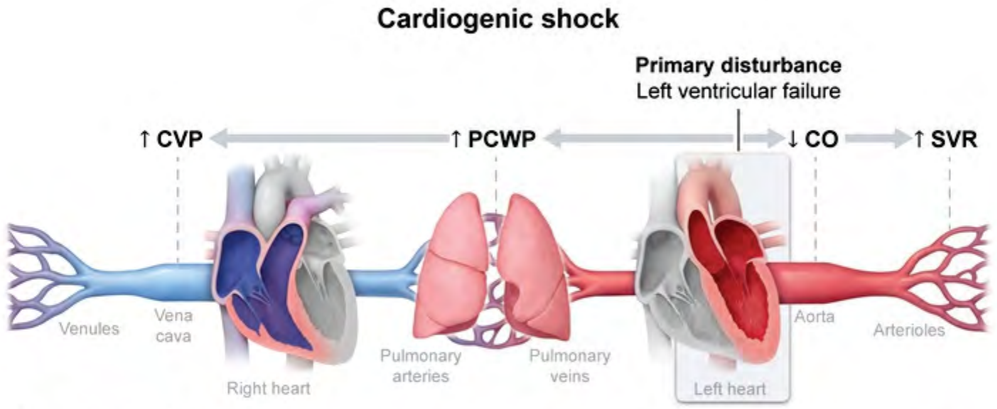

CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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An 18-year-old woman is referred to a cardiologist after a heart murmur is discovered during a routine checkup. The patient is healthy and has no symptoms. Medical history is unremarkable. She runs daily and wants to start actively training for a half marathon. The patient is concerned that the murmur is a sign of heart disease. She has no family history of sudden cardiac death. Auscultation reveals a midsystolic click followed by a short late-systolic murmur at the cardiac apex. The murmur disappears with squatting. This patient's condition is most likely related to an abnormality involving which of the following tissues?

- A. Cardiac muscle
- B. Connective tissue
- C. Coronary vessels
- D. Endocardial lining
- E. Parietal pericardium

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- ✗ A. Cardiac muscle (15%)
- ✓ B. Connective tissue (64%)
- C. Coronary vessels (2%)
- D. Endocardial lining (17%)
- E. Parietal pericardium (0%)

Incorrect
Correct answer
B

64%
Answered correctly

03 secs
Time Spent

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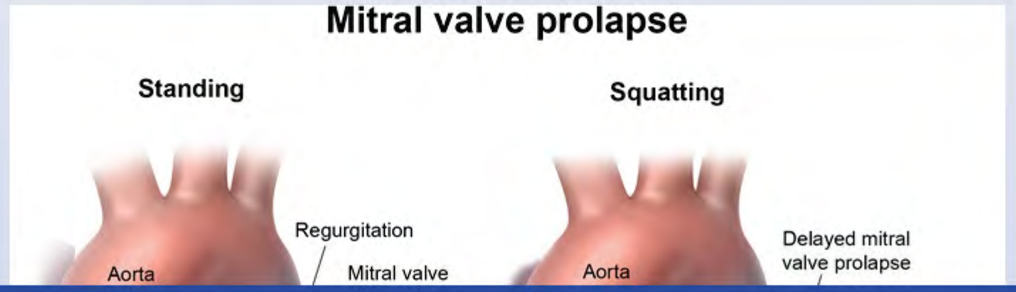
Explanation



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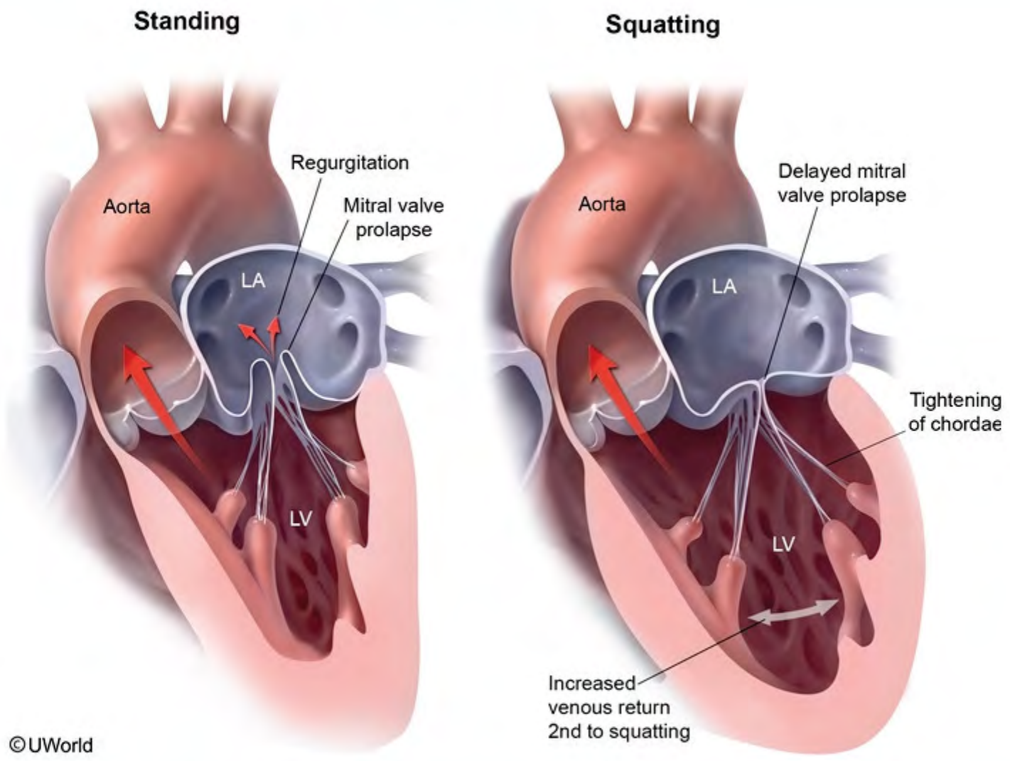

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The presence of a **midsystolic click** followed by a systolic murmur at the cardiac apex that disappears with squatting is most consistent with mitral valve prolapse (MVP). The click is caused by sudden tensing of the chordae tendineae as they are pulled taut by the ballooning valve leaflets, and the murmur results from regurgitation of blood due to malalignment of the valve margins with prolapse.

During systole, there is a critical left ventricular (LV) size at which prolapse occurs; therefore, maneuvers that change LV end-diastolic volume affect the timing of the click and murmur. **Squatting increases venous return** and **LV volume**, increasing the tension and **decreasing the slack** in the mitral valve apparatus. Because the ventricular walls have farther to travel to reach the critical LV size at which prolapse occurs, **prolapse occurs later** in systole or not at all.

Primary MVP is most commonly a sporadic disorder and is characterized by myxomatous degeneration (ie, pathologic **deterioration of the connective tissue**) affecting the mitral valve leaflets and chordae tendineae. Secondary MVP is associated with inherited connective tissue disorders, including Marfan or Ehlers-Danlos syndrome and osteogenesis imperfecta.

(Choice A) Hypertrophic cardiomyopathy (HCM) presents with a systolic murmur at the left sternal border that decreases in intensity with squatting (due to increased LV volume and decreased outflow tract obstruction). A midsystolic click is not heard with HCM.

(Choice C) Coronary artery disease can cause myocardial ischemia or infarction leading to papillary muscle dysfunction and acute mitral regurgitation. In such patients, squatting from a standing position increases murmur intensity due to a higher blood volume of regurgitation. In addition, such a murmur is not preceded by a midsystolic click.

(Choice D) Endomyocardial fibrosis and endocardial fibroelastosis are disorders that affect the endocardium and are not associated with MVP.

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(Choice D) Endomyocardial fibrosis and endocardial fibroelastosis are disorders that affect the endocardium and are not associated with MVP.

(Choice E) Pericardial disease may infrequently result in cardiac tamponade or constrictive pericarditis. Pericarditis may produce a friction rub; however, it is not associated with a cardiac murmur.

Educational objective:
Mitral valve prolapse is most often caused by defects in connective tissue proteins that predispose to myxomatous degeneration of the mitral leaflets and chordae tendineae. Cardiac auscultation typically reveals a midsystolic click followed by a mitral regurgitation murmur; the click and murmur occur later in systole or disappear completely with maneuvers (eg, squatting) that increase left ventricular end-diastolic volume.

References

- On systolic murmurs and cardiovascular physiological maneuvers.

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Exhibit Display

Variation in intensity of cardiac murmurs

Maneuver	Change in cardiac parameters	Murmurs that increase	Murmurs that decrease
Inspiration	↑ RV preload, ↓ LV preload	Most right-sided murmurs (↑ RV blood volume)	Most left-sided murmurs (↓ LV blood volume)
Valsalva strain phase	↓ RV & LV preload	MVP & HCM (↓ LV blood volume)	Most valvular murmurs (↓ flow through valve)
Abrupt standing			
Squatting	↑ RV & LV preload, ↑ LV afterload	Most valvular murmurs (↑ flow through valve)	MVP & HCM (↑ LV blood volume)
Passive leg raise	↑ RV & LV preload		
Hand grip	↑ LV afterload	AR, MR & VSD (↑ aortic & LV pressure)	MVP & HCM (↑ LV blood volume) AS (↓ transvalvular pressure gradient)

AR = aortic regurgitation; **AS** = aortic stenosis; **HCM** = hypertrophic cardiomyopathy; **LV** = left ventricular; **MR** = mitral regurgitation; **MVP** = mitral valve prolapse; **RV** = right ventricular; **VSD** = ventricular septal defect.

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A 56-year-old man comes to the emergency department due to chest palpitations. The patient feels that his heartbeat is fast and irregular. He has had no chest pain, shortness of breath, or dizziness. The patient normally drinks only 2-3 times a year, but he hosted a party last night for his wife's birthday and consumed a large amount of alcohol. He is otherwise healthy and takes no medications. Pulse check confirms the presence of an irregularly irregular rhythm with a rate of 138/min. This patient's ECG strip is most likely to show which of the following?

- A.
- B.
- C.
- D.
- E.

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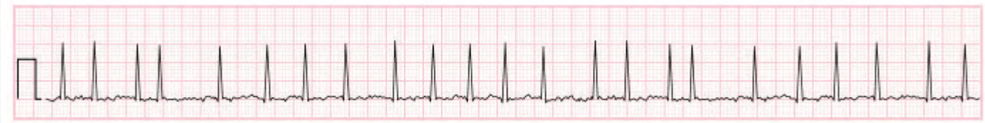

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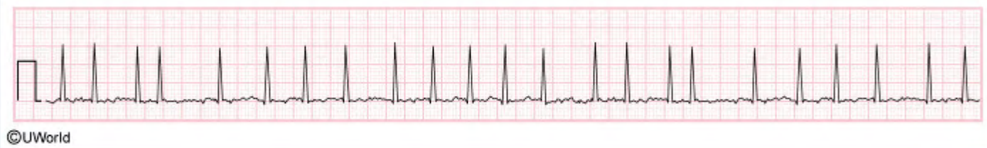

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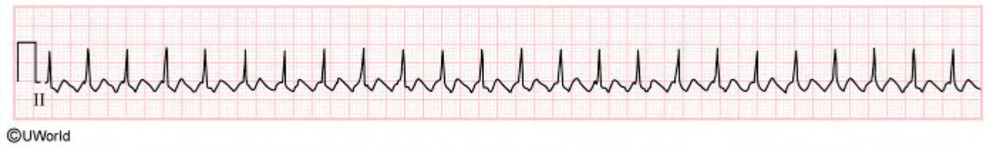
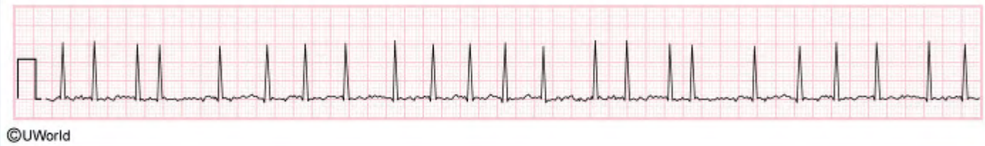
B. (1%)

C. (5%)



✓ D. (89%)



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Atrial fibrillation	

Pathogenesis	• Atrial remodeling due to:
	  ◦ Age-related myocardial changes
	  ◦ Atrial enlargement from heart disease (eg, HTN, MS)
	• Foci of rapid electrical activity:
	  ◦ Commonly originate in pulmonary veins
	  ◦ Alcohol intake & ↑ sympathetic drive may contribute

Clinical presentation	• Asymptomatic or palpitations ± dyspnea, fatigue • Irregularly irregular pulse • May present with thromboembolic event (eg, stroke)
Diagnosis	• ECG shows variable R-R intervals & no P waves • Continuous ECG often needed for paroxysmal disease
HTN = hypertension; **MS** = mitral stenosis.	

Palpitations are a subjective sensation of the heartbeat usually caused by rapid arrhythmia or forceful ventricular contraction. This patient with palpitations and an **irregularly irregular** pulse most likely has **atrial fibrillation**, the most common cause of an irregularly irregular rhythm.

In atrial fibrillation, the atria experience rapid and chaotic electrical activity, and organized atrial depolarization is lost. In addition, because the rate of atrial electrical impulses is so high (eg, 500-600/min) and the atrioventricular node does not transmit impulses that quickly, impulses are transmitted to the ventricles randomly and intermittently. Therefore, ECG typically shows an **absence of P waves** and an irregularly irregular rhythm with **varying R-R intervals**; the ventricular rate is often (but not always) >100/min. In addition, irregular, low-amplitude **fibrillatory waves** are sometimes present between the QRS complexes.

The development of atrial fibrillation is encouraged by **atrial remodeling**, which involves changes to atrial

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The development of atrial fibrillation is encouraged by **atrial remodeling**, which involves changes to atrial conduction pathways that occur as a result of aging and underlying chronic disease (eg, hypertension, heart failure). Once a certain degree of remodeling is present, acute illness (eg, sepsis or other causes of increased sympathetic tone) and other conditions (eg, hyperthyroidism) can initiate foci of electrical activity that **trigger** the arrhythmia. Excessive **alcohol consumption** is also a common trigger (ie, "holiday heart syndrome").

(Choice A) Ventricular bigeminy involves a premature ventricular contraction after each normal sinus beat and is often triggered by periods of increased sympathetic tone. Palpitations are common, but the rhythm is regularly irregular (ie, R-R interval varies in a predictable manner), with 2 beats occurring close together followed by a relatively long pause before the next beat.

(Choice B) Ventricular tachycardia demonstrates wide QRS complexes on ECG. It may cause palpitations, but the rhythm is regular.

(Choice C) Atrial flutter demonstrates saw-toothed flutter waves on ECG and may cause palpitations. Because atrial activity is slower (eg, 300/min) and somewhat more organized than in atrial fibrillation, the rhythm can be regular or irregular, depending on the steadiness of conduction through the atrioventricular node. This ECG shows

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Varying R-R intervals, the ventricular rate is often (but not always) >100/min. In addition, irregular, low-amplitude **fibrillatory waves** are sometimes present between the QRS complexes.

The development of atrial fibrillation is encouraged by **atrial remodeling**, which involves changes to atrial conduction pathways that occur as a result of aging and underlying chronic disease (eg, hypertension, heart failure). Once a certain degree of remodeling is present, acute illness (eg, sepsis or other causes of increased sympathetic tone) and other conditions (eg, hyperthyroidism) can initiate foci of electrical activity that **trigger** the arrhythmia. Excessive **alcohol consumption** is also a common trigger (ie, "holiday heart syndrome").

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(Choice E) Atrioventricular nodal reentrant tachycardia commonly causes palpitations, but the rhythm is regular. P waves are often not visible on ECG because they are buried within the QRS complexes.

Educational objective:
Atrial fibrillation is the most common cause of an irregularly irregular rhythm. It often presents with palpitations and is confirmed by ECG showing an absence of organized P waves and irregularly irregular R-R intervals.

Physiology	Cardiovascular System	Atrial fibrillation
Subject	System	Topic

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HTN = hypertension; MS = mitral stenosis

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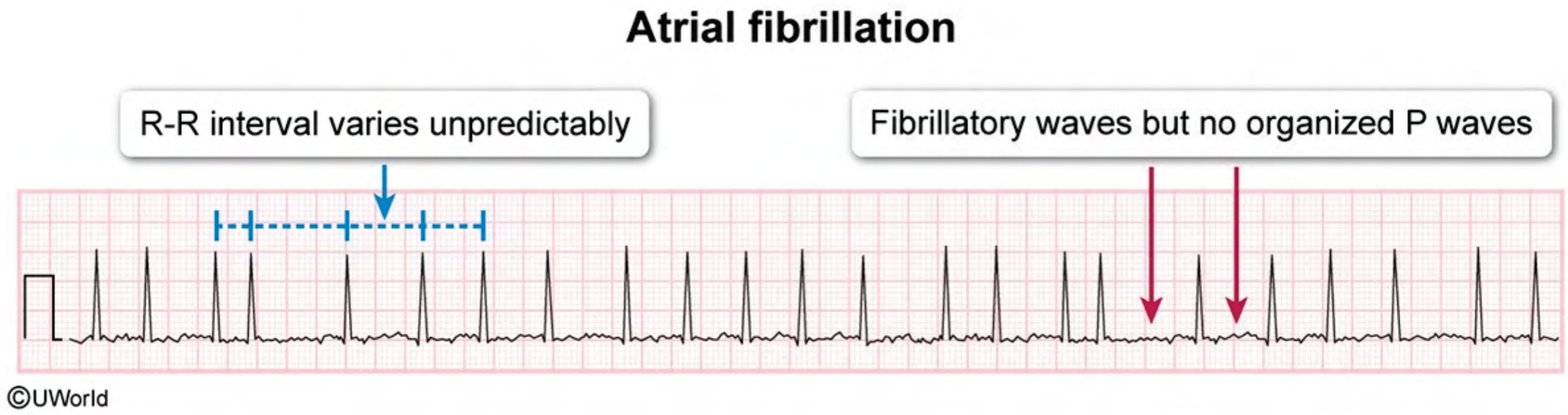

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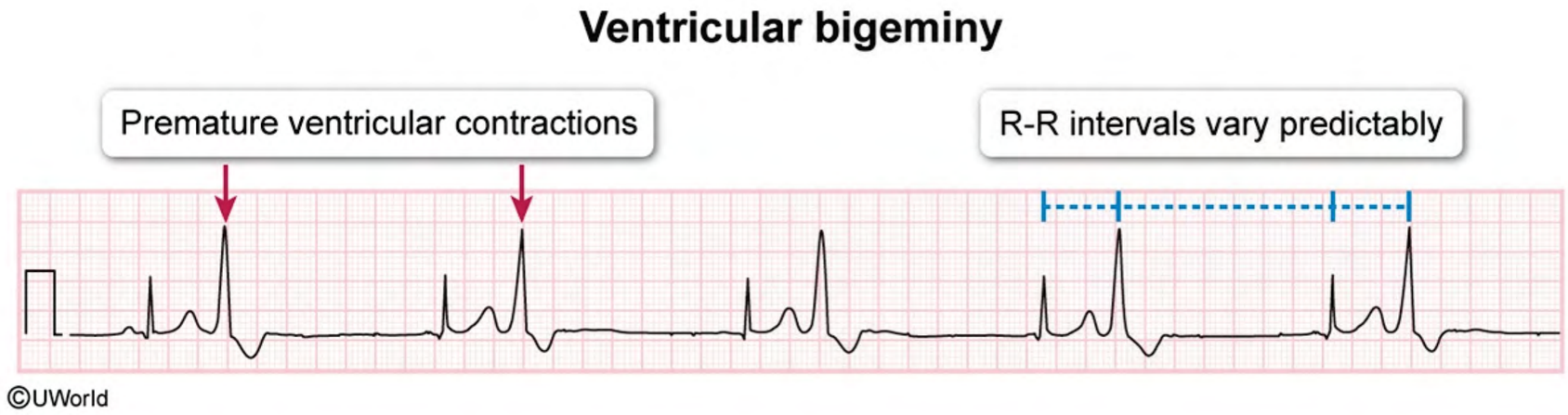

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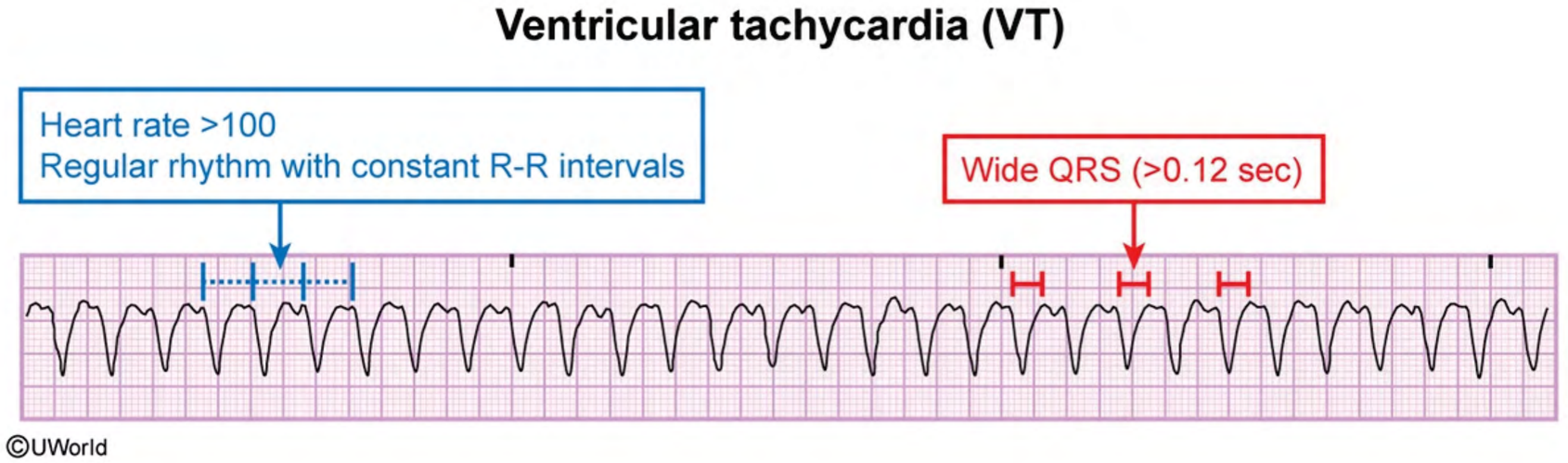

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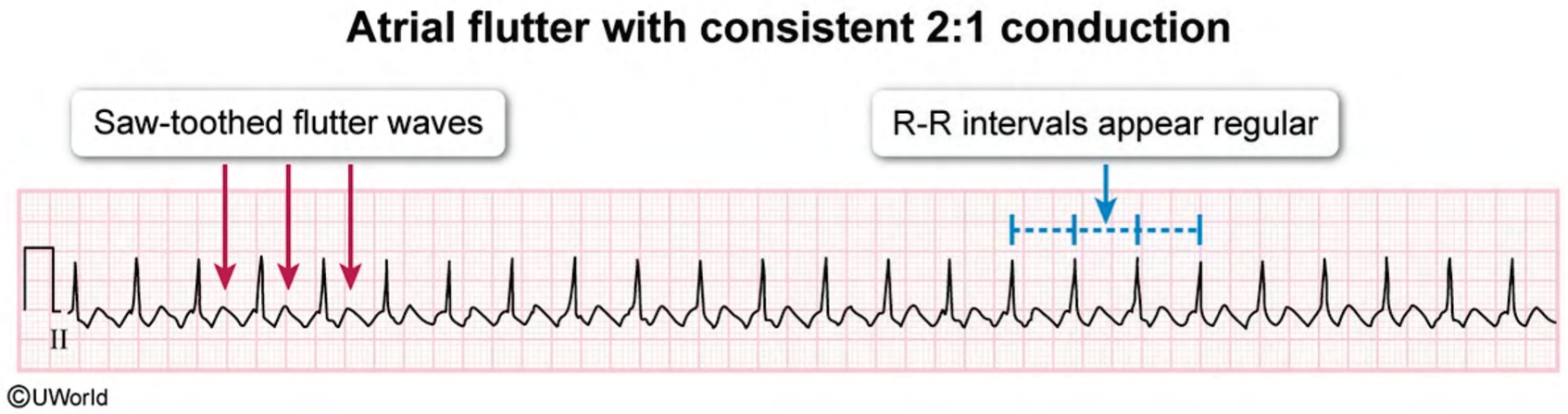

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Atrioventricular nodal reentrant tachycardia (AVNRT)

Pathophysiology	Key features
• Dual AV node conduction pathways (fast & slow) creating a reentrant circuit • Triggered by a premature atrial contraction	• 120-180 bpm • Regular R-R intervals • P waves buried within the QRS complexes • Narrow QRS complexes

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A 17-year-old boy is brought to the emergency department after passing out. The patient was standing behind the dugout watching his friends play baseball when he suddenly felt warm and light-headed. Then he remembers awakening on the ground. Bystanders reported that the patient was unresponsive for approximately 2 minutes. He feels fatigued but does not have chest pain or shortness of breath. The patient has no significant medical history and does not use alcohol or drugs. Temperature is 36.7 C (98.1 F), supine blood pressure is 105/60 mm Hg and standing blood pressure is 110/65 mm Hg, pulse is 84/min, and respirations are 16/min. Pulse oximetry is 100% on room air. The patient is awake and alert. Pupils are equal and reactive. Heart and lung examinations are normal. The abdomen is soft and nontender. Neurologic examination is normal. ECG is unremarkable. Which of the following is the most likely cause of this patient's syncope?

- A. Increased automaticity of the Purkinje fibers
- B. Increased parasympathetic output to the heart
- C. Increased sympathetic output to the small vessels
- D. Reentry arrhythmia using the atrioventricular node
- E. Systolic anterior motion of the mitral valve

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- ✗ A. Increased automaticity of the Purkinje fibers (2%)
- ✓ B. Increased parasympathetic output to the heart (65%)
- C. Increased sympathetic output to the small vessels (11%)
- D. Reentry arrhythmia using the atrioventricular node (14%)
- E. Systolic anterior motion of the mitral valve (5%)

Incorrect
Correct answer
B

65%
Answered correctly

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Explanation

Vasovagal & situational syncope	
Triggers	• Pain, anxiety, emotional stress, heat, prolonged standing • Situational: cough, micturition, defecation

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Vasovagal & situational syncope	
Triggers	• Pain, anxiety, emotional stress, heat, prolonged standing • Situational: cough, micturition, defecation
Pathophysiology	• Cardioinhibitory response (↑ parasympathetic tone): ↓ heart rate & contractility • Vasodepressor response (↓ sympathetic tone): vasodilation, ↓ preload
Clinical presentation	• Prodrome (eg, warmth, pallor, nausea, diaphoresis) • Rapid recovery of consciousness (eg, <1-2 min)
Diagnosis	• Based primarily on clinical history and examination • Upright tilt table testing sometimes indicated in uncertain cases

This patient has experienced **vasovagal syncope**, a type of reflex syncope. Patients with vasovagal syncope typically have a trigger (eg, prolonged standing) and experience a **prodrome** (eg, warmth, light-headedness) that immediately precedes loss of consciousness. The episodes usually have a **short duration** (eg, 1-2 min) and typically involve a **rapid recovery**. When vasovagal syncope is repeatedly triggered by a certain activity (eg, coughing, micturition), it is referred to as situational syncope.

Reflex syncope is the result of inappropriate autonomic output from the brainstem in response to signals relayed by various baroreceptors or mechanoreceptors. Vasovagal syncope is often triggered by cortical emotions that increase sensitivity to afferent signals, but it can also be triggered situationally by increased afferent transmission from pulmonary, gastrointestinal, or genitourinary mechanoreceptors. Once these afferent signals are received by the brainstem cardioregulatory center, some combination of the following effector responses occurs:

- Cardioinhibitory response: **Increased parasympathetic stimulation** to the sinoatrial and atrioventricular (AV) nodes slows the heart rate. In addition, decreased sympathetic output to the myocardium decreases myocardial contractility.
- Vasodepressor response: **Decreased sympathetic output** to vascular smooth muscle causes vasodilation of venous capacitance veins (ie, decreased cardiac preload) and systemic arterioles (ie, decreased perfusion

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The result of these responses is a brief but profound decrease in cardiac output that leads to hypotension, **decreased cerebral perfusion,** and syncope.

(Choices A and D) Cardiac syncope can occur due to the marked reduction in cardiac output that results from arrhythmia (eg, ventricular tachycardia, AV nodal reentrant tachycardia). Increased automaticity of the Purkinje fibers can trigger ventricular tachycardia, but this usually affects patients with ischemic or structural heart disease. AV nodal reentrant tachycardia usually affects young patients, but rarely reduces cardiac output enough to cause syncope. In addition, cardiac syncope of any etiology would not be associated with a vagal prodrome.

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(Choice E) Severe left ventricular outflow tract obstruction (eg, aortic stenosis, hypertrophic cardiomyopathy [HCM]) can cause cardiac syncope. Systolic anterior motion of the mitral valve can worsen outflow obstruction in patients with HCM, but syncope resulting from left ventricular outflow obstruction is typically exertional, and this patient has no systolic murmur to suggest HCM.

Educational objective:
Vasovagal syncope typically involves a trigger (eg, prolonged standing), a prodrome (eg, warmth, light-headedness), and rapid recovery (eg, within 1-2 min). It results from a transient, autonomic nervous system–mediated cardioinhibitory and vasodepressor response.

References

- Syncope in children and adolescents: evaluation and treatment

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Clinical presentation	• Prodrome (eg, warmth, pallor, nausea, diaphoresis) • Rapid recovery of consciousness (eg, <1-2 min)

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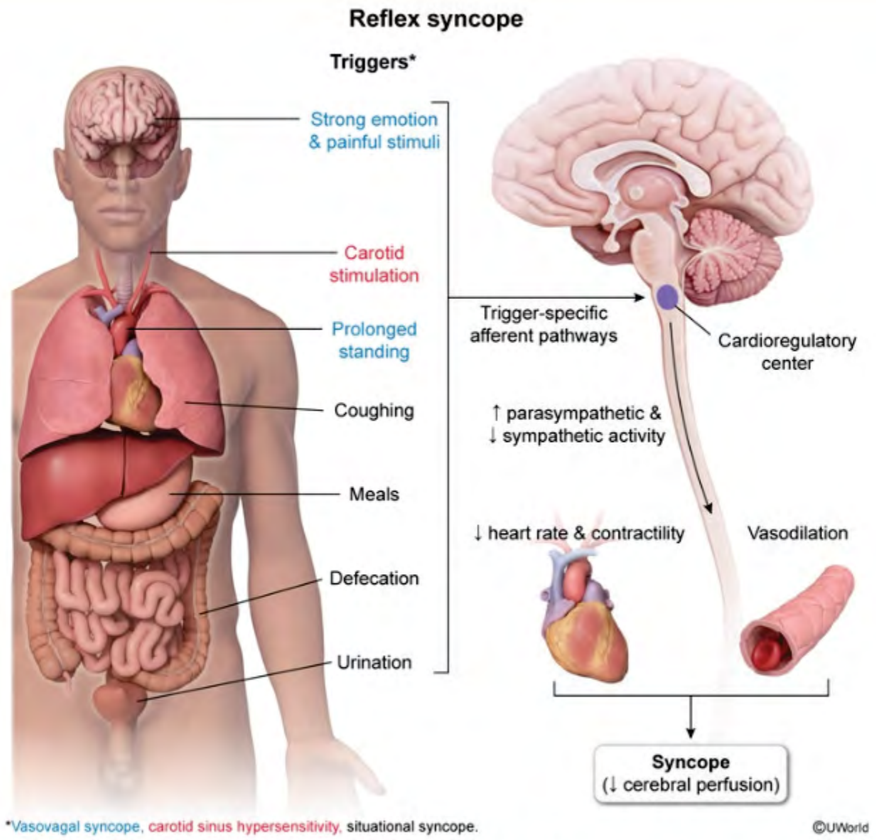

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A 77-year-old man comes to the office after he was found to have high blood pressure during a health fair. The patient denies any medical problems and feels proud that he has not needed to see a doctor for many years. His blood pressure is 170/70 mm Hg and pulse is 74/min. Other physical examination findings are normal. The patient is started on a medication to treat his hypertension. During his follow-up visit 3 weeks later, he reports bilateral leg swelling. He has no chest pain, shortness of breath, or abdominal symptoms. His blood pressure is 135/65 mm Hg and pulse is 80/min. Cardiopulmonary examination is normal, but there is bilateral, symmetrical, 2+ pitting lower extremity edema. Laboratory studies show serum creatinine of 0.8 mg/dL, and urinalysis is negative for proteinuria. Which of the following was most likely prescribed to treat this patient's hypertension?

- A. Amlodipine
- B. Eplerenone
- C. Hydrochlorothiazide
- D. Ramipril
- E. Torsemide
- F. Valsartan

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- ✓ A. Amlodipine (46%)
- B. Eplerenone (4%)
- C. Hydrochlorothiazide (15%)
- D. Ramipril (22%)
- E. Torsemide (6%)
- F. Valsartan (5%)

Correct

46% Answered correctly

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Explanation

Adverse effects of common antihypertensive medications	
Drugs	**Adverse effects**

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Adverse effects of common antihypertensive medications	
Drugs	**Adverse effects**
Thiazide diuretics	• Acute kidney injury • Electrolyte disturbances (hyponatremia, hypokalemia) • Hyperuricemia & acute gout • Elevated glucose & cholesterol levels
ACE inhibitors	• Cough • Angioedema • Hyperkalemia
Angiotensin II receptor blockers	• Hyperkalemia • Cough (rare)
Calcium channel blockers (eg, amlodipine, nifedipine)	• Peripheral edema • Dizziness or light-headedness
Beta blockers	• Bronchospasm • Bradycardia • Fatigue • Sexual dysfunction

Dihydropyridine calcium channel blockers (eg, amlodipine, nifedipine) are effective antihypertensive agents commonly used as monotherapy or in combination with other agents for treatment of hypertension. The major side effects associated with use of calcium channel blockers include headache, flushing, dizziness, and **peripheral edema**. Development of peripheral edema is related to preferential dilation of precapillary vessels (arteriolar dilation), which leads to increased capillary hydrostatic pressure and fluid extravasation into the interstitium.

Renin-angiotensin-system blockers (eg, ACE inhibitors or angiotensin II receptor blockers [ARBs]) cause

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- Sexual dysfunction

Dihydropyridine calcium channel blockers (eg, amlodipine, nifedipine) are effective antihypertensive agents commonly used as monotherapy or in combination with other agents for treatment of hypertension. The major side effects associated with use of calcium channel blockers include headache, flushing, dizziness, and **peripheral edema**. Development of peripheral edema is related to preferential dilation of precapillary vessels (arteriolar dilation), which leads to increased capillary hydrostatic pressure and fluid extravasation into the interstitium. Renin-angiotensin system blockers (eg, ACE inhibitors or angiotensin II receptor blockers [ARBs]) cause postcapillary venodilation and can normalize the increased capillary hydrostatic pressure, reducing the risk of peripheral edema in patients taking calcium channel blockers.

(Choice B) Eplerenone is an aldosterone antagonist and functions similarly to spironolactone as a potassium-sparing diuretic. The most frequent side effects of eplerenone are hyperkalemia, increased creatinine, and gynecomastia (~1% versus 10% with spironolactone).

(Choices C and E) The most common side effects of thiazide (eg, hydrochlorothiazide) and loop diuretics (eg, torsemide) are electrolyte abnormalities such as hypokalemia, hyponatremia, and hypomagnesemia. They do not cause peripheral edema.

(Choices D and F) The potential side effects of ACE inhibitors (eg, ramipril) and ARBs (eg, valsartan) include cough, hypotension, hyperkalemia, and angioedema. The risk of cough and angioedema is much lower with ARBs. These medications do not cause peripheral edema.

Educational objective:
Amlodipine is a dihydropyridine calcium channel blocker commonly used as monotherapy or in combination with other agents for treatment of hypertension. Major side effects include headache, flushing, dizziness, and peripheral edema.

References

- Vasodilatory edema: a common side effect of antihypertensive therapy.
- Peripheral edema associated with calcium channel blockers: incidence and withdrawal rate - a meta-analysis of

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A 36-year-old woman, gravida 2 para 1, at 30 weeks gestation comes to the office for a prenatal visit. The patient has noticed some ankle swelling at the end of the day that is relieved with elevating her legs, but she has no other concerns. The pregnancy has been uncomplicated, and her only medications are a prenatal vitamin and iron supplement. The patient has no chronic medical conditions. She does not use tobacco or alcohol. Blood pressure is 118/78 mm Hg and pulse is 72/min. BMI is 30 kg/m^2. Fetal heart tones are normal. There is trace bilateral pedal edema to the shins. Physical examination is otherwise unremarkable. Urinalysis shows no protein. Compared to a nonpregnant state, which of the following cardiac changes are most likely to be found in this patient?

	Cardiac output	Preload	Afterload
○ A.	↓	↓	No change
○ B.	↓	No change	↓
○ C.	No change	↓	↓
○ D.	No change	↑	↑
○ E.	↑	↑	↓
○ F.	↑	↑	↑

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	Cardiac output	Preload	Afterload	
✗ A.	↓	↓	No change	(3%)
B.	↓	No change	↓	(0%)
C.	No change	↓	↓	(2%)
D.	No change	↑	↑	(2%)
✓ E.	↑	↑	↓	(68%)
F.	↑	↑	↑	(21%)

Incorrect
Correct answer
E

68%
Answered correctly

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Explanation

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Maternal cardiopulmonary adaptations to pregnancy	

Maternal adaptations	• Cardiac
	  ◦ ↑ Cardiac output
	  ◦ ↑ Plasma volume
	  ◦ ↓ Systemic vascular resistance
	• Respiratory
	  ◦ ↑ Tidal volume
	  ◦ ↓ Functional residual capacity (elevation of diaphragm)

Clinical manifestations	• Peripheral edema • ↓ Blood pressure • ↑ Heart rate • Systolic ejection murmur • Dyspnea

During **pregnancy**, the increased metabolic demand required for fetal growth and development leads to multiple physiologic maternal adaptations, particularly **cardiovascular changes**. These changes both benefit the fetus and protect the patient against the risks of delivery (eg, hemorrhage).

The driving force behind maternal hemodynamic changes is a significant **decrease in systemic vascular resistance** (SVR) due to both increased release of peripheral vasodilators (eg, nitric oxide, prostacyclin) and formation of a high-flow, low-resistance uteroplacental circuit (increases blood flow to the placenta and fetus). There is also significantly **increased blood volume**. These changes have the following effects on cardiac preload and afterload:

- Both increased blood volume and decreased SVR (which allows blood to return to the heart more quickly and easily) contribute to increased cardiac venous return (ie, **increased preload**).

The marked reduction in SVR leads to decreased blood pressure (ie, **reduced afterload**).

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- Dyspnea

During **pregnancy**, the increased metabolic demand required for fetal growth and development leads to multiple physiologic maternal adaptations, particularly **cardiovascular changes**. These changes both benefit the fetus and protect the patient against the risks of delivery (eg, hemorrhage).

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- Both increased blood volume and decreased SVR (which allows blood to return to the heart more quickly and easily) contribute to increased cardiac venous return (ie, **increased preload**).
- The marked reduction in SVR leads to decreased blood pressure (ie, **reduced afterload**).

Both the increased preload and decreased afterload facilitate an increase in stroke volume, which is the primary cause of **increased cardiac output** (ie, stroke volume x heart rate) in early pregnancy. As the pregnancy progresses, the stroke volume decreases but maternal heart rate gradually increases, contributing to an overall increase in cardiac output of up to 30%-50% during pregnancy.

Educational objective:
During pregnancy, increased metabolic demands lead to multiple physiologic cardiovascular changes, including decreased systemic vascular resistance and increased blood volume. These changes cause increased preload and decreased afterload, resulting in increased stroke volume and cardiac output. Heart rate also gradually increases during pregnancy and is the major contributor to increased cardiac output in late pregnancy.

References

- Cardiovascular physiology of pregnancy.

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A 64-year-old man comes to the emergency department due to flank discomfort and red urine. He has a history of hypertension and type 2 diabetes mellitus. Three months ago, the patient had an ischemic stroke and now has mild, residual, right-sided weakness. Serum creatinine is 0.9 mg/dL, and serum lactate dehydrogenase is elevated. Urine microscopy shows many red blood cells. The findings of a CT scan of the abdomen with contrast are shown in the image below:

Which of the following is the most likely cause of this patient's symptoms?

- A. Atrial fibrillation
- B. Deep venous thrombosis of the lower extremities
- C. Focal segmental glomerulosclerosis
- D. Hypersensitivity interstitial nephritis
- E. Pulmonary hypertension

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Which

A. Atrial fibrillation

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- D. Hypersensitivity interstitial nephritis
- E. Pulmonary hypertension

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Which of the following is the most likely cause of this patient's symptoms?

- ✓ A. Atrial fibrillation (48%)
- ✗ B. Deep venous thrombosis of the lower extremities (10%)
- C. Focal segmental glomerulosclerosis (25%)
- D. Hypersensitivity interstitial nephritis (12%)
- E. Pulmonary hypertension (2%)

Incorrect
Correct answer
A

48%
Answered correctly

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E. Pulmonary hypertension (2%)

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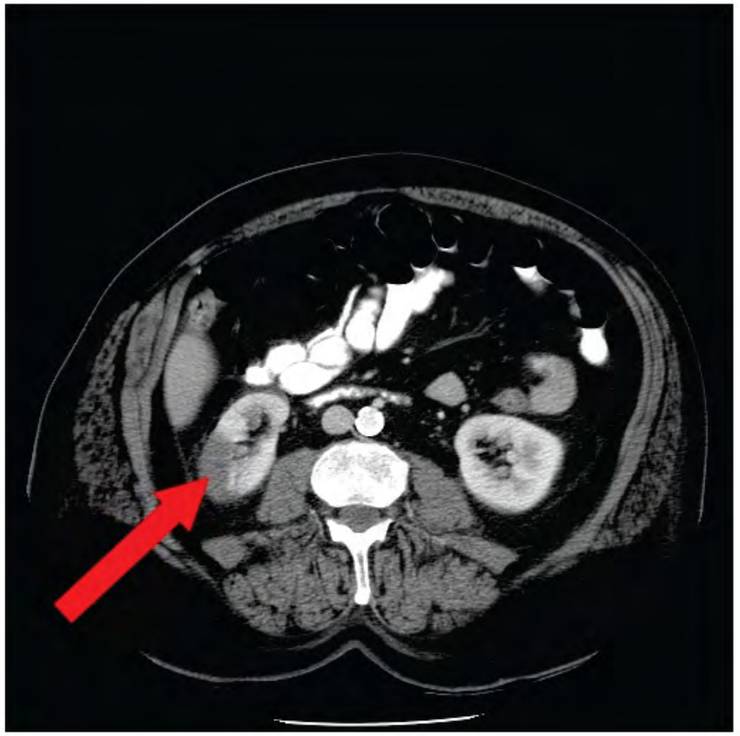

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infarction, which results from interruption of the normal blood supply to the kidney. The lack of collaterals between segmental renal arteries (**"end-organ"** blood supply) means that any interruption of blood flow can lead to

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This patient with flank pain, hematuria, and a wedge-shaped kidney lesion on CT scan likely has a **renal infarction**, which results from interruption of the normal blood supply to the kidney. The lack of collaterals between segmental renal arteries ("**end-organ**" blood supply) means that any interruption of blood flow can lead to coagulative infarcts. Common laboratory findings include elevated lactate dehydrogenase (reflecting cell necrosis), mild leukocytosis, and hematuria. Serum creatinine is usually normal due to compensation by the unaffected kidney. Macroscopically, renal infarcts appear as pale **wedges**, with the base (widest part) at the renal cortex and the apex pointing to the medulla.

The most common cause of renal infarction is **systemic thromboembolism** from the left atrium or ventricle. Thromboembolism is a common complication of **atrial fibrillation** because the irregular contractions lead to sluggish, uneven flow in the left atrium and facilitate clot formation. Emboli from atrial fibrillation, which can be paroxysmal (thereby going undiagnosed), may have also caused this patient's recent stroke. The **brain and kidneys** are more likely than other organs to suffer embolic infarctions because they are perfused at a higher rate.

(Choice B) Lower extremity venous thromboembolism can lead to pulmonary embolism because returning blood passes through the right heart and subsequently to the lungs. Although communication from the right to the left side of the heart (eg, atrial or ventricular septal defect) can allow for systemic (paradoxical) embolus to the brain or kidneys, this is relatively rare.

(Choice C) Focal segmental glomerulosclerosis manifests as nephrotic syndrome. Urinary loss of certain natural anticoagulants (eg, antithrombin III) leads to a hypercoagulable state. However, this rarely causes systemic embolization and patients usually have elevated creatinine.

(Choice D) Acute interstitial nephritis, a hypersensitivity reaction that can occur 1-2 weeks after starting a new medication, presents with fever, rash, elevated creatinine, eosinophilia, and eosinophiluria.

(Choice E) Pulmonary hypertension can cause right-sided heart failure (cor pulmonale) and is sometimes associated with secondary polycythemia, causing increased blood viscosity. Although thrombotic complications may occur, embolic complications are less common.

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(Choice E) Pulmonary hypertension can cause right-sided heart failure (cor pulmonale) and is sometimes associated with secondary polycythemia, causing increased blood viscosity. Although thrombotic complications may occur, embolic complications are less common.

Educational objective:
Renal infarction presents with flank pain, hematuria, elevated lactate dehydrogenase, and a wedge-shaped kidney lesion on CT scan. The most common cause of renal infarction is systemic thromboembolism, often due to thrombus formation during atrial fibrillation. The brain and kidneys are more likely than other organs to suffer embolic infarctions because they are perfused at a higher rate.

Pathology Cardiovascular System Renal infarction

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A 67-year-old man is brought to the emergency department by his son after a syncopal episode. The son was helping his father clean out the garage when the father said he felt dizzy. As the son was helping him into a chair, the patient lost consciousness. He woke up spontaneously about a minute later without any disorientation or confusion. ECG demonstrates bradycardia with a regular rhythm and narrow QRS complexes. There is complete desynchronization between the P waves and QRS complexes. Which of the following locations is most likely pacing this patient's ventricles?

- A. His bundle
- B. Left bundle branch
- C. Left ventricular muscle
- D. Purkinje system
- E. Sinoatrial node

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- ✓ A. His bundle (52%)
- B. Left bundle branch (5%)
- C. Left ventricular muscle (8%)
- D. Purkinje system (25%)
- E. Sinoatrial node (7%)

Correct

52% Answered correctly

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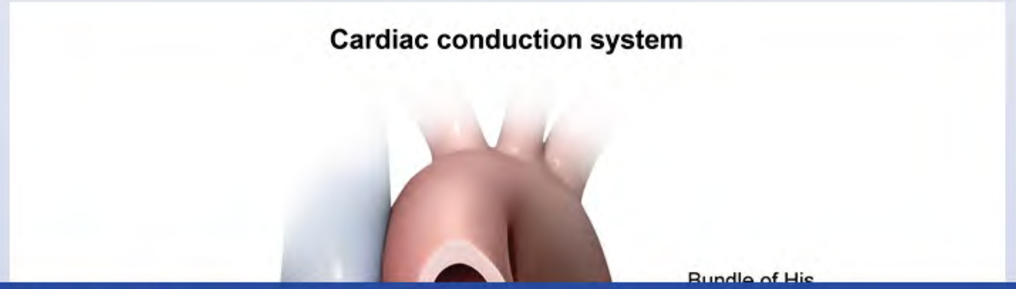
Explanation



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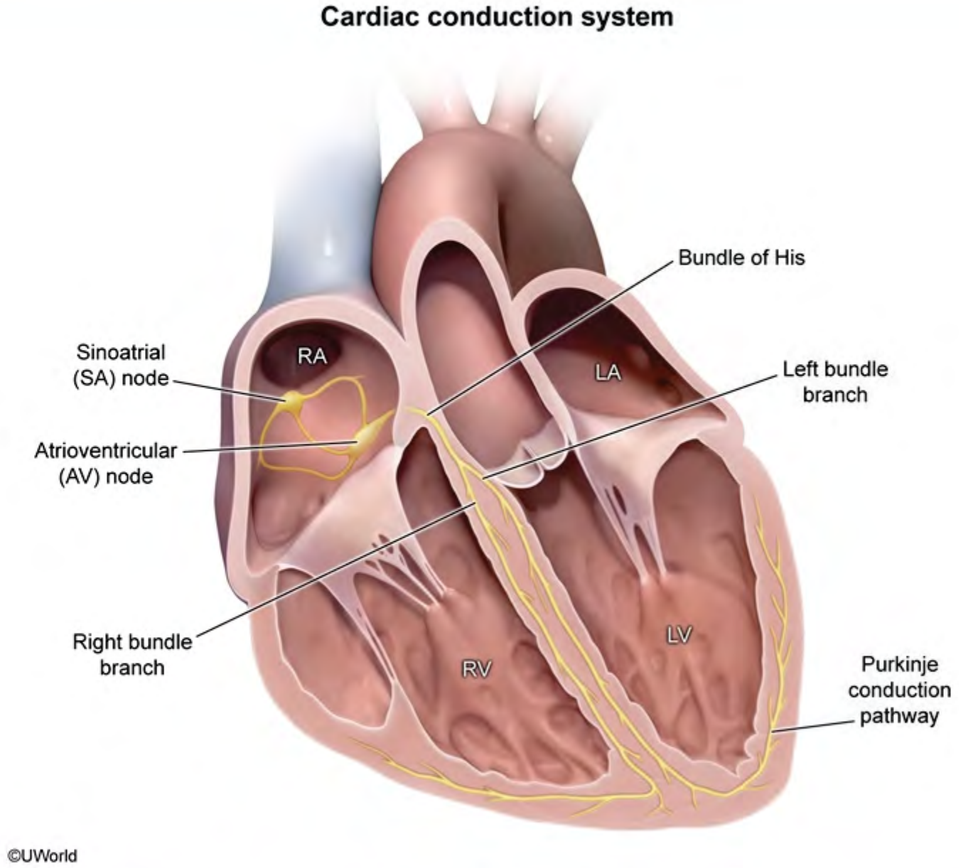

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The electrical impulses in the **myocardial conduction system** are normally initiated by the sinoatrial (SA) node at a rate of 60-100/min. These impulses are then transmitted through the atria to the atrioventricular (AV) node, then on to the His bundle, bundle branches, Purkinje fibers, and ventricular myocardium. Most of the conduction system has its own **intrinsic pacemaker**, which is normally suppressed by the more rapid SA node pacemaker but triggers when a signal from further up the conduction system is not received.

This patient's ECG shows complete **dissociation of P waves and QRS complexes** consistent with **third-degree (complete) AV block**. In third-degree AV block, electrical impulses coming from the SA node are blocked before being transmitted to the ventricles; the **point of blockade usually is within the AV node or His bundle**. The cells located immediately distal to the blockade (eg, in the His bundle) never receive the impulse from the SA node and therefore begin generating their own pacemaker impulse that is transmitted to the ventricles.

On ECG, the SA node impulses continue to march out as P waves, and the His bundle impulses generate QRS complexes (an **escape rhythm**) at the intrinsic rate of the His bundle pacemaker (eg, 40-60/min). Because the atria and the ventricles are not communicating, the P waves and QRS complexes have no relation to one another.

(Choices B and D) When electrical impulses are initiated below the AV node and His bundle, the heart rate typically slows to 25-40/min (based on the intrinsic pacemaker rate of the bundle branches and Purkinje system). On ECG, the QRS complexes are typically wide compared to the narrow QRS complexes initiated by the AV node and His bundle.

(Choice C) The ventricular myocardium does not have an intrinsic pacemaker and typically must receive electrical impulses from the Purkinje system.

(Choice E) When the SA node is initiating impulses through a normal conduction system, the heart rate is 60-100/min. ECG shows P waves quickly followed by a narrow QRS complex, representing normal atrial depolarization followed by normal ventricular depolarization (normal sinus rhythm).

Educational objective:

The sinoatrial node is the main pacemaker of the cardiac conduction system, typically initiating electrical impulses

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Educational objective:
The sinoatrial node is the main pacemaker of the cardiac conduction system, typically initiating electrical impulses at a rate of 60-100/min. Other parts of the conduction system (eg, atrioventricular node, His bundle) have their own intrinsic pacemakers, and they initiate impulses at a slower rate when impulses from the sinoatrial node are blocked.

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conduction pathway

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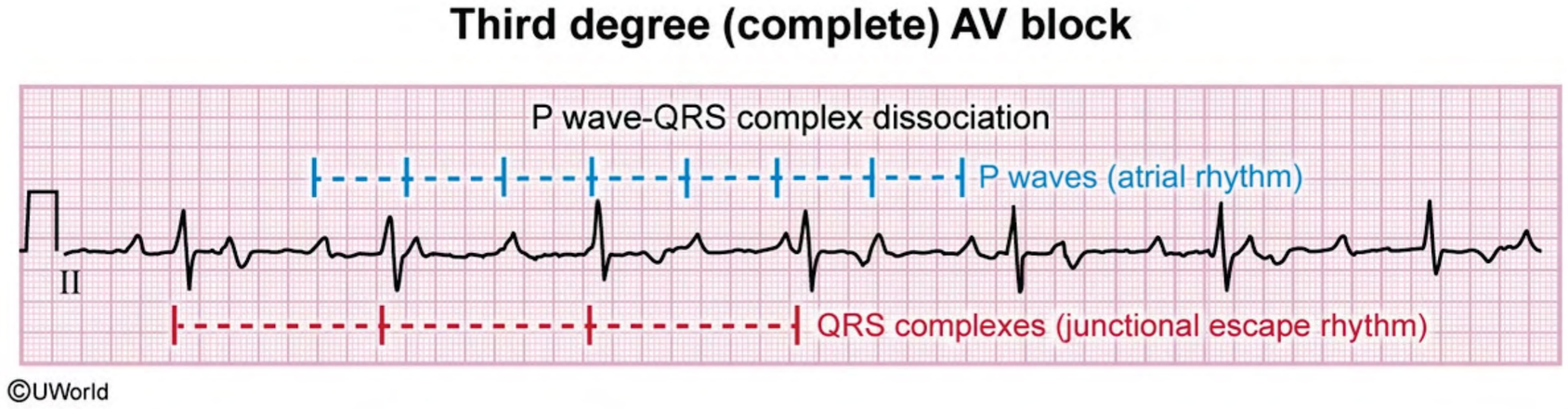

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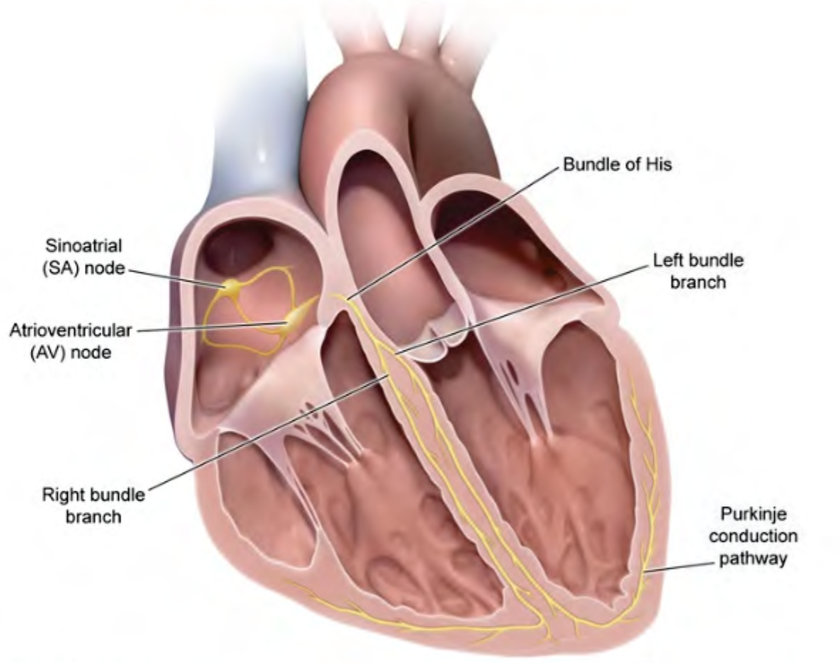
Intrinsic pacemaker rates of the conduction system

SA node	60-100 bpm
AV node & His bundle	40-60 bpm
Bundle branches & Purkinje system	25-40 bpm



bpm = beats per minute.

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Educational objective:

The sinoatrial node is the main pacemaker of the cardiac conduction system, typically initiating electrical impulses

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An 82-year-old man is brought to the emergency department after a syncopal episode. He has had no chest pain or dyspnea but he has constipation of recent onset. He was hospitalized 2 weeks ago for atrial fibrillation with rapid ventricular response and was discharged home with oral medications after appropriate management. Medical history is also significant for hypertension and severe chronic obstructive pulmonary disease requiring home supplemental oxygen. Blood pressure is 105/60 mm Hg and pulse is 50/min. Examination reveals bilaterally decreased breath sounds, no wheezing, and normal heart sounds. ECG shows new-onset second-degree atrioventricular block. Which of the following drugs is the most likely cause of his current condition?

- A. Amlodipine
- B. Diltiazem
- C. Hydrochlorothiazide
- D. Lidocaine
- E. Propranolol
- F. Terazosin
- G. Valsartan

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- ✗ A. Amlodipine (10%)
- ✓ B. Diltiazem (50%)
- C. Hydrochlorothiazide (1%)
- D. Lidocaine (4%)
- E. Propranolol (30%)
- F. Terazosin (1%)
- G. Valsartan (0%)

Incorrect
Correct answer
B

50%
Answered correctly

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Time Spent

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Explanation

This patient's **constipation** and new-onset second-degree **atrioventricular (AV) block** (causing syncope) are likely adverse effects of therapy with a **nondihydropyridine calcium channel blocker** (CCB) (eg, diltiazem, verapamil). These drugs are frequently used to treat hypertension, angina pectoris, and supraventricular

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This patient's **constipation** and new-onset second-degree **atrioventricular (AV) block** (causing syncope) are likely adverse effects of therapy with a **nondihydropyridine calcium channel blocker** (CCB) (eg, diltiazem, verapamil). These drugs are frequently used to treat hypertension, angina pectoris, and supraventricular arrhythmias, including atrial fibrillation (as in this patient).

Nondihydropyridine CCBs exert their primary action by blocking the **L-type calcium channels**, thereby decreasing phase 0 depolarization and conduction velocity in the sinoatrial and AV nodes. This leads to **slowing of the sinus rate** and slowing of conduction through the AV node, which can cause bradycardia and varying degrees of AV block. The drugs also have a **negative inotropic effect** and are relatively contraindicated in patients with left ventricular systolic dysfunction. Constipation, which is typically more prominent with verapamil than diltiazem, likely occurs due to reduced contraction of colonic smooth muscle.

(Choice A) Amlodipine is a dihydropyridine CCB that has a predominant vasodilator effect with minimal negative effect on cardiac conduction and contractility. Major adverse effects include lightheadedness, flushing, and peripheral edema.

(Choice C) Hydrochlorothiazide is a thiazide diuretic used for the treatment of hypertension. It has no role in the management of atrial fibrillation. Adverse effects include hypokalemia and hyponatremia.

(Choice D) Lidocaine is occasionally used in the management of symptomatic ventricular arrhythmias, usually in the setting of acute myocardial infarction. It is not used to treat atrial arrhythmias.

(Choice E) Beta blockers are commonly used for heart rate control in patients with atrial fibrillation and can cause or worsen AV block. However, propranolol is a nonselective beta blocker that impairs bronchodilation and is typically avoided in patients with severe chronic obstructive pulmonary disease. In addition, constipation is not a significant adverse effect of beta blockers.

(Choice F) Terazosin is an alpha-1 adrenergic antagonist used in the treatment of benign prostatic hyperplasia and hypertension. Major adverse effects include lightheadedness and orthostatic hypotension.

(Choice G) Valsartan is an angiotensin II receptor blocker used in the management of hypertension and heart

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rate and slowing of conduction through the AV node, which can cause bradycardia and varying degrees of AV block. The drugs also have a **negative inotropic effect** and are relatively contraindicated in patients with left ventricular systolic dysfunction. Constipation, which is typically more prominent with verapamil than diltiazem, likely occurs due to reduced contraction of colonic smooth muscle.

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(Choice F) Terazosin is an alpha-1 adrenergic antagonist used in the treatment of benign prostatic hyperplasia and hypertension. Major adverse effects include lightheadedness and orthostatic hypotension.

(Choice G) Valsartan is an angiotensin II receptor blocker used in the management of hypertension and heart failure with reduced ejection fraction. Common adverse effects include hypotension and hyperkalemia.

Educational objective:
Common adverse effects of nondihydropyridine calcium channel blockers (eg, diltiazem, verapamil) include constipation, bradycardia, atrioventricular block (negative chronotropic effect), and worsening of heart failure in patients with left ventricular systolic dysfunction (negative inotropic effect).

Pharmacology Cardiovascular System Heart block

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The following vignette applies to the next **2** items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 41-year-old man comes to the emergency department due to 3 days of fever, chills, and nonproductive cough. The patient has also felt tired and weak during this period. He has a history of injection drug use and has been hospitalized several times for injection site cellulitis. Temperature is 38.3 C (101 F), blood pressure is 125/72 mm Hg, and pulse is 112/min and regular. Cardiopulmonary auscultation reveals scattered rhonchi and a cardiac murmur, which were not previously present. Laboratory evaluation shows leukocytosis, and chest x-ray reveals nodular opacities.

Item 1 of 2

Echocardiography of this patient is most likely to reveal which of the following findings?

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Interventricular septal defect
- D. Mitral regurgitation
- E. Pulmonic stenosis
- F. Tricuspid regurgitation

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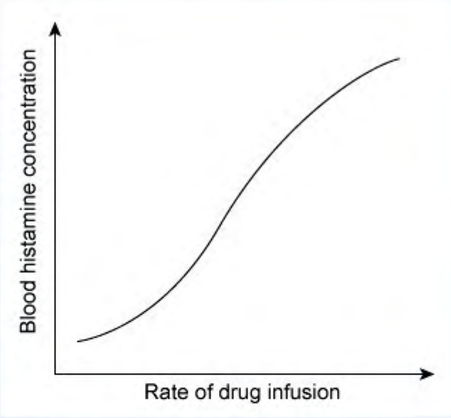
Item 2 of 2

The patient is started on an intravenous antibiotic for his condition but develops severe pruritus and a rash immediately upon infusion. The antibiotic used in this patient demonstrates the following dynamics when tested in several healthy volunteers:



Which of the following antibiotics was most likely given to this patient?

- A. Gentamicin
- B. Imipenem
- C. Penicillin G
- D. Trimethoprim-sulfamethoxazole
- E. Vancomycin

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Item 1 of 2

Echocardiography of this patient is most likely to reveal which of the following findings?

- A. Aortic regurgitation (0%)
- ✗ B. Aortic stenosis (0%)
- C. Interventricular septal defect (0%)
- D. Mitral regurgitation (8%)
- E. Pulmonic stenosis (0%)
- ✓ F. Tricuspid regurgitation (89%)

Incorrect
Correct answer
F

89%
Answered correctly

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Explanation

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Infective endocarditis in intravenous drug users	
Pathogenesis	• Injection of abrasive particulates → damaged valvular surface → increased bacterial adhesion • Right-sided valves at greatest risk (particularly tricuspid) ◦ Pulmonary capillaries prevent particulates from reaching the left side of the heart • Most common organism: *Staphylococcus aureus* (injected from skin)
Manifestations	• Nonspecific symptoms (eg, fever, chills, weight loss) • Septic pulmonary emboli (eg, cough, pleuritic pain, dyspnea) • Right-sided heart failure (eg, elevated jugular venous pressure, edema)

Injection drug use is associated with the concurrent injection of **abrasive particulate matter** and **microorganisms** into the bloodstream. Over time, injected particulate material denudes the surface of the heart valves, thereby creating a variegated surface for microorganism attachment. This dramatically increases the risk of **infective endocarditis** (IE). The **right-sided valves** are at greatest risk because pulmonary capillaries prevent large particulate matter from reaching the left side of the heart; the **tricuspid valve** is involved in >90% of cases.

Common manifestations of tricuspid valve IE include right-sided heart failure (eg, jugular venous distension, ascites, lower extremity edema) and/or **septic pulmonary emboli** (eg, multiple pulmonary nodules on chest x-ray). Cardiopulmonary evaluation usually reveals a blowing, holosystolic murmur heard best along the left lower sternal border that is intensified during inspiration and reduced with standing. Crackles and rhonchi are often present (in the setting of septic emboli). **Echocardiography** will show valvular vegetations, which frequently result in **regurgitation** due to incomplete valve closure.

(Choices A and D) Aortic and mitral valve IE is less common in injection drug users than tricuspid valve IE because the pulmonary vasculature filters out particulate matter and microorganisms. In addition, septic emboli from the left-side of the heart usually lodge in capillary beds (eg, cutaneous) prior to returning to the right-side of the heart; therefore, septic pulmonary emboli are less common.

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(Choice B) Aortic stenosis is common in older adults and usually causes dyspnea, angina, or syncope. Stenosis is not typically seen in IE, and left-sided IE does not usually present with pulmonary septic emboli.

(Choice C) An interventricular septal defect (VSD) can cause a left-to-right cardiac shunt, which can limit systemic blood flow and result in pulmonary hypertension. A VSD is less likely in this patient with intravenous drug use, signs of infection, and likely septic pulmonary emboli.

(Choice E) Pulmonary valve IE sometimes occurs in injection drug users; however, it generally causes pulmonary valve regurgitation (not stenosis) due to incomplete valvular closure. Patients with pulmonary stenosis often have signs of right-sided heart failure (eg, edema, ascites, elevated jugular venous distension) or dyspnea.

Educational objective:
Patients who inject drugs are at high risk for infective endocarditis (IE) due to the concurrent injection of abrasive particulate material and microorganisms. Most cases arise on the tricuspid valve. Patients with tricuspid valve IE often present with right-sided heart failure or septic pulmonary emboli. Echocardiography generally reveals valvular vegetations and incomplete valve closure (regurgitation).

Pathophysiology	Cardiovascular System	Endocarditis
Subject	System	Topic

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Item 2 of 2

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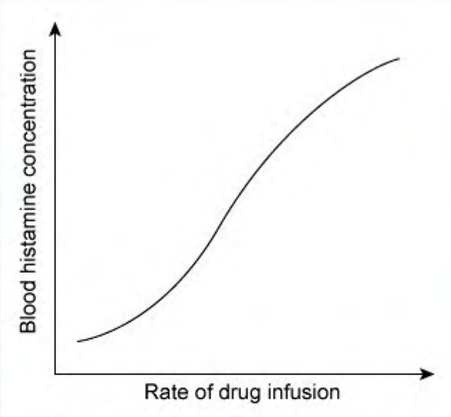

Which of the following antibiotics was most likely given to this patient?

- A. Gentamicin (1%)
- ✕ B. Imipenem (1%)
- C. Penicillin G (25%)
- D. Trimethoprim-sulfamethoxazole (3%)
- ✓ E. Vancomycin (67%)

Incorrect
Correct answer
E

67%
Answered correctly

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Vancomycin infusion reaction

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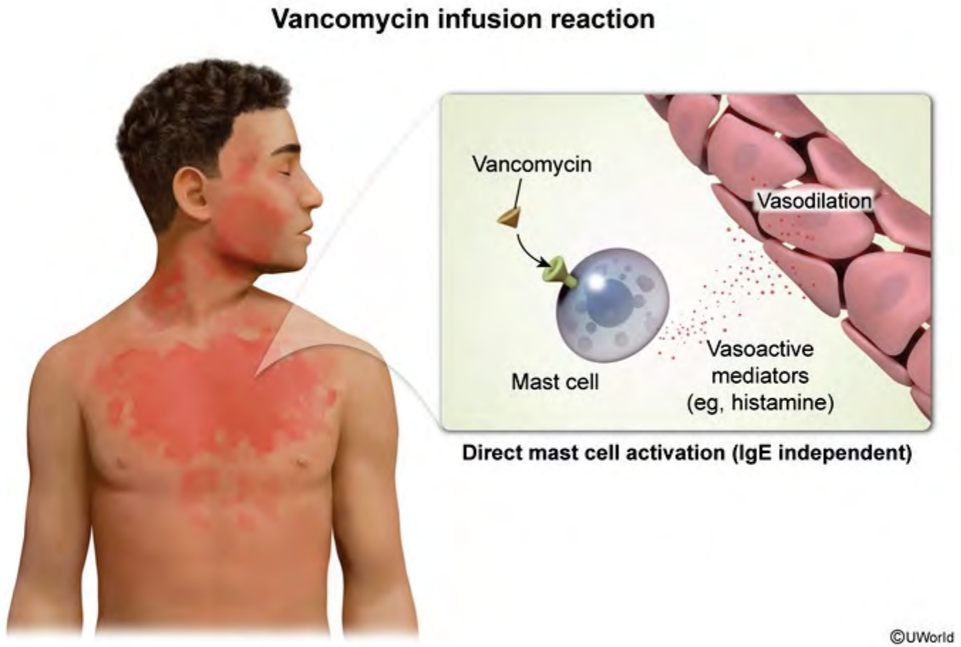

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Vancomycin inhibits cell wall synthesis of gram-positive bacteria by binding to the D-Alanine-D-Alanine terminus of nascent peptidoglycan pentapeptides, thereby inhibiting peptidoglycan elongation and cross-linking. Although vancomycin is generally well tolerated, a minority of patients develop **vancomycin infusion reaction** (VIR), a **non-allergic** drug reaction that occurs when vancomycin is **infused too rapidly**.

VIR arises due to the direct activation of mast cells by vancomycin, which results in the release of vasoactive mediators (eg, **histamine**). Manifestations typically arise within seconds/minutes of vancomycin infusion and include **flushing, erythema, and pruritus**, primarily in the neck and upper torso. Most cases resolve spontaneously with the cessation of vancomycin and the administration of diphenhydramine. Because VIR is not a true allergic reaction **(not IgE-mediated)**, vancomycin can be safely resumed at a **slower rate** once the symptoms have resolved.

(Choice A) Gentamicin, an aminoglycoside antibiotic, is associated with ototoxicity and nephrotoxicity.

(Choices B and C) Beta-lactam antibiotics (eg, imipenem, penicillin) generally cause IgE-mediated hypersensitivity reactions. In allergic reactions, histamine release is not linked to infusion rate, and patients usually have a history of previous exposure.

(Choice D) Trimethoprim-sulfamethoxazole can occasionally cause megaloblastic anemia, thrombocytopenia, leukopenia, and/or toxic epidermal necrolysis.

Educational objective:
Rapid infusion of vancomycin can lead to the direct activation of mast cells, which results in a massive release of histamine. This causes vancomycin infusion reaction, a nonallergic reaction marked by flushing, erythema, and pruritus within seconds or minutes of vancomycin infusion. Because this reaction is not IgE-mediated, vancomycin can be safely resumed at a slower rate once symptoms have resolved.

Pathophysiology Cardiovascular System Vancomycin

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A 37-year-old man is found unresponsive under a tree during a thunderstorm. He is not breathing when paramedics arrive on the scene. On examination, his pupils are fixed and dilated. Several cutaneous erythematous marks in a fern-leaf pattern are seen on his lower extremities. Second-degree burns are present on both arms. Cardiopulmonary resuscitation is started; however, the patient does not respond and is pronounced dead on arrival at the hospital. Which of the following is most likely to be the primary cause of his death?

- A. Cardiac arrhythmia
- B. Extensive deep-tissue burns
- C. Intracranial hemorrhage
- D. Myocardial contusion
- E. Pulmonary barotrauma

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- ✓ A. Cardiac arrhythmia (83%)
- ✗ B. Extensive deep-tissue burns (5%)
- C. Intracranial hemorrhage (6%)
- D. Myocardial contusion (2%)
- E. Pulmonary barotrauma (1%)

Incorrect
Correct answer
A

83%
Answered correctly

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Explanation

Lightning-related complications

Organ system	Complications
Cardiac	Cardiac arrest, arrhythmias
Neurologic	Peripheral nerve damage, seizures, confusion, respiratory arrest, autonomic dysfunction

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A 37-year-old man is found unresponsive under a tree during a thunderstorm. He is not breathing when

Exhibit Display

Lightning-related complications

Organ system	Complications
Cardiac	Cardiac arrest, arrhythmias
Neurologic	Peripheral nerve damage, seizures, confusion, respiratory arrest, autonomic dysfunction
Dermatologic	Lichtenberg figures (pathognomonic of lightning strike), burns
Musculoskeletal	Rhabdomyolysis, bone fractures, compartment syndrome
Other	Cataracts (late sequelae), ruptured tympanic membranes, curling ulcers

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respiratory arrest, autonomic dysfunction

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The finding of an unconscious person on wet ground or under trees during a thunderstorm suggests a lightning injury. This patient's Lichtenberg figures (erythematous cutaneous marks in a fern-leaf pattern) and second-degree burns confirm the suspicion. Although lightning injuries are rare, they are associated with a 25% fatality rate, and more than 70% of survivors have long-lasting complications. Two-thirds of lightning-related deaths occur within the first hour following injury; the most common causes are fatal arrhythmias and respiratory failure. A comprehensive and thorough physical examination should always be performed on lightning strike victims as injury patterns are widely variable and skin findings can significantly underestimate the extent of internal injury.

(Choice B) Superficial burns are common in lightning injury. In contrast, deep-tissue burns are relatively rare due to the short duration of electrical contact and the "flashover effect," the tendency of lightning to travel over the skin surface and discharge to the ground. This effect is responsible for the formation of Lichtenberg figures.

(Choice C) Intracranial hemorrhage only rarely occurs following lightning injury. Lightning strikes can damage the autonomic system and cause fixed and dilated or asymmetric pupils. These signs may not accurately reflect the extent of neurologic injury and should not be a reason to stop resuscitation.

(Choices D and E) Lightning induces rapid heating of the surrounding air, generating shock waves that can travel through the body and cause mechanical trauma. As a result, patients should be thoroughly assessed for skeletal fractures. Myocardial contusion and pulmonary barotrauma occur less frequently and would be extremely rare causes of death.

Educational objective:
Although lightning injuries are rare, they are associated with a 25% fatality rate. Two-thirds of lightning-related deaths occur within the first hour after injury, with fatal arrhythmias and respiratory failure as the most common causes. Patients with minor cutaneous involvement may still have major internal injury after lightning strikes and high-voltage electrical contact.

References

- Emergent care of lightning and electrical injuries.

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A 24-year-old man comes to the emergency department due to the sudden onset of palpitations. The patient says it feels like "my heart is racing." He had an episode similar to this a year ago that resolved spontaneously. Blood pressure is 126/74 mm Hg and pulse is 164/min. Rapid intravenous administration of a medication to this patient results in instantaneous resolution of the arrhythmia but is accompanied by short-lived flushing, burning in the chest, and shortness of breath. Which of the following medications was used to treat this patient's condition?

- A. Adenosine
- B. Amiodarone
- C. Digoxin
- D. Ibutilide
- E. Lidocaine
- F. Verapamil

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- ✓ A. Adenosine (75%)
- B. Amiodarone (6%)
- C. Digoxin (4%)
- D. Ibutilide (1%)
- E. Lidocaine (6%)
- F. Verapamil (5%)

Correct

75% Answered correctly

03 secs Time Spent

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Explanation

Pharmacotherapy for supraventricular arrhythmias	
Medication	**Short-term adverse effects**
Flecainide/propafenone (class IC)	• Monomorphic VT*

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Pharmacotherapy for supraventricular arrhythmias	
Medication	**Short-term adverse effects**
Flecainide/propafenone (class IC)	• Monomorphic VT*
Metoprolol (class II)	• Bradycardia & advanced AV block
Amiodarone (class III)	• Bradycardia • Hepatic toxicity, thyroid dysfunction
Ibutilide/dofetilide/sotalol (class III)	• Torsade de pointes
Verapamil/diltiazem (class IV)	• Bradycardia & advanced AV block • Decreased ventricular contractility • Constipation (verapamil)
Adenosine	• Flushing & hypotension • Bronchospasm • High-grade AV block
Digoxin	• Bradycardia & other arrhythmias • Nausea & vomiting ±

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Metoprolol (class II)	• Bradycardia & advanced AV block
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Verapamil/diltiazem (class IV)	• Bradycardia & advanced AV block • Decreased ventricular contractility • Constipation (verapamil)
Adenosine	• Flushing & hypotension • Bronchospasm • High-grade AV block
Digoxin	• Bradycardia & other arrhythmias • Nausea & vomiting ± visual disturbances

*Especially a concern with ischemic or structural heart disease.
AV = atrioventricular; **VT** = ventricular tachycardia.

This patient is exhibiting symptoms consistent with **paroxysmal supraventricular tachycardia (PSVT)**. As the name implies, PSVT comes on suddenly and the focus of automaticity lies above the ventricles (ie, atria or atrioventricular node). When due to development of a **reentrant circuit**, the tachyarrhythmia may be terminated

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Adenosine is a coronary arteriole vasodilator, but it also affects cardiac conduction. As an anti-arrhythmic, it acts by hyperpolarizing the nodal pacemaker and conducting cells to briefly **block conduction through the atrioventricular node**. Its effects are rapid-onset and short-lived (half-life <10 seconds); by temporarily blocking signal conduction from the atria to the ventricles, administration of the drug will often terminate the reentrant circuit and lead to conversion of PSVT to normal sinus rhythm.

The most frequent **adverse effects** associated with adenosine are flushing, chest burning (due to bronchospasm), hypotension, and high-grade atrioventricular block.

(Choice B) Amiodarone (class III anti-arrhythmic) is one of the broadest-spectrum anti-arrhythmic drugs available, and can be used to treat both supraventricular (eg, PSVT, atrial fibrillation) and ventricular arrhythmias. It has a wide range of adverse effects, including photodermatitis, blue/gray skin discoloration, pulmonary fibrosis, liver toxicity, and hyper- or hypothyroidism.

(Choice C) Digoxin slows conduction through the atrioventricular node and is useful in treating supraventricular tachycardia, especially atrial fibrillation. It causes a well-characterized toxicity in overdose; symptoms include fatigue, yellow-tinted vision, nausea/vomiting, diarrhea, and confusion.

(Choice D) Ibutilide (class III anti-arrhythmic) is used in the treatment of atrial fibrillation and flutter. Its principal adverse effect is QTc prolongation leading to torsades de pointes.

(Choice E) Lidocaine (class IB anti-arrhythmic) is sometimes used for ventricular arrhythmias but has little utility for supraventricular arrhythmias. Lidocaine toxicity most commonly causes neurologic symptoms or arrhythmias.

(Choice F) Verapamil (class IV anti-arrhythmic) is the most cardioselective of the calcium channel blockers, and is often used in the treatment of supraventricular tachycardia. Common adverse reactions noted with verapamil are

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(Choice F) Verapamil (class IV anti-arrhythmic) is the most cardioselective of the calcium channel blockers, and is often used in the treatment of supraventricular tachycardia. Common adverse reactions noted with verapamil are bradycardia and constipation. Because verapamil decreases cardiac contractility, it can also exacerbate systolic heart failure.

Educational objective:
Adenosine causes hyperpolarization of the nodal pacemaker to briefly block conduction through the atrioventricular node, and it is effective in the initial treatment of paroxysmal supraventricular tachycardia. Common adverse effects include flushing, chest burning (due to bronchospasm), hypotension, and high-grade atrioventricular block.

References

- Diagnosis and management of common types of supraventricular tachycardia.

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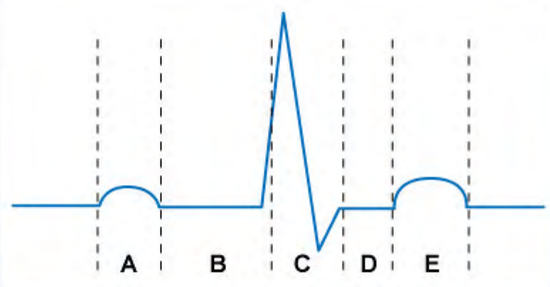
A 21-year-old man comes to the office due to recurrent heart palpitations that start and stop abruptly. Further evaluation is indicative of an abnormal conduction pathway in the patient's heart that bypasses the atrioventricular node. Which of the following portions of this patient's ECG is most likely to be affected during normal sinus rhythm?



- A. A
- B. B
- C. C
- D. D
- E. E

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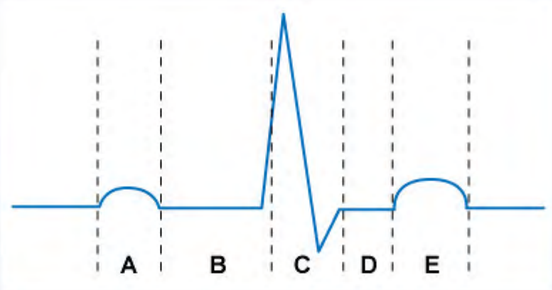

- A. A (9%)
- ✓ B. B (71%)
- C. C (16%)
- D. D (1%)
- E. E (0%)

Correct

71% Answered correctly

05 secs Time Spent

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Explanation

Wolff-Parkinson-White triad

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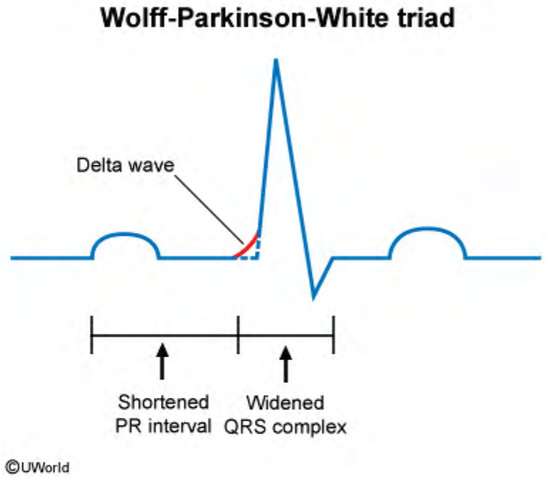

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Many patients with an accessory conduction pathway are asymptomatic and diagnosed only when a WPW pattern

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This young patient has paroxysmal supraventricular tachycardia (eg, repeated palpitation episodes) due to **Wolff-Parkinson-White** (WPW) syndrome. WPW syndrome is caused by an **accessory pathway** (bundle of Kent) that allows electrical impulses to bypass the atrioventricular node and cause **preexcitation** of the ventricles. This preexcitation leads to characteristic findings on baseline ECG during sinus rhythm, including a **shortened PR interval** (often <120 msec), slurred upslope of the QRS complex (**delta wave**), and a widened QRS complex.

Many patients with an accessory conduction pathway are asymptomatic and diagnosed only when a WPW pattern is incidentally recognized on ECG. However, some patients will develop symptomatic arrhythmias. Atrioventricular reentrant tachycardia (AVRT), a type of paroxysmal supraventricular tachycardia, is the most common arrhythmia that occurs with WPW. AVRT results from a rapid reentrant circuit that typically travels down the AV node to the ventricles and back up the accessory pathway, with patients often experiencing palpitations or a feeling of a racing heart.

(Choice A) When in sinus rhythm, a patient with an accessory conduction pathway should have a normal P wave on ECG.

(Choice C) The terminal portion of the QRS complex on ECG is typically unaffected in patients with an accessory conduction pathway.

(Choices D and E) The ST segment and T wave roughly correspond to the plateau and repolarization phases of the ventricular cardiomyocyte action potential, respectively. Because preexcitation causes abnormal ventricular depolarization, it follows that repolarization changes may be present as well, manifesting as alterations in the ST segment and T wave. However, these findings are less common than the usual WPW triad of ECG abnormalities and, when present, are nonspecific for diagnosing an accessory conduction pathway.

Educational objective:
An accessory atrioventricular conduction pathway causes ventricular preexcitation that manifests on ECG with a Wolff-Parkinson-White (WPW) pattern, consisting of a shortened PR interval, slurred upslope of the QRS complex (delta wave), and a widened QRS interval. Some patients with an accessory conduction pathway develop

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preexcitation leads to characteristic findings on baseline ECG during sinus rhythm, including a **shortened PR interval** (often <120 msec), slurred upslope of the QRS complex (**delta wave**), and a widened QRS complex.

Many patients with an accessory conduction pathway are asymptomatic and diagnosed only when a WPW pattern is incidentally recognized on ECG. However, some patients will develop symptomatic arrhythmias. Atrioventricular reentrant tachycardia (AVRT), a type of paroxysmal supraventricular tachycardia, is the most common arrhythmia that occurs with WPW. AVRT results from a rapid reentrant circuit that typically travels down the AV node to the ventricles and back up the accessory pathway, with patients often experiencing palpitations or a feeling of a racing heart.

(Choice A) When in sinus rhythm, a patient with an accessory conduction pathway should have a normal P wave on ECG.

(Choice C) The terminal portion of the QRS complex on ECG is typically unaffected in patients with an accessory conduction pathway.

(Choices D and E) The ST segment and T wave roughly correspond to the plateau and repolarization phases of the ventricular cardiomyocyte action potential, respectively. Because preexcitation causes abnormal ventricular depolarization, it follows that repolarization changes may be present as well, manifesting as alterations in the ST segment and T wave. However, these findings are less common than the usual WPW triad of ECG abnormalities and, when present, are nonspecific for diagnosing an accessory conduction pathway.

Educational objective:
An accessory atrioventricular conduction pathway causes ventricular preexcitation that manifests on ECG with a Wolff-Parkinson-White (WPW) pattern, consisting of a shortened PR interval, slurred upslope of the QRS complex (delta wave), and a widened QRS interval. Some patients with an accessory conduction pathway develop paroxysmal tachyarrhythmias, most commonly atrioventricular reentrant tachycardia, which constitutes WPW syndrome.

References

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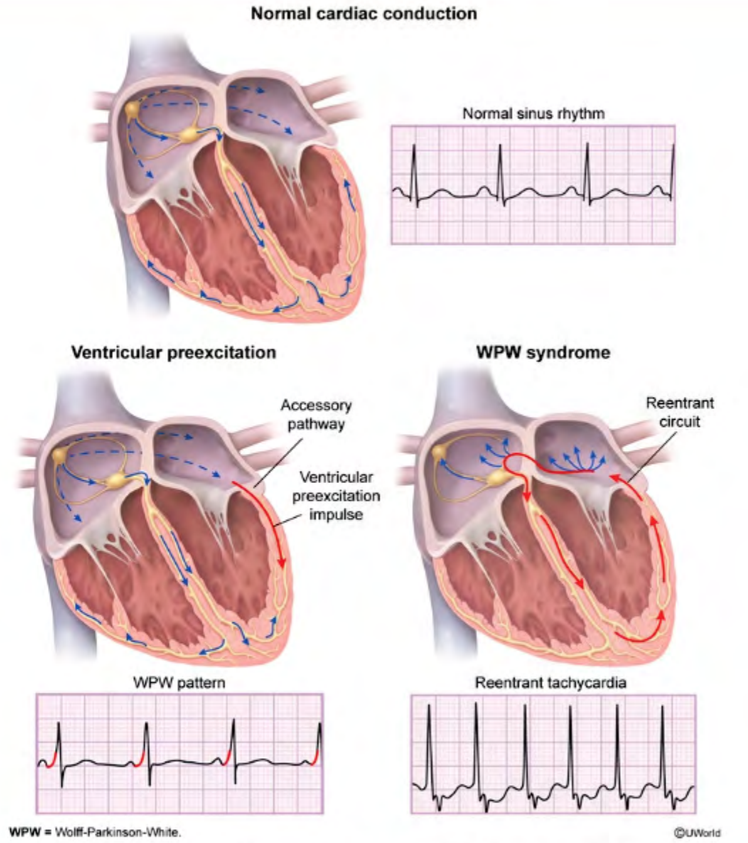

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Atrioventricular reentrant tachycardia (AVRT)

Pathophysiology	Key features
• Accessory pathway connecting atria & ventricles creates a macrorentrant circuit • Usually triggered by a PAC or PVC • Characteristic arrhythmia of WPW syndrome	• Rate 140-250 bpm • Regular R-R intervals • Retrograde P waves often visible • Usually narrow QRS complexes

PAC = premature atrial contraction; **PVC** = premature ventricular contraction; **WPW** = Wolff-Parkinson-White.

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A 46-year-old man is diagnosed with resistant hypertension. He had a comprehensive workup for secondary hypertension, which was unrevealing. The patient has tried multiple antihypertensive medications without significant effect. Past medical history is otherwise unremarkable. He agrees to enroll in a trial of a new long-acting medication that causes selective direct relaxation of the smooth muscle of arterioles but does not affect the veins. Which of the following adverse effects is most likely to be caused by the drug during the clinical trial?

- A. Angioedema
- B. Bradycardia
- C. Cold extremities
- D. Decreased cardiac output
- E. Persistent cough
- F. Sodium and fluid retention
- G. Transient hypertension

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- ✗ A. Angioedema (18%)
- B. Bradycardia (9%)
- C. Cold extremities (8%)
- D. Decreased cardiac output (14%)
- E. Persistent cough (6%)
- ✓ F. Sodium and fluid retention (33%)
- G. Transient hypertension (9%)

Incorrect
Correct answer
F

33%
Answered correctly

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Explanation

Selective **arteriolar vasodilators** (eg, hydralazine, minoxidil) lower blood pressure by reducing systemic vascular resistance. However, this effect is limited by subsequent stimulation of baroreceptors with resulting reflex **sympathetic activation**. This leads to increased heart rate, contractility, and cardiac output **(Choices B and D)**. In addition, sympathetic stimulation of the **renin-angiotensin-aldosterone** axis results in **sodium and fluid retention** with peripheral edema. These effects offset much of the blood pressure lowering effect of these drugs

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Explanation

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These agents are rarely used as monotherapy for chronic management of hypertension. However, they are useful acutely for patients with severely elevated blood pressure. They can also be given in combination with sympatholytics and diuretics to mitigate the side effects and provide synergistic blood pressure lowering in patients with resistant hypertension.

(Choices A and E) Persistent cough and angioedema are well recognized side effects of ACE inhibitors. Angioedema refers to acute, localized subcutaneous or submucosal edema caused by increased vascular permeability. It differs from the chronic peripheral edema due to sodium retention seen with arteriolar dilators.

(Choice C) Beta blockers decrease cardiac output, leading to peripheral vasoconstriction and resulting in cold hands and feet. However, direct arteriolar vasodilators enhance blood flow to peripheral tissues, including skin and muscle, and they can be used to relieve symptoms in patients with Raynaud phenomenon.

(Choice G) Although selective arteriolar vasodilators trigger reflex sympathetic activation, this does not raise blood pressure above baseline unless they are discontinued abruptly.

Educational objective:
Direct arteriolar vasodilators lower blood pressure but trigger reflex sympathetic activation and stimulate the renin-angiotensin-aldosterone axis. This results in tachycardia and edema. To counteract such compensatory effects, these agents are often given in combination with sympatholytics and diuretics.

Pharmacology Cardiovascular System Primary hypertension

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A 24-year-old man is evaluated due to an episode of syncope. He was jogging when he felt light-headed and passed out, but he did not sustain a head injury. The patient has had 2 similar episodes of light-headedness while jogging over the last year, but this was the first time he passed out. He considers himself in good health and has no other medical conditions. The patient does not use tobacco, alcohol, or recreational drugs. His father died suddenly at age 30. Vital signs are within normal limits. On physical examination, the patient has a harsh systolic murmur. The lungs are clear to auscultation. There is no peripheral edema. Transthoracic echocardiography shows asymmetric interventricular septal hypertrophy. This patient's symptoms are most likely explained by left ventricular outflow obstruction created by which of the following structures?

- A. Aortic valve cusp and interventricular septum
- B. Aortic valve cusp and papillary muscle
- C. Aortic valve cusps
- D. Ascending aorta and interventricular septum
- E. Mitral valve leaflet and interventricular septum

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- ✗ A. Aortic valve cusp and interventricular septum (42%)
- B. Aortic valve cusp and papillary muscle (2%)
- C. Aortic valve cusps (5%)
- D. Ascending aorta and interventricular septum (5%)
- ✓ E. Mitral valve leaflet and interventricular septum (43%)

Incorrect
Correct answer
E

43%
Answered correctly

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Explanation

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D. Ascending aorta and interventricular septum (5%)

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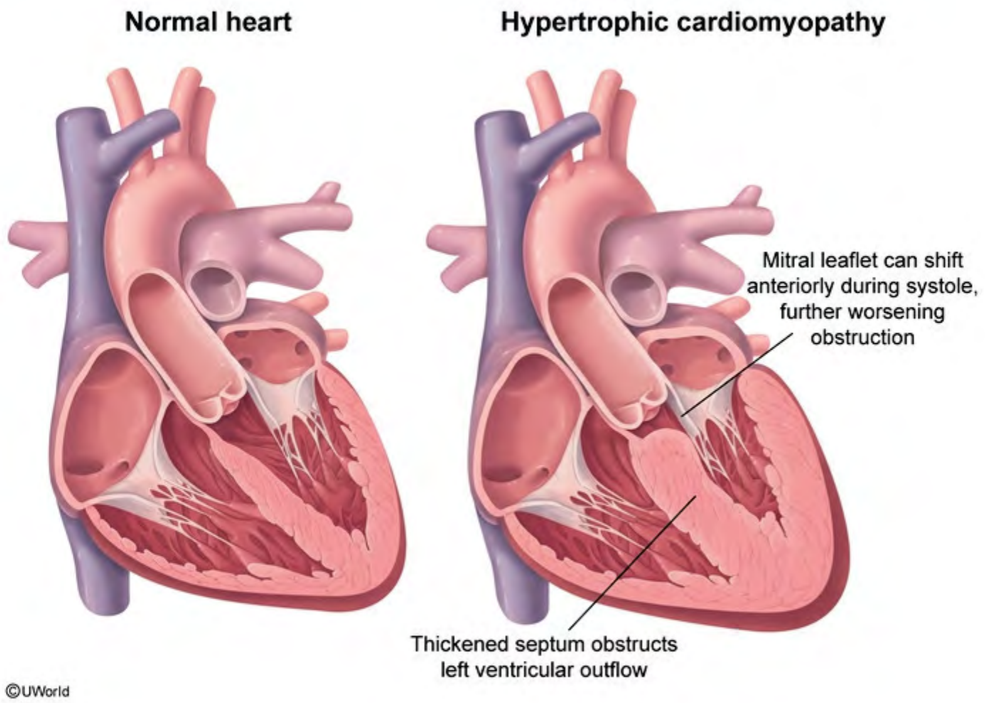

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This patient's presentation suggests **hypertrophic cardiomyopathy** (HCM), an autosomal dominant disorder

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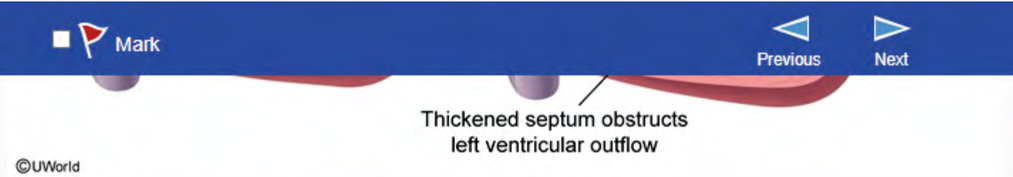

This patient's presentation suggests **hypertrophic cardiomyopathy** (HCM), an autosomal dominant disorder resulting from mutations in cardiac sarcomere proteins. HCM is characterized by asymmetric ventricular septal hypertrophy, which often causes **left ventricular outflow tract (LVOT) obstruction**. Systolic anterior motion of the **mitral valve** toward the **interventricular septum** can exacerbate the obstruction, with the anterior leaflet of the mitral valve physically blocking the LVOT.

Many patients with HCM are asymptomatic and may be diagnosed via an abnormal ECG or murmur during routine evaluation. However, patients can also experience exertional dyspnea, chest pain, fatigue, palpitations, lightheadedness, syncope, or sudden cardiac death (due to ventricular arrhythmias). Examination often reveals a **harsh crescendo-decrescendo systolic murmur** at the apex and left lower sternal border; the murmur is caused by blood flow through the narrowed LVOT. Because the degree of LVOT obstruction is dynamic and changes with left ventricular blood volume, the murmur varies in intensity with physiologic maneuvers.

(Choices A, B, and C) The aortic valve does not contribute to LV obstruction in HCM as the obstruction is subvalvular, resulting from an enlarged intraventricular septum and transient movement of the anterior mitral valve leaflet into the LVOT during ventricular systole. Valvular aortic stenosis often results from age-related leaflet calcification and can present with a murmur and symptoms similar to those of HCM, but it typically affects patients age >40 and does not involve enlargement of the interventricular septum.

(Choice D) Narrowing of the ascending aorta just distal to the aortic valve (ie, supravalvular aortic stenosis) is a characteristic cause of LV obstruction in Williams syndrome, a rare genetic disorder that does not involve enlargement of the interventricular septum. Abnormalities of the ascending aorta do not contribute to LV obstruction in HCM.

Educational objective:
In patients with hypertrophic cardiomyopathy, left ventricular outflow tract (LVOT) obstruction results from a hypertrophied interventricular septum and abnormal systolic anterior motion of the anterior leaflet of the mitral

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Educational objective:
In patients with hypertrophic cardiomyopathy, left ventricular outflow tract (LVOT) obstruction results from a hypertrophied interventricular septum and abnormal systolic anterior motion of the anterior leaflet of the mitral valve. The degree of LVOT obstruction is dynamic and varies with changes in left ventricular blood volume.

Pathology | Cardiovascular System | Hypertrophic cardiomyopathy

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Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	**Left ventricular blood volume**	**Murmur intensity**
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

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enlargement of the interventricular septum. Abnormalities of the ascending aorta do not contribute to LV obstruction in HCM.

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A 28-year-old professional endurance athlete comes to the office after his team physician detected a heart murmur. The patient has no symptoms and has no exercise limitations. Medical history is nonsignificant. Blood pressure is 110/66 mm Hg and pulse is 54/min. Physical examination shows a soft systolic murmur best heard at the left second intercostal space in the supine position. The murmur disappears when the patient is upright but reappears after brief exercise. Which of the following cardiovascular adaptations best explains this patient's finding?

- A. Decreased pulmonary vascular capacitance
- B. Decreased vagal tone
- C. Increased stroke volume
- D. Increased systemic vascular resistance
- E. Increased ventricular wall thickness

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- A. Decreased pulmonary vascular capacitance (13%)
- ✗ B. Decreased vagal tone (3%)
- ✓ C. Increased stroke volume (45%)
- D. Increased systemic vascular resistance (11%)
- E. Increased ventricular wall thickness (25%)

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Correct answer
C

45%
Answered correctly

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Explanation

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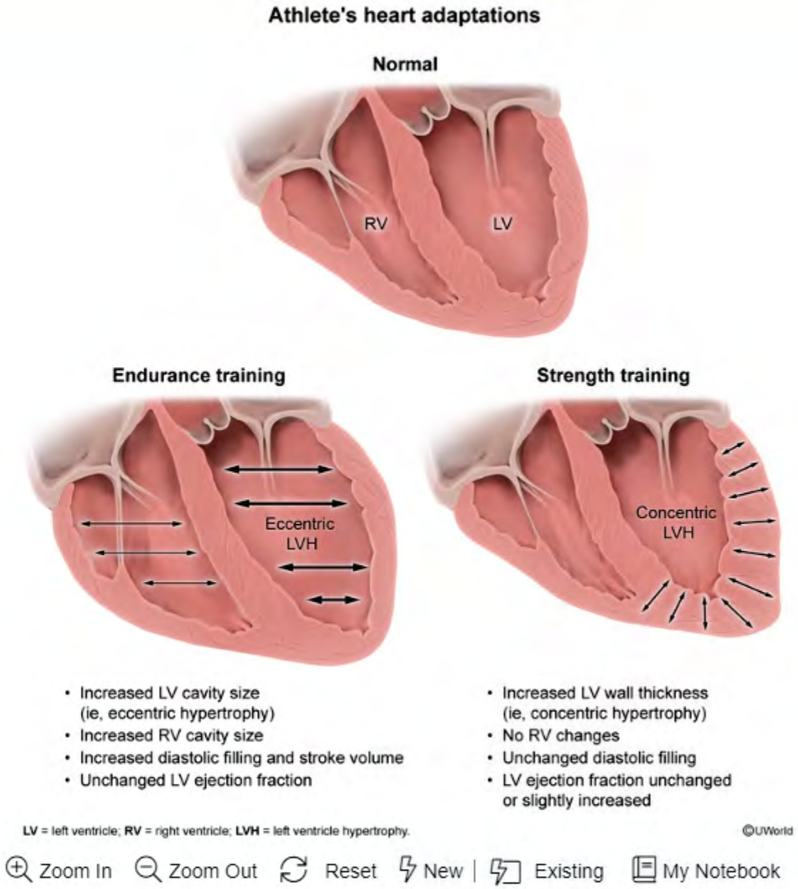

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This professional athlete most likely has the cardiovascular adaptations of **athlete's heart**. The soft systolic murmur likely represents a pulmonic flow murmur, a common finding due to the high stroke volume that develops with intensive **endurance training**.

The adaptations of athlete's heart differ depending on whether intensive endurance training (eg, long-distance running) or resistance training (eg, heavy weightlifting) is pursued. With endurance training, systemic vascular resistance (SVR) is reduced (due to vasodilation in skeletal muscle) and blood volume is increased, leading to **increased left ventricular (LV) volume load**. To compensate, LV **eccentric hypertrophy** (ie, LV wall lengthening) develops, which increases LV cavity size to increase diastolic filling capacity and facilitate **increased stroke volume** and cardiac output. Right ventricular (RV) cavity enlargement also takes place to increase diastolic filling and accommodate the increase in cardiac output; because the right ventricle and left ventricle are arranged in series and contract at the same rate, RV stroke volume must increase to match LV stroke volume and prevent backup of blood flow.

An increase in stroke volume is maintained at rest and allows for adequate cardiac output at **low resting heart rate** (eg, <55/min). The anterior location of the pulmonic valve and slightly smaller valve area compared to the aortic valve often create a **pulmonic flow murmur** due to the high stroke volume. The murmur is best heard at the left second intercostal space in the supine position and may disappear when venous return is decreased with standing.

(Choice A) Pulmonary vascular capacitance is increased with endurance training, allowing the pulmonary circulation to accommodate high flow while minimizing the afterload on the right ventricle.

(Choice B) Baseline vagal tone is increased with endurance training, facilitating low resting heart rate.

(Choices D and E) Intensive resistance training increases SVR during exercise (due to vascular compression by vigorously contracting muscles), which increases LV pressure load and leads to mild concentric hypertrophy (ie, LV wall thickening). Unlike pathologic concentric LV hypertrophy, the LV cavity remains normal in size and diastolic

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(Choice B) Baseline vagal tone is increased with endurance training, facilitating low resting heart rate.

(Choices D and E) Intensive resistance training increases SVR during exercise (due to vascular compression by vigorously contracting muscles), which increases LV pressure load and leads to mild concentric hypertrophy (ie, LV wall thickening). Unlike pathologic concentric LV hypertrophy, the LV cavity remains normal in size and diastolic function is maintained. LV stroke volume and ejection fraction remain normal or are slightly increased, and the right ventricle is unaffected. A flow murmur is not expected.

Educational objective:
Intensive endurance training causes cavity enlargement of both the left ventricle and right ventricle to facilitate an increase in stroke volume and cardiac output. The high stroke volume may cause a pulmonic valve flow murmur.

References

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A 55-year-old man comes to the emergency department due to an hour of intense substernal chest burning accompanied by sweating. He has a history of obesity and diet-controlled type 2 diabetes mellitus. On initial evaluation, blood pressure is 110/70 mm Hg and pulse is 60/min and regular. Oxygen saturation is 98% on room air. The patient is comfortable lying flat and no heart murmurs are heard. Lungs are clear on auscultation. Immediate ECG shows normal sinus rhythm with a 2-mm ST-segment elevation in leads II, III, and aVF. Aspirin and morphine are administered. As the patient is about to be transported to the catheterization lab for urgent percutaneous coronary intervention, he develops dizziness and the telemetry monitor shows sinus bradycardia at 40/min. The patient appears pale and diaphoretic and peripheral pulses are faint. Blood pressure is 70/40 mm Hg. Which of the following is the best immediate step for managing this patient's condition?

- A. Adenosine
- B. Alteplase
- C. Amiodarone
- D. Atropine
- E. Naloxone
- F. Physostigmine

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- A. Adenosine (14%)
- ✗ B. Alteplase (13%)
- C. Amiodarone (3%)
- ✓ D. Atropine (54%)
- E. Naloxone (13%)
- F. Physostigmine (1%)

Incorrect
Correct answer
D

54%
Answered correctly

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Explanation

This patient with ST elevation in leads II, III, and aVF has an acute **inferior wall myocardial infarction** (MI), which most commonly occurs due to occlusion of the right coronary artery (RCA). The RCA usually supplies blood to the

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This patient with ST elevation in leads II, III, and aVF has an acute **inferior wall myocardial infarction** (MI), which most commonly occurs due to occlusion of the right coronary artery (RCA). The RCA usually supplies blood to the sinoatrial node, the atrioventricular node, and most of the bundle of His; therefore, **conduction impairment** (eg, sinus bradycardia, atrioventricular block) is common in patients with inferior wall MI.

Sinus bradycardia is the most common arrhythmia in those with inferior wall MI (nearly 50% of patients); it occurs due to both nodal ischemia and **enhanced vagal tone** triggered by infarction of myocardial tissue. The bradycardia is usually transient and resolves with restoration of coronary blood flow; however, patients with hypotension or symptoms of bradycardia (eg, lightheadedness, syncope) should be managed with **intravenous atropine**. The **anticholinergic** effects of atropine counteract the enhanced vagal tone to increase heart rate and improve symptoms.

(Choice A) Adenosine hyperpolarizes the atrioventricular node to cause transient atrioventricular conduction delay. It is useful in the treatment of paroxysmal supraventricular tachycardia, but it is likely to worsen bradycardia.

(Choice B) Alteplase is a fibrinolytic agent indicated for the acute management of patients with ST-elevation MI who cannot undergo percutaneous coronary intervention (PCI) in a timely manner. PCI is associated with reduced mortality and a lower rate of complications (eg, cerebral hemorrhage) compared with fibrinolytic therapy and is the preferred intervention whenever possible.

(Choice C) Amiodarone is a class III antiarrhythmic agent that can be used for the management of ventricular arrhythmia (eg, ventricular tachycardia) in patients with acute MI. However, the drug causes negative chronotropy and is not used to treat bradyarrhythmias.

(Choice E) Naloxone, an opioid-receptor antagonist, is used to reverse the effects of opioid overdose (eg, somnolence, respiratory and cardiac depression). Although morphine may cause bradycardia, this patient's bradycardia is more likely due to his inferior wall MI.

(Choice F) Physostigmine, an acetylcholinesterase inhibitor that prolongs the effects of acetylcholine, is used in the treatment of myasthenia gravis. Its effects essentially oppose those of atropine; it would worsen the

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due to both nodal ischemia and **enhanced vagal tone** triggered by infarction of myocardial tissue. The bradycardia is usually transient and resolves with restoration of coronary blood flow; however, patients with hypotension or symptoms of bradycardia (eg, lightheadedness, syncope) should be managed with **intravenous atropine**. The **anticholinergic** effects of atropine counteract the enhanced vagal tone to increase heart rate and improve symptoms.

(Choice A) Adenosine hyperpolarizes the atrioventricular node to cause transient atrioventricular conduction delay. It is useful in the treatment of paroxysmal supraventricular tachycardia, but it is likely to worsen bradycardia.

(Choice B) Alteplase is a fibrinolytic agent indicated for the acute management of patients with ST-elevation MI who cannot undergo percutaneous coronary intervention (PCI) in a timely manner. PCI is associated with reduced mortality and a lower rate of complications (eg, cerebral hemorrhage) compared with fibrinolytic therapy and is the preferred intervention whenever possible.

(Choice C) Amiodarone is a class III antiarrhythmic agent that can be used for the management of ventricular arrhythmia (eg, ventricular tachycardia) in patients with acute MI. However, the drug causes negative chronotropy and is not used to treat bradyarrhythmias.

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(Choice F) Physostigmine, an acetylcholinesterase inhibitor that prolongs the effects of acetylcholine, is used in the treatment of myasthenia gravis. Its effects essentially oppose those of atropine; it would worsen the bradycardia in this patient.

Educational objective:
Conduction impairment is common with acute inferior wall myocardial infarction. Sinus bradycardia often occurs due to nodal ischemia and an increase in vagal tone triggered by infarction of myocardial tissue; the enhanced vagal tone can be counteracted by the anticholinergic effects of atropine.

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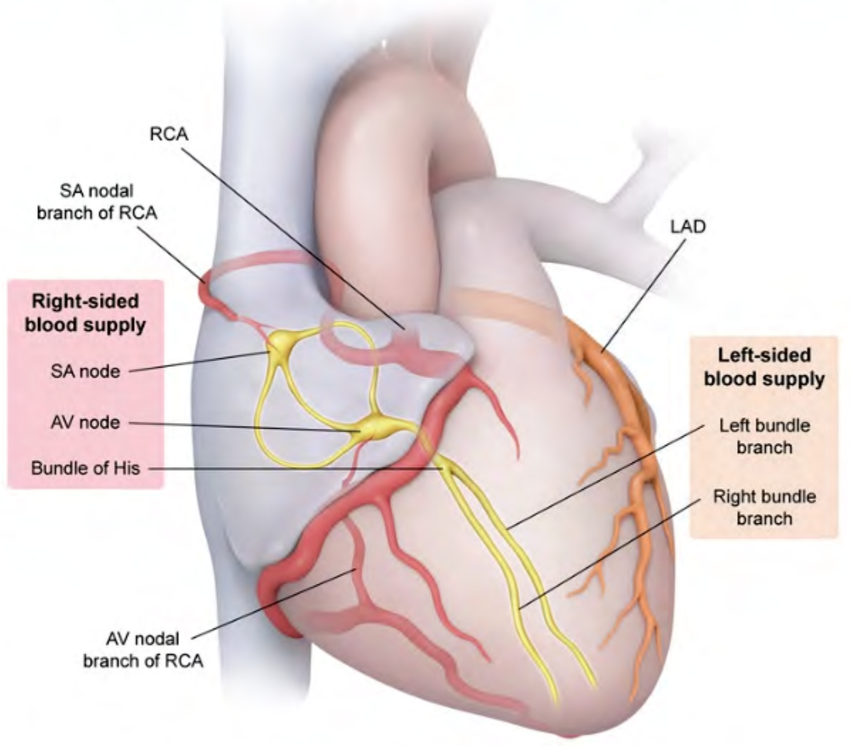
Typical blood supply to the cardiac conduction system



AV = atrioventricular; **LAD** = left anterior descending artery; **RCA** = right coronary artery; **SA** = sinoatrial.

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A 72-year-old man comes to the clinic due to episodic lightheadedness. The episodes typically occur in the morning when the patient adjusts his shirt collar or tightens his tie and he briefly feels like he is going to pass out. The patient had a thyroidectomy 5 years ago for a large benign tumor. He also has hypertension and takes amlodipine. Blood pressure is 135/78 mm Hg and pulse is 78/min. Physical examination reveals no heart murmurs or carotid bruits. ECG shows normal sinus rhythm. The patient is given an ambulatory ECG monitoring device. During symptomatic episodes, the device is most likely to show which of the following?

- A. Atrial fibrillation
- B. Mobitz type II second-degree heart block
- C. Paroxysmal supraventricular tachycardia
- D. Sinus pause
- E. Ventricular tachycardia

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- ✗ A. Atrial fibrillation (4%)
- B. Mobitz type II second-degree heart block (11%)
- C. Paroxysmal supraventricular tachycardia (10%)
- ✓ D. Sinus pause (69%)
- E. Ventricular tachycardia (3%)

Incorrect
Correct answer
D

69%
Answered correctly

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Explanation

This patient with episodes of **presyncope** (eg, lightheadedness, feeling of impending loss of consciousness) that occur when he adjusts his shirt collar or necktie most likely has **carotid sinus hypersensitivity** (CSH). The condition results from **overly sensitive** carotid baroreceptors that stimulate an **excessive vasovagal response** to tactile stimulation. CSH is most commonly seen in **elderly men**; underlying carotid atherosclerotic disease and prior neck surgery (eg, thyroidectomy) are risk factors.

The baroreceptor response in CSH mimics the normal response to increased blood pressure but is more

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The baroreceptor response in CSH mimics the normal response to increased blood pressure but is more exaggerated; increased parasympathetic output leads to a prolonged **sinus pause** (eg, 3-5 seconds) and decreased sympathetic outflow causes marked **peripheral vasodilation**. The resulting delay in ventricular contraction and decreased systemic vascular resistance leads to a profound reduction in blood pressure and **transient loss of cerebral perfusion** that manifests as presyncope or syncope.

(Choices A, C, and E) Intermittent tachyarrhythmias, including atrial fibrillation with rapid ventricular response, paroxysmal supraventricular tachycardia (eg, atrioventricular nodal reentrant tachycardia), and ventricular tachycardia, can cause syncope due to loss of cardiac output and reduced cerebral perfusion (of the 3 tachyarrhythmias, ventricular tachycardia is most concerning). However, CSH is more likely in this patient with symptoms associated with tactile stimulation of the carotid sinus.

(Choice B) Advanced atrioventricular block (ie, complete heart block, Mobitz type II second-degree heart block) can cause presyncope or syncope but is also less likely than CSH in this patient with symptoms associated with tactile stimulation of the carotid sinus.

Educational objective:
Carotid sinus hypersensitivity is most commonly seen in elderly men and involves an exaggerated vasovagal response to tactile stimulation of carotid sinus baroreceptors (eg, adjusting a shirt collar or necktie). The resulting increase in parasympathetic output leads to a prolonged sinus pause that contributes to the excessive drop in blood pressure, leading to transient loss of cerebral perfusion that manifests as presyncope (eg, lightheadedness) or syncope.

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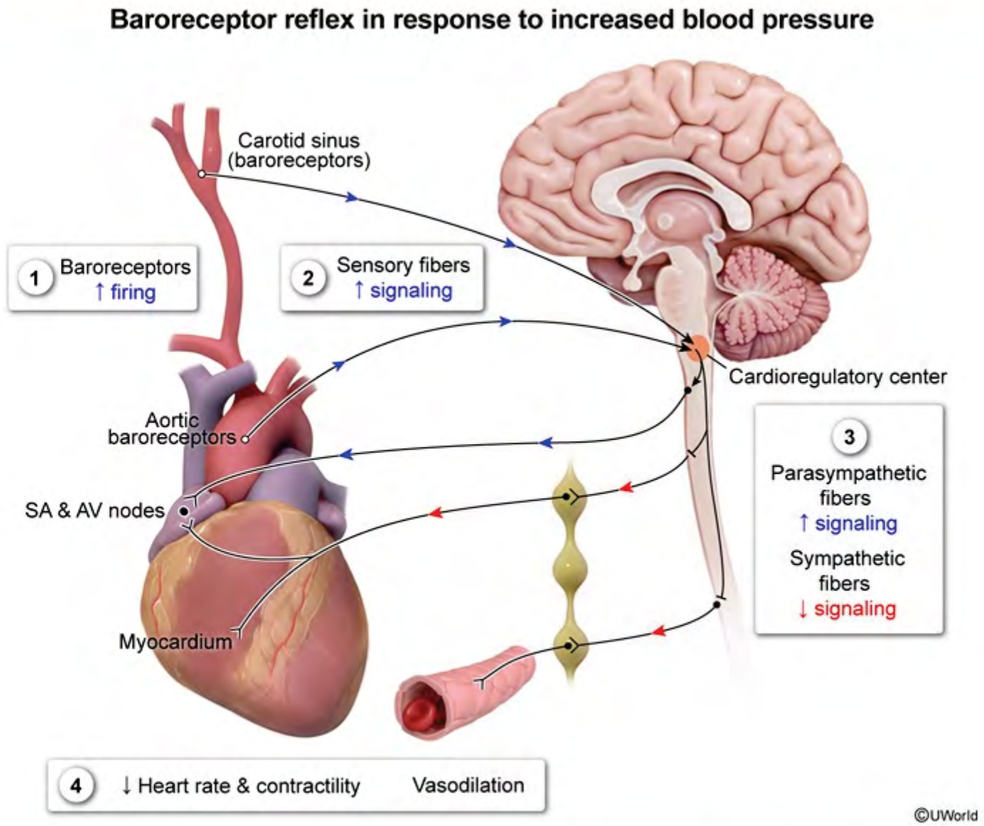

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A 42-year-old man comes to the emergency department due to an episode of syncope. He was standing in his kitchen this morning when he felt palpitations for about 3 seconds; the next thing he remembers is waking up on the floor. The patient has had intermittent palpitations for the last few weeks, sometimes with associated lightheadedness. He has no significant medical history other than chronic lower back pain that began following a fall from a ladder 3 years ago. The patient fractured several lumbar vertebrae in that fall and required a surgical lumbar fusion. His back pain was uncontrolled with conservative measures, and he has been treated with escalating doses of methadone for the past year. He does not smoke or drink alcohol. The patient has a remote history of intravenous heroin abuse but has not used it for 20 years. He has no family history of early-onset heart disease, blood clots, or sudden death. Blood pressure is 120/70 mm Hg when supine and 125/75 mm Hg when standing. Physical examination is unremarkable. Which of the following ECG findings is most likely in this patient?

- A. Atrioventricular block
- B. Deep Q waves
- C. QT interval prolongation
- D. Sinus pauses
- E. Ventricular preexcitation

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- ✗ A. Atrioventricular block (24%)
- B. Deep Q waves (2%)
- ✓ C. QT interval prolongation (35%)
- D. Sinus pauses (13%)
- E. Ventricular preexcitation (24%)

Incorrect
Correct answer
C

35%
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Explanation

Causes of QT-interval prolongation	
	• Medications

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Causes of QT-interval prolongation	
Acquired	• Medications ◦ Macrolides & fluoroquinolones ◦ Antiemetics (eg, ondansetron) ◦ Azoles (eg, fluconazole) ◦ Antipsychotics, TCAs & SSRIs ◦ Some opioids (eg, methadone, oxycodone) ◦ Class Ia antiarrhythmics (eg, quinidine) ◦ Class III antiarrhythmics (eg, dofetilide, sotalol) • Electrolyte abnormalities ◦ ↓ Magnesium, ↓ potassium, ↓ calcium
Congenital	• Romano-Ward syndrome (autosomal dominant) • Jervell & Lange-Nielsen syndrome (autosomal recessive)*
*Associated with sensorineural hearing loss. **SSRI** = selective serotonin reuptake inhibitor; **TCA** = tricyclic antidepressant.	

This patient most likely has **QT interval prolongation**, a common adverse effect of **methadone**. QT interval prolongation is the ECG manifestation of delayed ventricular repolarization, which occurs due to impairment of the voltage-gated potassium channels that control the delayed rectifier potassium current. Significant slowing of ventricular repolarization can trigger **torsade de pointes** (TdP), a form of polymorphic ventricular tachycardia named for a repetitive "twisting of the peaks" pattern. TdP is a serious arrhythmia that can cause palpitations, lightheadedness, **syncope**, and sudden cardiac death (SCD).

Many **medications**, including methadone, are known to **inhibit the delayed rectifier potassium current** to prolong the QT interval and predispose to TdP. Methadone undergoes slow and variable hepatic clearance, making consistent avoidance of toxic blood levels challenging; therefore, the use of high doses of methadone

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(Choice A) Advanced atrioventricular block (ie, Mobitz type II or complete atrioventricular block) can present with syncope. Common causes include infection (eg, Lyme disease), ischemia, and medications (eg, beta blockers, nondihydropyridine calcium channel blockers). Methadone is not associated with atrioventricular block.

(Choice B) Deep Q waves on ECG typically represent a previous myocardial infarction (MI). Patients with a history of MI are at risk for monomorphic ventricular tachycardia causing syncope or SCD, but a prior MI is unlikely in this relatively young patient without significant cardiovascular risk factors (eg, hypertension, smoking, family history).

(Choice D) Sinus pauses occur with sick sinus syndrome, which most commonly results from age-related degeneration of the conduction system. Affected patients are usually age >65 and may experience progressive fatigue and episodes of syncope.

(Choice E) Ventricular preexcitation is recognized by a slurred upslope of the QRS complex (delta wave) on ECG. Patients with this characteristic Wolff-Parkinson-White (WPW) pattern may develop paroxysmal supraventricular tachycardia (ie, WPW syndrome), which can present with syncope or SCD. However, the onset of WPW syndrome is usually age <40, and this patient's methadone use makes a prolonged QT interval more likely.

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Educational objective:
Methadone slows the delayed rectifier potassium current responsible for ventricular repolarization, and high doses are associated with QT interval prolongation. QT interval prolongation predisposes to the development of torsade de pointes, a serious ventricular arrhythmia that can cause syncope and sudden cardiac death.

References

- Methadone, QTc prolongation and torsades de pointes: current concepts, management, and a hidden twist in

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Congenital	• Romano-Ward syndrome (autosomal dominant) • Jervell & Lange-Nielsen syndrome (autosomal recessive)*

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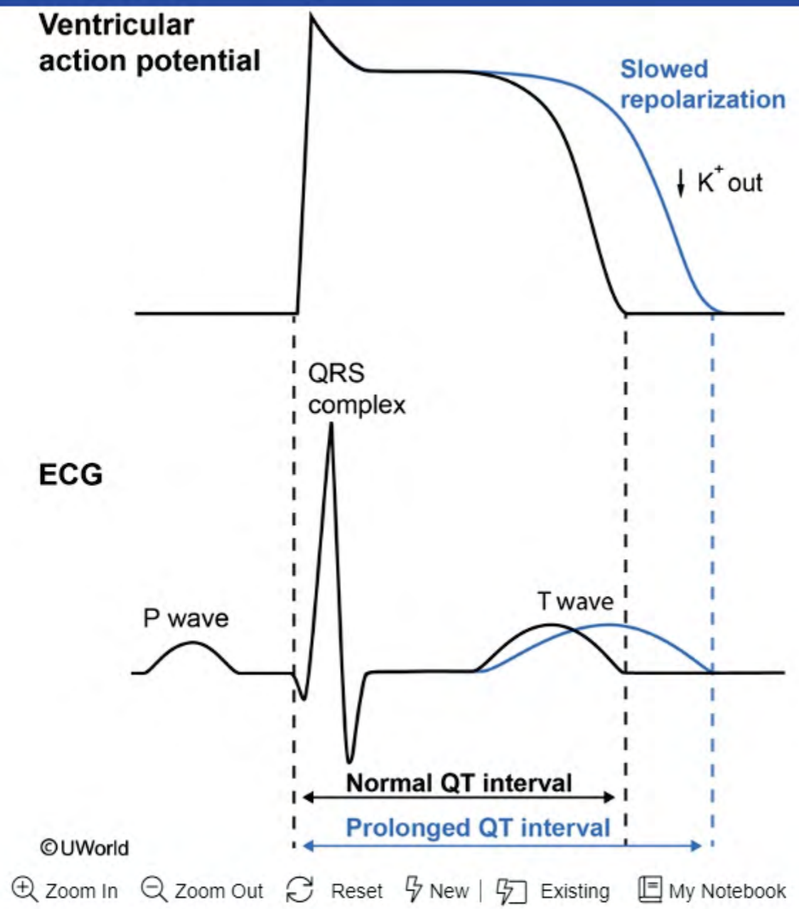

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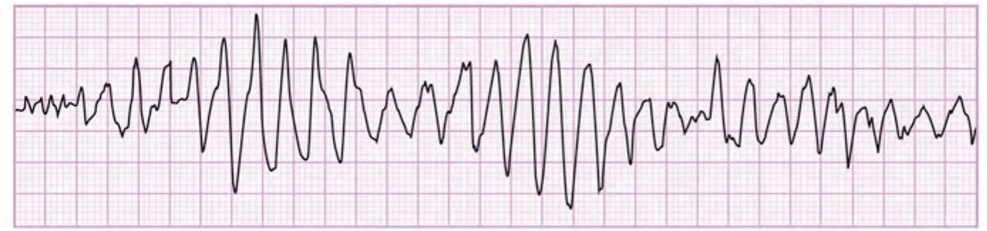

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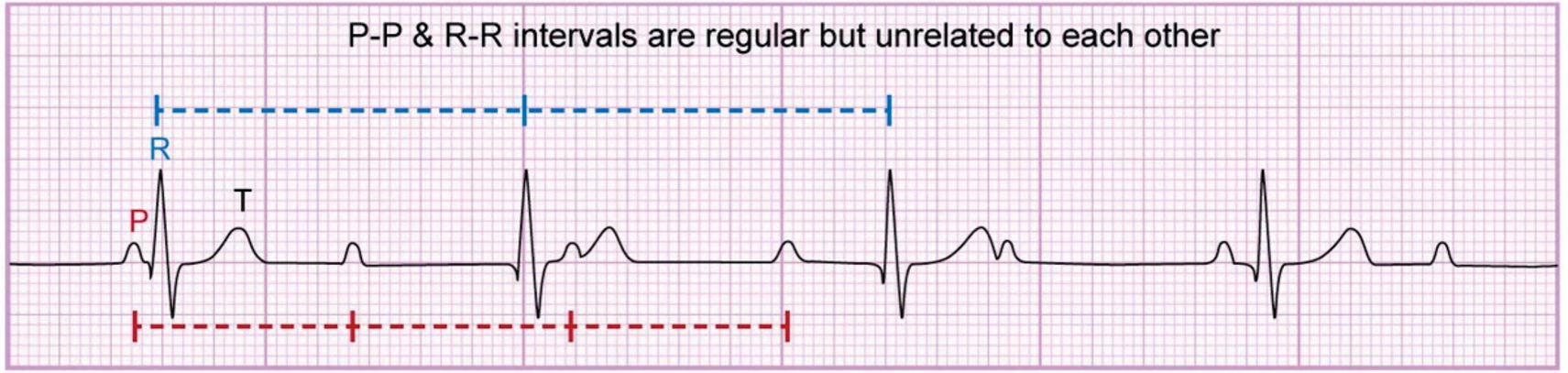
Complete heart block



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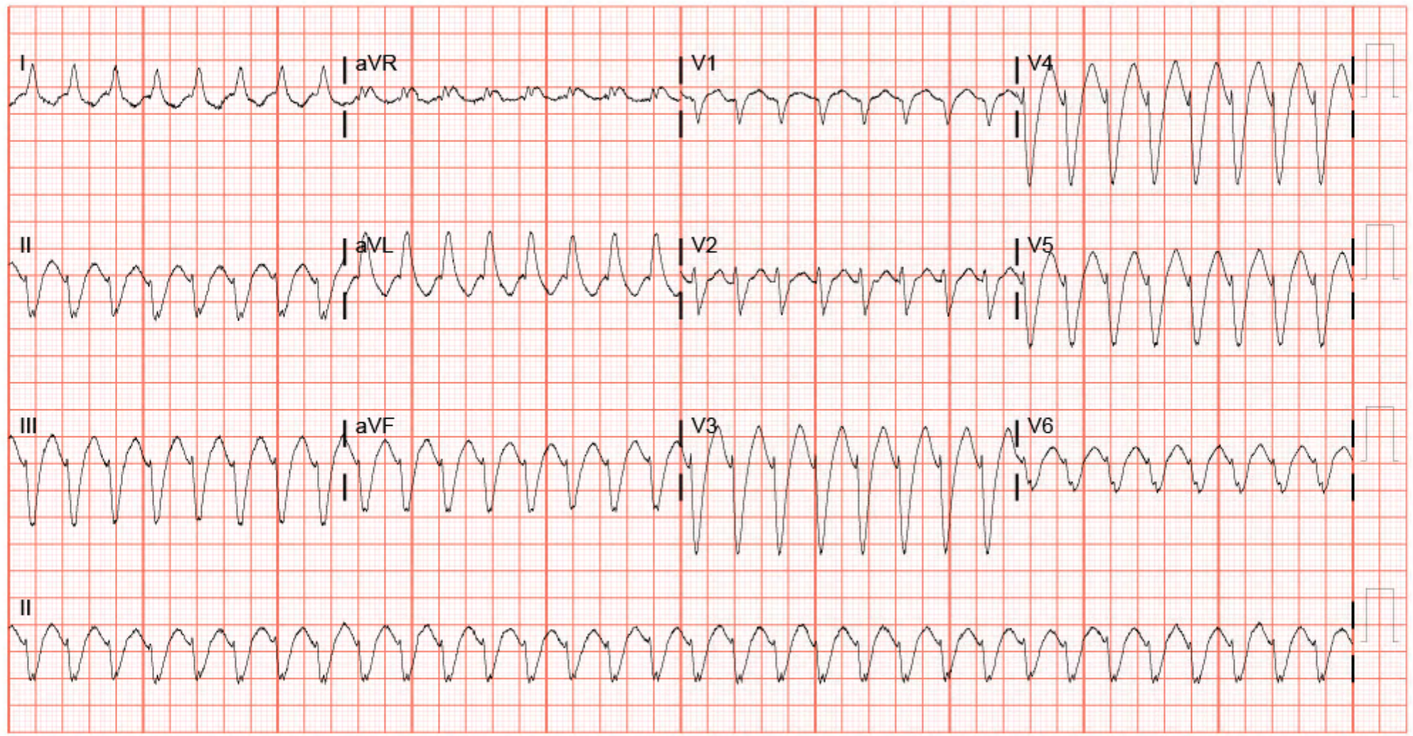

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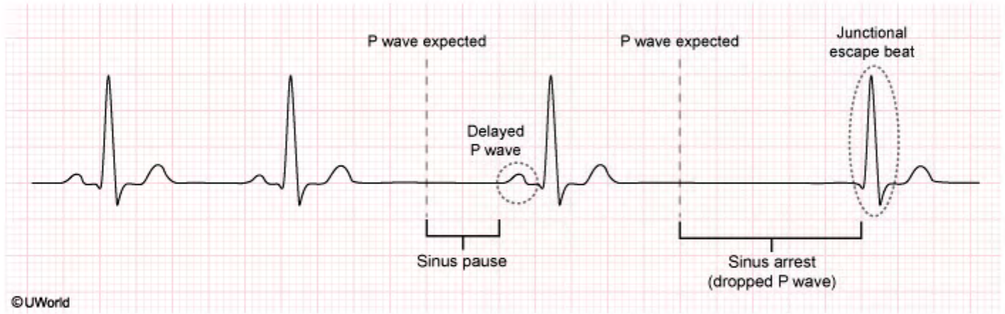

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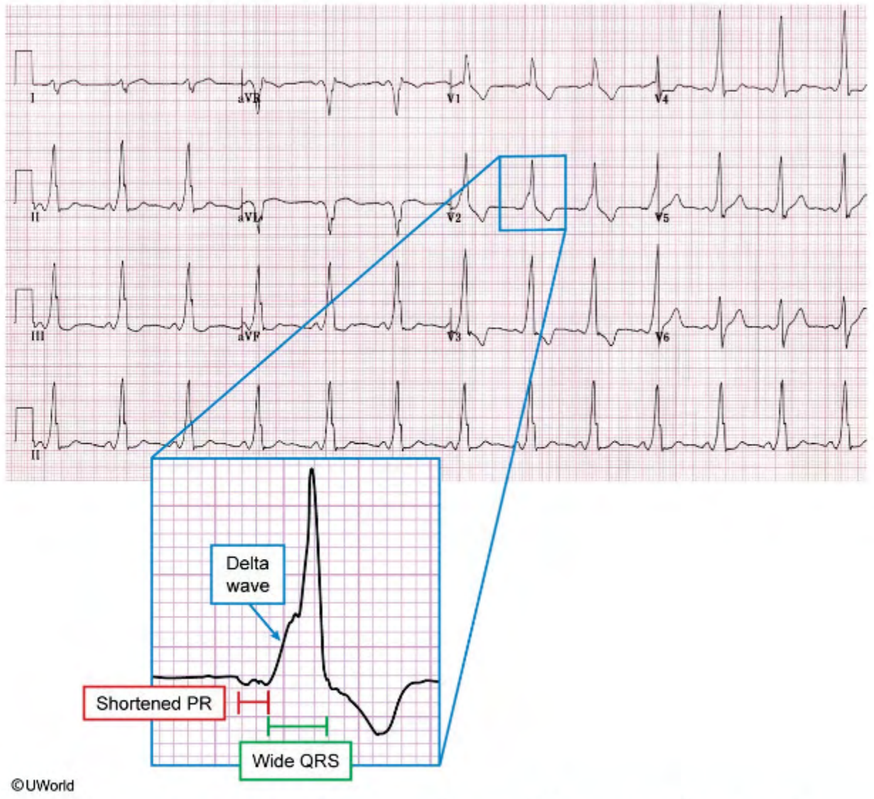

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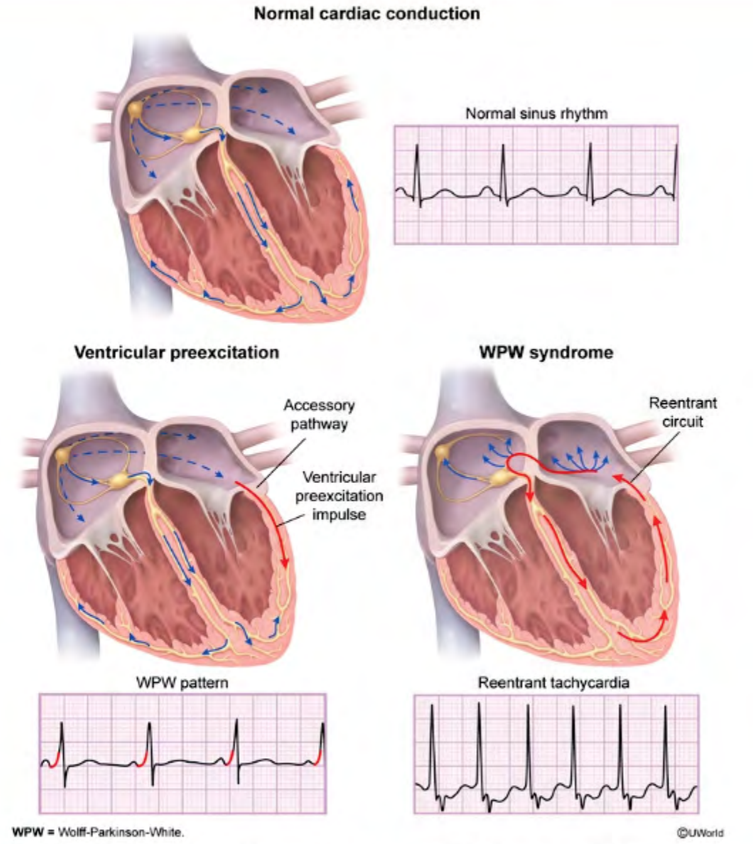

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A 34-year-old Asian female is hospitalized with progressive exertional dyspnea, lower extremity edema and cough. She also describes frequent nocturnal episodes of breathlessness and recent hoarseness. She does not use tobacco, alcohol or drugs. Auscultation reveals loud first and second heart sounds and a mid-diastolic rumble best heard at the cardiac apex. This patient's hoarseness is most likely caused by:

- A. Laryngeal edema
- B. Impaired arterial supply
- C. Nerve impingement
- D. Epithelial sloughing
- E. Vocal cord polyps

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- ✗ A. Laryngeal edema (11%)
- B. Impaired arterial supply (6%)
- ✓ C. Nerve impingement (78%)
- D. Epithelial sloughing (1%)
- E. Vocal cord polyps (1%)

Incorrect
Correct answer
C

78%
Answered correctly

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Explanation

In rare instances, the left recurrent laryngeal nerve may be compressed to the point of neurapraxia (failure of nerve conduction due to blunt injury) by enlargement of the left atrium and/or other structures in the vicinity of its course as it loops behind the ligamentum arteriosum, underneath and around the aortic arch, and back up alongside the trachea to the larynx. Mitral stenosis can cause left atrial dilatation sufficient to impinge on the left recurrent laryngeal nerve (Ortner syndrome).

The recurrent laryngeal nerves innervate all of the intrinsic muscles of the larynx except the cricothyroid muscle. Paresis of vocal cord muscles innervated by left recurrent laryngeal nerve can cause hoarseness.

(Choice A) Laryngeal edema can cause hoarseness but is commonly due to direct laryngeal inflammation (e.g. by

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(Choice A) Laryngeal edema can cause hoarseness but is commonly due to direct laryngeal inflammation (e.g. by an upper respiratory infection).

(Choice B) Vascular disease or injury resulting in ischemia of the vocal cords, the recurrent laryngeal nerves, and/or the vagal motor nuclei in the brain stem could result in laryngeal neuromuscular dysfunction, producing hoarseness. However, the patient's history and presentation make MS a more likely culprit.

(Choice D) Hoarseness is predominantly due to vocal cord disease (including mucosal inflammation or edema) or vocal cord malfunction. Epithelial sloughing could be associated with laryngeal mucosal disease and might be seen in asthma or airway exposure to noxious external gases or gastric acid. However, the other components of the patient's history and physical make severe MS more likely as an underlying pathogenic factor.

(Choice E) Vocal cord epithelial or mucosal polyps can also cause hoarseness, but are less likely etiologies here, given the patient's history.

Educational Objective:
Left atrial enlargement can sometimes cause left recurrent laryngeal nerve impingement. Neurapraxia resulting in left vocal cord paresis and hoarseness may result.

Pathology	Cardiovascular System	Mitral stenosis
Subject	System	Topic

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A 65-year-old man reports multiple episodes of lightheadedness while buttoning a tight shirt collar. During 2 episodes, he passed out briefly but sustained no injuries. His blood pressure was 70/40 mm Hg and pulse was 45/min during one of the episodes. Past medical history is significant for hypertension and diet-controlled diabetes mellitus. The patient is a lifetime nonsmoker and drinks alcohol on social occasions. On physical examination, his blood pressure is 125/72 mm Hg and pulse is 76/min without orthostatic changes. Stimulation of afferent sensory fibers in which of the following nerves is most likely responsible for this patient's symptoms?

- A. Accessory
- B. Glossopharyngeal
- C. Hypoglossal
- D. Trigeminal
- E. Vagus

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- ✗ A. Accessory (0%)
- ✓ B. Glossopharyngeal (52%)
- C. Hypoglossal (1%)
- D. Trigeminal (0%)
- E. Vagus (45%)

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Correct answer
B

52%
Answered correctly

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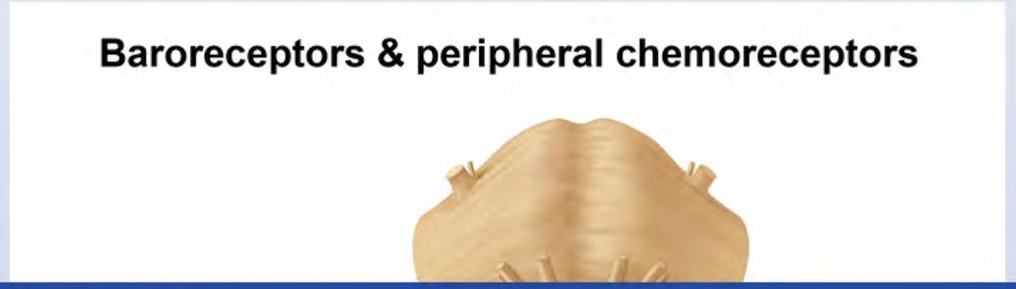
Explanation



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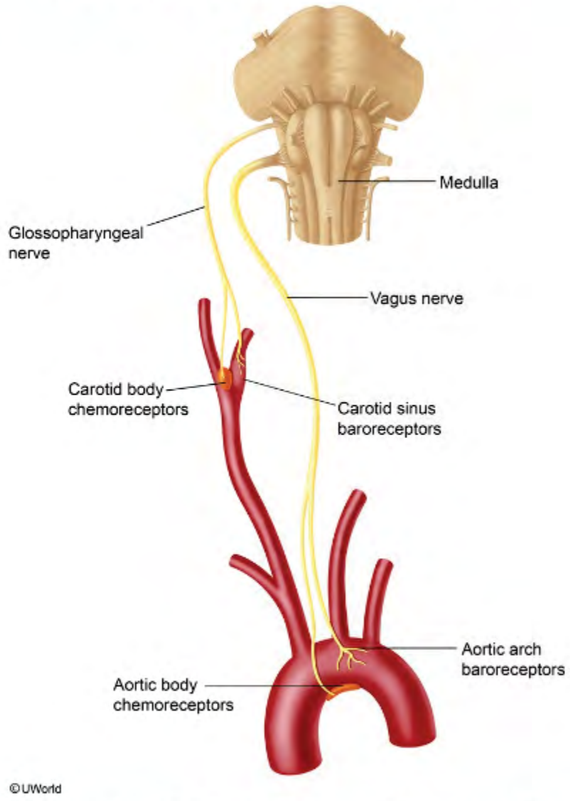
Baroreceptors & peripheral chemoreceptors



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The patient's history is suggestive of **carotid sinus hypersensitivity**, triggered by pressure on the carotid sinus by a tight shirt collar. The carotid sinus baroreceptors are important in blood pressure control and use arterial wall stretch as an indicator of systemic blood pressure.

The carotid sinus is a dilation of the internal carotid artery located just above the bifurcation of the common carotid artery. The carotid sinus reflex has an **afferent limb** that arises from the baroreceptors in the carotid sinus and travels to the medullary centers via the Hering nerve, a branch of the **glossopharyngeal nerve** (CN IX). The **efferent limb** of the carotid sinus carries parasympathetic impulses via the **vagus nerve** (CN X). Carotid sinus pressure or massage stimulates the baroreceptors and increases the firing rate from the carotid sinus, leading to an increase in parasympathetic output and withdrawal of sympathetic output to the heart and peripheral vasculature. The result is decreased blood pressure (via peripheral vasodilation) and decreased cardiac output (decreased contractility/stroke volume and heart rate). In sensitive individuals, this response can cause severe bradycardia, hypotension, and sometimes syncope.

(Choices A, C, and D) The accessory (CN XI), hypoglossal (CN XII), and trigeminal (CN V) nerves do not transmit baroreceptor signals. The accessory nerve controls the sternocleidomastoid and trapezius muscles, the hypoglossal nerve primarily controls the muscles of the tongue, and the trigeminal nerve mediates facial sensation and also controls the muscles of mastication (chewing).

(Choice E) The vagus nerve (CN X) acts as the afferent limb for the nerve fibers originating from the aortic arch baroreceptors and as an efferent limb for the carotid sinus reflex. However, a tight collar would stimulate carotid sinus (not aortic arch) baroreceptor afferents carried by the glossopharyngeal nerve.

Educational objective:
The carotid sinus is a dilation of the internal carotid artery located just above the bifurcation of the common carotid artery. The carotid sinus reflex has an afferent limb that arises from the baroreceptors in the carotid sinus and travels to the vagal nucleus and medullary centers via the glossopharyngeal nerve (CN IX); the efferent limb

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Physiology	Cardiovascular System	Carotid sinus hypersensitivity
Subject	System	Topic

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A 67-year-old man is admitted to the hospital due to acute right-sided weakness. Medical history includes hypertension, hyperlipidemia, and coronary artery disease, for which the patient underwent placement of 2 stents in the left anterior descending artery. He also has intermittent claudication that limits exercise capacity. The patient's antihypertensive regimen includes chlorthalidone, amlodipine, and lisinopril, which he takes as prescribed. He is an ex-smoker with a 35-pack-year history. Blood pressure is 180/110 mm Hg and pulse is 80/min. Serum creatinine is 1.6 mg/dL. CT scan of the head reveals hemorrhage of the left basal ganglia, and renal ultrasound shows that the right kidney is 7 cm long and the left kidney is 11 cm long. Which of the following mechanisms most likely explains the renal morphology in this patient?

- A. Congenital hypoplasia
- B. Denervation
- C. Lack of trophic hormones
- D. Oxygen and nutrient deprivation
- E. Senile changes

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- ✗ A. Congenital hypoplasia (4%)
- B. Denervation (2%)
- C. Lack of trophic hormones (2%)
- ✓ D. Oxygen and nutrient deprivation (86%)
- E. Senile changes (3%)

Incorrect
Correct answer
D

86%
Answered correctly

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Explanation

Renal artery stenosis	
Etiology	• Atherosclerotic narrowing of the renal artery (>90% of cases) • Fibromuscular dysplasia (younger women)

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Explanation

Renal artery stenosis	
Etiology	• Atherosclerotic narrowing of the renal artery (>90% of cases) • Fibromuscular dysplasia (younger women)
Risk factors	• Other atherosclerotic disease (eg, CAD, carotid stenosis) • Smoking, diabetes, hypertension
Presentation	• Refractory hypertension • Recurrent flash pulmonary edema • Abdominal bruits • Acute kidney injury after initiation of ACE inhibitor
Pathology	• Gross: Shrunken, atrophic kidney • Microscopic: Crowded glomeruli, tubular atrophy, interstitial fibrosis, focal inflammatory infiltrates
CAD = coronary artery disease.	

This patient has an intraparenchymal hemorrhage in the setting of severe, **refractory hypertension**. He has coronary artery disease and peripheral vascular disease (claudication) with multiple risk factors (eg, smoking, hyperlipidemia) for diffuse atherosclerosis. The elevated serum creatinine and the renal ultrasound demonstrating a small (atrophic) right kidney suggest that the patient's refractory hypertension is most likely due to **unilateral renal artery stenosis**.

The most common cause of renal artery stenosis is obstruction by **atheromatous plaque** at the origin of the renal artery, leading to unilateral **renal ischemia**. The ischemic kidney secretes high levels of renin, which activates the renin-angiotensin-aldosterone system and results in systemic hypertension that is often difficult to control despite aggressive antihypertensive therapy. Eventually, the stenosis may also cause **renal atrophy** due to **oxygen and**

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(Choice A) Congenital renal hypoplasia is a failure of the kidneys to develop to normal size. Involvement is unilateral in most cases. However, this condition does not cause secondary hypertension.

(Choice B) Unilateral denervation of a kidney, either during or after development, is not known to have significant trophic effects (affecting kidney growth or morphology).

(Choice C) The role of trophic hormones in renal development remains unclear. Still, a deficiency of such hormones would be expected to produce bilateral renal dysplasia.

(Choice E) Aging is associated with loss of nephron mass as a result of variable degrees of arteriolosclerosis, focal glomerulosclerosis, and tubulointerstitial fibrosis. These processes produce symmetric, not unilateral, decrements in kidney size and functional capacity.

Educational objective:
Renal artery stenosis is most often due to atherosclerosis. It can cause severe, refractory hypertension due to activation of the renin-angiotensin-aldosterone system. Over time, renal atrophy may occur due to chronic oxygen and nutrient deprivation. Histologic examination may show crowded glomeruli, tubulointerstitial atrophy and

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Renal artery stenosis is most often due to atherosclerosis. It can cause severe, refractory hypertension due to activation of the renin-angiotensin-aldosterone system. Over time, renal atrophy may occur due to chronic oxygen and nutrient deprivation. Histologic examination may show crowded glomeruli, tubulointerstitial atrophy and fibrosis, and focal inflammatory infiltrates.

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Renal artery stenosis

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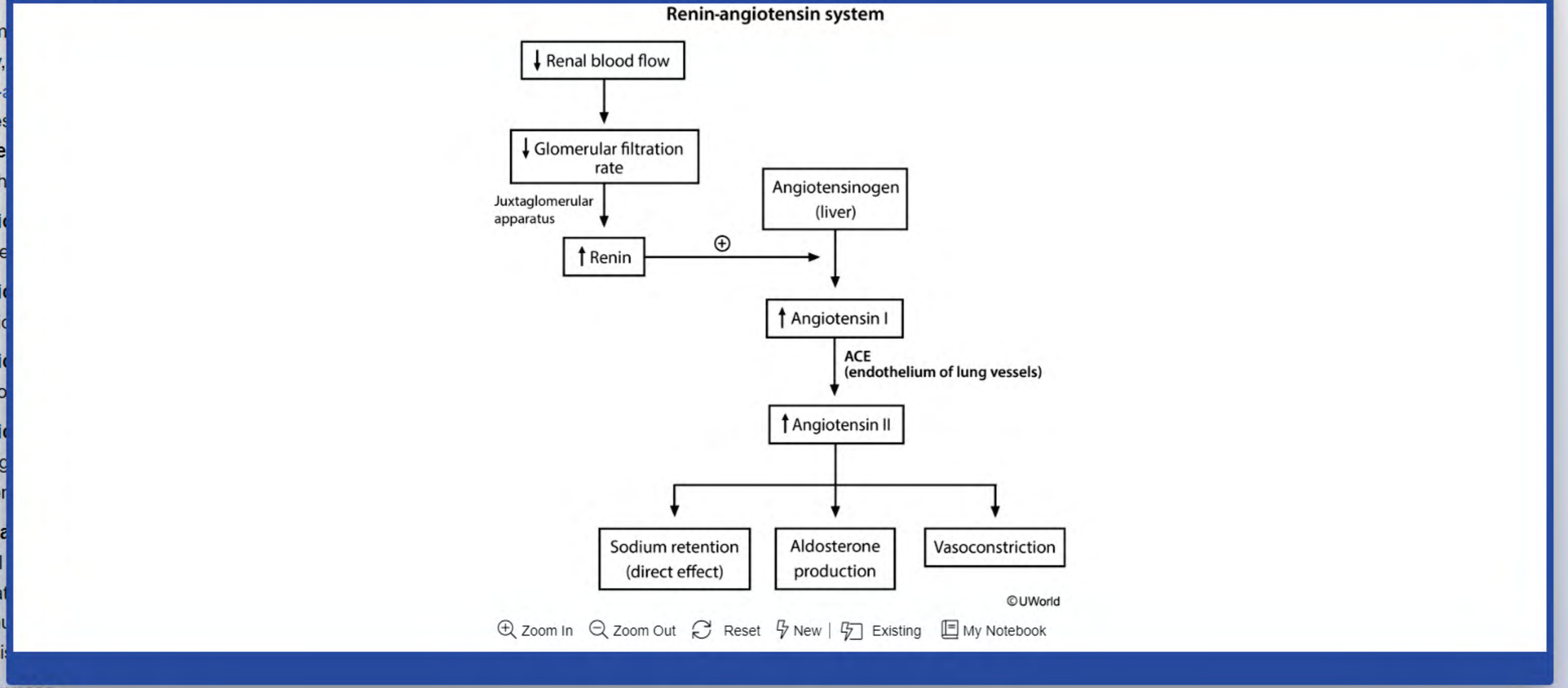

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A 34-year-old woman is evaluated in the emergency department due to 1-day of chest and left shoulder pain. The patient describes the pain as constant and worse in certain positions. A month ago, she was treated for a skin rash and joint pain. Her medical history is otherwise unremarkable. She is a lifetime nonsmoker and does not use alcohol or illicit drugs. Her father has hypertension and had a stroke at age 64. Temperature is 37.5 C (99.5 F), blood pressure is 122/70 mm Hg, and pulse is 97/min and regular. A triphasic, scratchy sound is heard over the left sternal border with the patient sitting up. The lungs are clear on auscultation. ECG shows sinus rhythm with ST-segment elevation in all leads except avR and V1. Which of the following is the most likely diagnosis?

- A. Eosinophilic myocarditis
- B. Fibrinous pericarditis
- C. Hemorrhagic pericarditis
- D. Nonbacterial endocarditis
- E. Purulent pericarditis

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- ✗ A. Eosinophilic myocarditis (7%)
- ✓ B. Fibrinous pericarditis (67%)
- C. Hemorrhagic pericarditis (2%)
- D. Nonbacterial endocarditis (11%)
- E. Purulent pericarditis (11%)

Incorrect
Correct answer
B

67%
Answered correctly

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Explanation

Acute pericarditis	
Etiology	• Viral or idiopathic • Autoimmune disease (eg, SLE) • Uremia (acute or chronic renal failure) • Post myocardial infarction

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Acute pericarditis	

Etiology	• Viral or idiopathic
	• Autoimmune disease (eg, SLE)
	• Uremia (acute or chronic renal failure)
	• Post myocardial infarction
	  ◦ Early: peri-infarction pericarditis
	  ◦ Late: Dressler syndrome

Clinical features & diagnosis	• Pleuritic chest pain (↓ when sitting up) ± fever • Pericardial friction rub • ECG: diffuse ST-segment elevation • Pericardial effusion on echocardiogram
SLE = systemic lupus erythematosus.	

This patient with pleuritic-type chest pain radiating to the shoulder and a recent history of skin rash and joint pain likely has acute **fibrinous pericarditis** due to **systemic lupus erythematosus**. Fibrinous pericarditis is the most common type of pericarditis and consists of pericardial inflammation with serous fluid and **fibrin-containing exudate** in the pericardial space.

Patients typically have pleuritic (sharp, worse with breathing or movement) anterior chest pain that can radiate to the left shoulder or posteriorly to the bilateral scapulae. Fibrin deposition causes roughening of the visceral and parietal pericardium, often heard as a **triphasic friction rub** (occurring during atrial systole, ventricular systole, and early ventricular diastole) on cardiac auscultation; however, the rub can be absent if significant pericardial effusion is present. ECG typically shows **diffuse ST elevation** due to inflammation of the ventricular myocardium.

Other causes of fibrinous pericarditis include viral infection, myocardial infarction, uremia, and other rheumatologic disease (eg, rheumatoid arthritis). If acute fibrinous pericarditis goes without treatment, chronic constrictive

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Other causes of fibrinous pericarditis include viral infection, myocardial infarction, uremia, and other rheumatologic disease (eg, rheumatoid arthritis). If acute fibrinous pericarditis goes without treatment, chronic constrictive pericarditis can develop in some patients.

(Choice A) Eosinophilic myocarditis can occur with malignancy, parasitic infection, or drug hypersensitivity and often presents with symptoms of heart failure. A friction rub on cardiac auscultation is not expected.

(Choices C and E) Hemorrhagic and purulent pericarditis are both less common than fibrinous pericarditis. Hemorrhagic pericarditis consists of blood mixed with fibrinous exudate; it most commonly occurs in the setting of malignancy but may also occur due to tuberculosis, following cardiac surgery, or in patients with underlying coagulopathy. Purulent pericarditis results from active bacterial infection (eg, *Staphylococcus aureus*, *Streptococcus* species) in the pericardial space, which may occur due to hematogenous spread or direct extension of pneumonia or endocarditis.

(Choice D) Nonbacterial endocarditis can occur in systemic lupus erythematosus and involves deposition of sterile, platelet-rich thrombi on the mitral or aortic valve. It would not explain a friction rub or diffuse ST elevation on ECG.

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Educational objective:
Fibrinous pericarditis is the most common type of pericarditis and is characterized by pericardial inflammation with a serous, fibrin-containing exudate in the pericardial space. Pleuritic chest pain and a triphasic friction rub are frequently seen. Common causes include viral infection, myocardial infarction, uremia, and rheumatologic disease (eg, systemic lupus erythematosus, rheumatoid arthritis).

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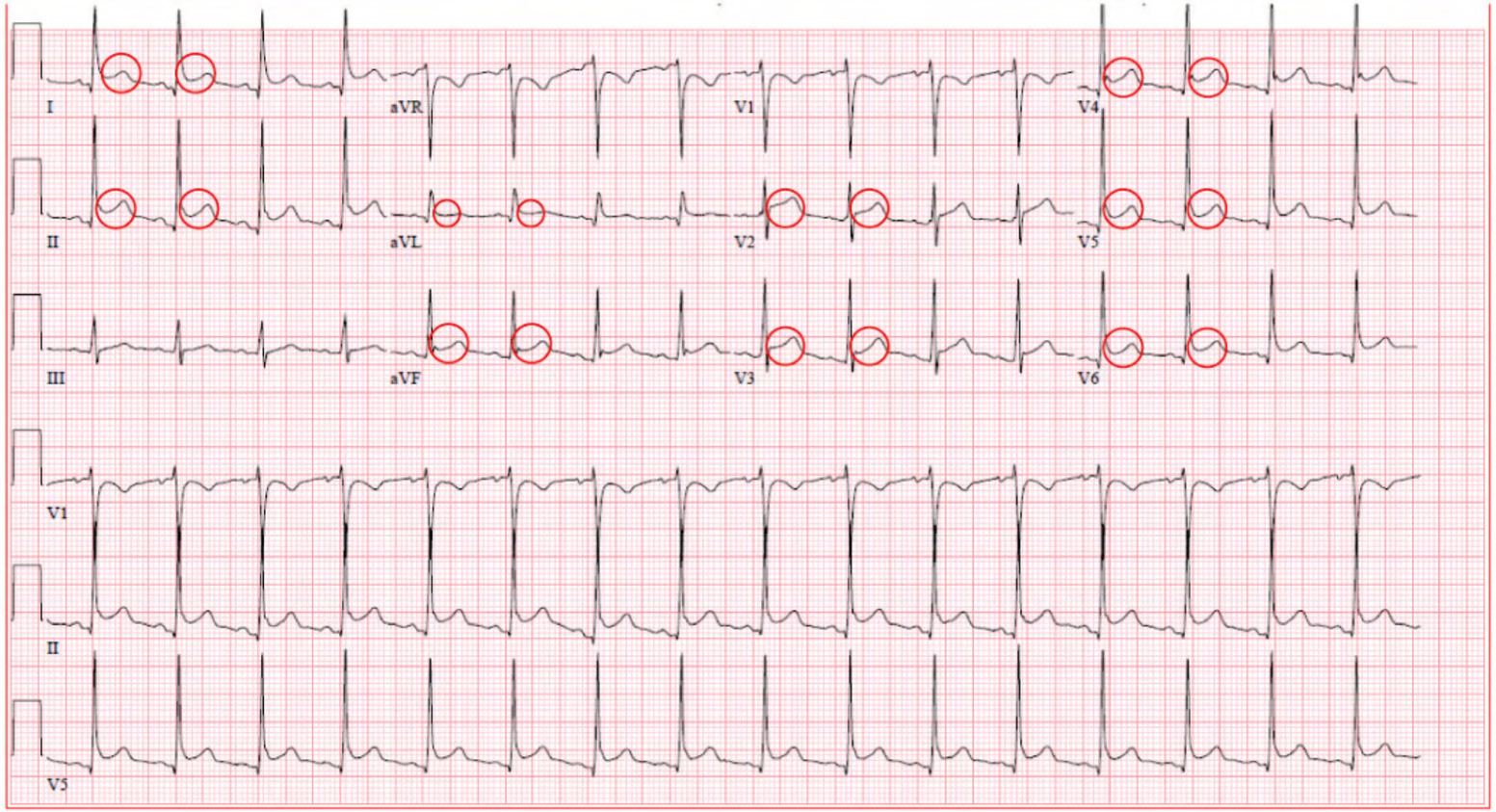

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A 39-year-old man comes to the emergency department with anterior chest pain. He had felt well until the pain developed 4 days ago. The patient says the pain is sharp and makes it difficult to take a deep breath. Since yesterday, he has also felt out of breath. Medical history is unremarkable. The patient's father died of a heart attack at age 52, and his mother suffers from rheumatoid arthritis. He does not use alcohol or tobacco. Temperature is 37.4 C (99.3 F), blood pressure is 112/65 mm Hg, and pulse is 103/min and regular. Bedside ultrasound examination demonstrates a moderate pericardial effusion. Which of the following is the most likely cause of this patient's current condition?

- A. Autoimmune disease
- B. Coronary artery disease
- C. Gram-positive cocci
- D. Malignancy
- E. Viral infection

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- ✗ A. Autoimmune disease (20%)
- B. Coronary artery disease (6%)
- C. Gram-positive cocci (4%)
- D. Malignancy (2%)
- ✓ E. Viral infection (66%)

Incorrect
Correct answer
E

66%
Answered correctly

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Explanation

Acute pericarditis	
Etiology	• Viral or idiopathic • Autoimmune disease (eg, SLE) • Uremia (acute or chronic renal failure) • Post myocardial infarction

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Acute pericarditis	

Etiology	• Viral or idiopathic
	• Autoimmune disease (eg, SLE)
	• Uremia (acute or chronic renal failure)
	• Post myocardial infarction
	  ◦ Early: peri-infarction pericarditis
	  ◦ Late: Dressler syndrome

Clinical features & diagnosis	• Pleuritic chest pain (↓ when sitting up) ± fever • Pericardial friction rub • ECG: diffuse ST-segment elevation • Pericardial effusion on echocardiogram
SLE = systemic lupus erythematosus.	

This patient most likely has **acute viral pericarditis**; viral infection is the most common cause of pericarditis and many viruses (eg, adenovirus, coxsackievirus, echovirus, influenza virus) have been implicated. Because the viral infection often cannot be confirmed, presumed viral pericarditis is sometimes referred to as **idiopathic**. Pericarditis typically presents with substernal **pleuritic chest pain** that may radiate to the bilateral scapulae posteriorly. The pain is typically worse when lying flat and improves with sitting up and leaning forward. Fever is common but often not present.

Cardiac auscultation in acute viral pericarditis classically reveals a triphasic **pericardial friction rub** (occurring during atrial systole, ventricular systole, and early ventricular diastole); however, the rub may be absent, especially in the presence of significant **pericardial effusion**. Mild to moderate-sized pericardial effusion is common and can rarely lead to cardiac tamponade. ECG characteristically demonstrates diffuse ST elevation caused by inflammation of the ventricular myocardium.

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in the presence of significant **pericardial effusion**. Mild to moderate-sized pericardial effusion is common and can rarely lead to cardiac tamponade. ECG characteristically demonstrates diffuse ST elevation caused by inflammation of the ventricular myocardium.

(Choice A) Autoimmune disease (eg, systemic lupus erythematosus, rheumatoid arthritis) is a potential cause of pericarditis, but it is less common than viral pericarditis and is less likely in the absence of a personal history of autoimmune disease (pericarditis is only rarely the presenting manifestation of autoimmune disease).

(Choice B) Myocardial infarction due to coronary artery disease (CAD) can lead to peri-infarction pericarditis (a localized reaction to transmural myocardial necrosis) within 2-4 days or to Dressler syndrome (an autoimmune response to infarction-induced antigens) within weeks to months. Despite a family history of CAD, myocardial infarction is relatively unlikely in this man age <40.

(Choice C) Gram-positive cocci (eg, *Staphylococcus aureus*, *Streptococcus pneumoniae*) in rare cases can cause pericarditis (purulent pericarditis) via direct extension of pneumonia or hematogenous bacterial spread. Affected patients are typically quite ill and present with high fever and sepsis.

(Choice D) Malignant pericarditis can occur in the setting of metastatic cancer (eg, lung cancer, breast cancer); however, it is less likely to present with pain and inflammation and more likely to present with symptoms of fluid accumulation (ie, dyspnea due to subacute cardiac tamponade). Malignant pericarditis is also relatively uncommon compared to viral pericarditis and less likely in this relatively young man.

Educational objective:
Viral infection is the most common cause of acute pericarditis. It causes a fibrinous or serofibrinous pericarditis that is often characterized by pleuritic chest pain, a friction rub on cardiac auscultation, diffuse ST elevation on ECG, and mild to moderate-sized pericardial effusion.

Pathology	Cardiovascular System	Acute pericarditis
Subject	System	Topic

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Clinical features	• Pleuritic chest pain (↓ when sitting up) ± fever • Pericardial friction rub

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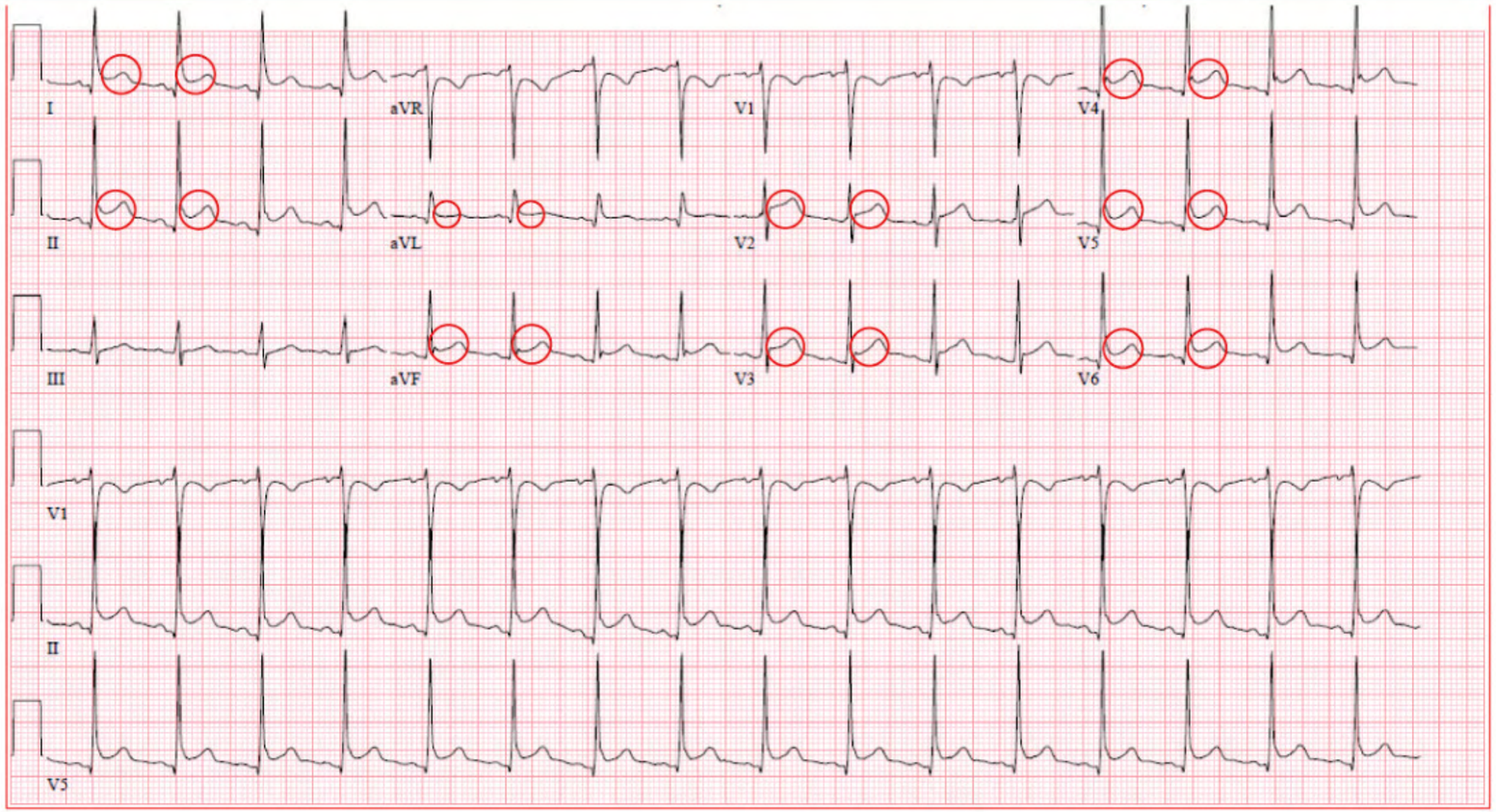

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pericarditis (purulent pericarditis) via direct extension of pneumonia or hematogenous bacterial spread. Affected

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A 22-year-old man comes to the office for an exercise evaluation. He is an avid long-distance runner. One of his team members was disqualified recently due to health issues, which made him worried about his own health. He has no medical history and takes no medications or recreational drugs. Blood pressure is 125/75 mm Hg and pulse is 55/min. Examination shows no abnormalities. Transthoracic echocardiogram results are as follows:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	small
LV septal thickness	increased
LV posterior wall thickness	normal
LV mass	increased
LV ejection fraction	70%

The right ventricle appears normal, and there are no valvular lesions. Which of the following pathologic findings are most likely to be present within this patient's heart?

- A. Accumulation of amorphous substance in interstitial space
- B. Distorted myocardial structure and myofibril disarray
- C. Increase of individual myocardial fiber length more than width
- D. Myocardial atrophy and replacement with fibrous fatty tissue
- E. Subendocardial replacement fibrosis

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Left ventricular (LV) cavity size	small
LV septal thickness	increased
LV posterior wall thickness	normal
LV mass	increased
LV ejection fraction	70%

The right ventricle appears normal, and there are no valvular lesions. Which of the following pathologic findings are most likely to be present within this patient's heart?

- ✗ A. Accumulation of amorphous substance in interstitial space (3%)
- ✓ B. Distorted myocardial structure and myofibril disarray (66%)
- C. Increase of individual myocardial fiber length more than width (26%)
- D. Myocardial atrophy and replacement with fibrous fatty tissue (0%)
- E. Subendocardial replacement fibrosis (2%)

Incorrect
Correct answer
B

66%
Answered correctly

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Explanation

Expected echocardiographic findings				
	LV mass	**LV cavity size**	**LV ejection fraction**	**LV diastolic function**
Dilated cardiomyopathy	Increased	Increased	Decreased	Normal
Hypertrophic cardiomyopathy	Increased	Decreased	Preserved	Impaired
Restrictive cardiomyopathy	Normal	Normal	Preserved	Impaired
Athlete's heart	Increased	Normal or increased	Preserved	Normal
LV = left ventricular.				

This patient most likely has **hypertrophic cardiomyopathy** (HCM), an autosomal dominant inherited condition resulting from mutations in the cardiac sarcomere and leading to **distorted myocardial structure** and **myofibril disarray**. Recognition of HCM is important because it is one of the most common causes of sudden cardiac death (SCD) in young adults. Although patients may experience exertional dyspnea, chest pain, lightheadedness, or syncope, they are often asymptomatic.

Echocardiographic findings consistent with HCM include:

- Overall increase in left ventricular (LV) mass
- **Reduced LV cavity size** (with impaired diastolic function)
- **Asymmetric** increase in **LV wall thickness** predominantly affecting the **septum**

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Echocardiographic findings consistent with HCM include:

- Overall increase in left ventricular (LV) mass
- **Reduced LV cavity size** (with impaired diastolic function)
- **Asymmetric** increase in **LV wall thickness** predominantly affecting the **septum**
- Normal or **increased LV ejection fraction**
- Left atrial enlargement (secondary to increased LV end-diastolic pressure)

Another characteristic of HCM is a poorly developed coronary capillary network in hypertrophied regions with evidence of chronic ischemia (ie, fibrosis, scarring).

(Choice A) Accumulation of amorphous substance in the cardiac interstitial space occurs with cardiac amyloidosis, which usually affects older patients (eg, age >60). In early disease, a restrictive pattern develops, with stiff and thickened LV walls leading to diastolic dysfunction. LV cavity size remains normal.

(Choice C) An increase in individual myocardial fiber length more than width (ie, eccentric hypertrophy) occurs with athlete's heart, constituting physiologic adaptation to high-level endurance training. There is enlarged LV cavity size and a small degree of uniformly increased LV wall thickness. The changes of athlete's heart must be differentiated from those associated with pathologic disease; this patient's reduced LV cavity size and localized septal wall thickening are red flag features suggesting underlying pathology due to HCM.

(Choice D) Myocardial atrophy and replacement with fibrous fatty tissue occur with arrhythmogenic right ventricular cardiomyopathy, an autosomal dominant condition involving mutations affecting cardiac desmosomes. The disease primarily affects the right ventricle, causing ventricular arrythmias and sometimes right ventricular

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(Choice D) Myocardial atrophy and replacement with fibrous fatty tissue occur with arrhythmogenic right ventricular cardiomyopathy, an autosomal dominant condition involving mutations affecting cardiac desmosomes. The disease primarily affects the right ventricle, causing ventricular arrythmias and sometimes right ventricular failure. When the left ventricle is involved, it commonly demonstrates eccentric enlargement with increased LV cavity size.

(Choice E) Subendocardial replacement fibrosis can occur following viral myocarditis and can lead to dilated cardiomyopathy. LV contractile function is disrupted, causing increased LV cavity size and reduced LV ejection fraction.

Educational objective:
Patients with hypertrophic cardiomyopathy (HCM) may be asymptomatic. Echocardiographic findings consistent with HCM include an overall increase in left ventricular (LV) mass, reduced LV cavity size with impaired diastolic function, LV hypertrophy predominantly affecting the septum, and normal or increased LV ejection fraction.

Pathophysiology	Cardiovascular System	Hypertrophic cardiomyopathy
Subject	System	Topic

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evidence of chronic ischemia (ie, fibrosis, scarring).

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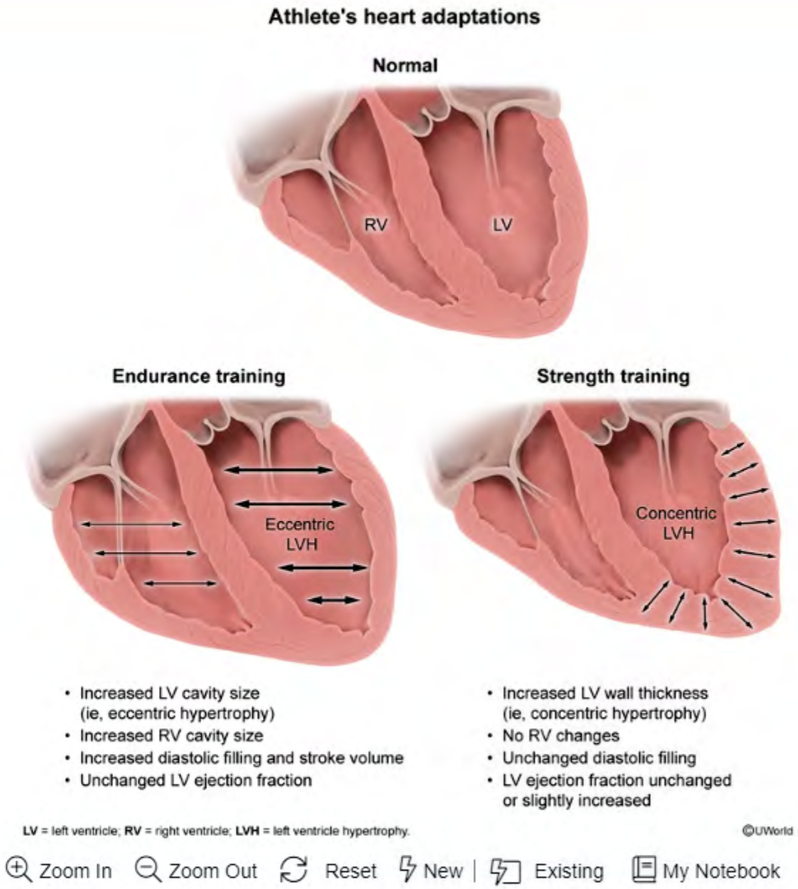

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A 56-year-old woman comes to the emergency department due to 3 days of fever, chills, and retrosternal chest pain. She has end-stage kidney disease related to previous uncontrolled hypertension and receives intermittent hemodialysis through a tunneled catheter. Temperature is 39 C (102.2 F), blood pressure is 108/64 mm Hg, and pulse is 120/min. The patient is ill-appearing. The lungs are clear to auscultation, but a pericardial friction rub is present. Echocardiography reveals a moderate-sized pericardial effusion. Pericardiocentesis yields turbid fluid with a large number of neutrophils. Microbiologic analysis of this patient's pericardial fluid is most likely to reveal which of the following pathogens?

- A. Bacilli with acid-fast cell walls
- B. Budding yeasts forming germ tubes
- C. Gram-positive cocci growing in clusters
- D. Lactose-fermenting gram-negative bacilli
- E. Silver stain–positive motile spirochetes
- F. Single-stranded RNA virus

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- A. Bacilli with acid-fast cell walls (2%)
- ✗ B. Budding yeasts forming germ tubes (4%)
- ✓ C. Gram-positive cocci growing in clusters (74%)
- D. Lactose-fermenting gram-negative bacilli (9%)
- E. Silver stain–positive motile spirochetes (0%)
- F. Single-stranded RNA virus (8%)

Incorrect
Correct answer
C

74%
Answered correctly

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Explanation

Purulent pericarditis	
Pathogenesis	• Hematologic spread from a distant source • Direct extension from a heart or lung infection

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Explanation

Purulent pericarditis	
Pathogenesis	• Hematologic spread from a distant source • Direct extension from a heart or lung infection • Complication of penetrating injury or chest wall trauma
Common pathogens	• *Staphylococcus aureus* (portal from skin) • *Streptococcus pneumoniae* (adjacent pneumonia) • *Candida albicans* (TPN, severe immunosuppression)
Manifestations	• Fever, chills, fatigue, chest pain • Examination: pericardial friction rub • Echocardiogram: pericardial effusion • Pericardiocentesis: high number of leukocytes/frank pus
TPN = total parenteral nutrition.	

This patient with fever and chest pain has a **turbid** pericardial effusion with a high number of neutrophils, indicating **purulent pericarditis**. Most cases are caused by bacteria or fungi and develop due to:

- Hematologic spread from a distant infection
- Direct extension from an adjacent pneumonia or cardiac infection (eg, endocarditis, perivalvular abscess)
- Complication of penetrating chest injury or cardiothoracic surgery

Staphylococcus aureus, a **gram-positive cocci** that grows in **clusters**, is the most frequently isolated organism. It is particularly likely in patients who have portals from the skin to the bloodstream (eg, **tunneled dialysis catheter**) or from the skin directly to the pericardium (eg, chest injury, recent cardiothoracic surgery).
Streptococcus pneumoniae is the most common organism in patients with adjacent pneumonia.

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Although *Candida albicans*, a budding yeast that forms germ tubes, is a leading cause of fungal pericarditis, it is seen primarily in persons with significant risk factors for candidemia such as parenteral feeding, prolonged corticosteroid use, or immunosuppression due to malignancy; *S aureus* is far more common in patients with end-stage renal disease who have vascular catheters **(Choice B)**.

(Choice A) *Mycobacterium tuberculosis*, an acid-fast bacilli, can occasionally cause purulent pericarditis due to direct spread from the lung or a mediastinal/hilar lymph node, but *S aureus* is far more likely in someone with a hemodialysis catheter.

(Choice D) Lactose-fermenting gram-negative bacilli (eg, *Escherichia coli*, *Klebsiella pneumoniae*, Enterobacter) are uncommon causes of purulent pericarditis. Although *Klebsiella* pericarditis can occasionally occur due to adjacent anaerobic lung abscess, this patient with an indwelling catheter is far more likely to have *S aureus* infection.

(Choice E) *Borrelia burgdorferi* is a motile spirochete seen with silver stain. It can cause Lyme myopericarditis as a consequence of early disseminated disease, but most cases are mild and asymptomatic. In addition, an effusion caused by *B burgdorferi* infection would be lymphocyte (not neutrophil) predominant.

(Choice F) Although single-stranded RNA viruses such as Coxsackievirus can cause pericarditis, the pericardial

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(Choice F) Although single-stranded RNA viruses such as Coxsackievirus can cause pericarditis, the pericardial fluid is usually lymphocyte (not neutrophil) predominant.

Educational objective:
Purulent pericarditis is usually caused by hematogenous dissemination from distant infection or direct extension from an adjacent infection or chest wall trauma. Although a variety of organisms cause purulent pericarditis, *Staphylococcus aureus* is the most common pathogen, particularly in the setting of a portal from the skin to the bloodstream (eg, catheter) or pericardium (eg, recent chest surgery, penetrating injury).

References

- Diagnosis and management of pericardial effusion.

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A term newborn is evaluated for cyanosis immediately after birth. On examination, the patient's oxygen saturation is 70% in all 4 extremities and does not change despite 100% oxygen administration. The lungs are clear to auscultation and there are no heart murmurs or gallops. Peripheral pulses are normal. A chest radiograph reveals clear lungs and a normal cardiac silhouette but a narrowed mediastinal shadow. Which of the following is the most likely cause of cyanosis in this patient?

- A. Impairment of alveolar-capillary gas diffusion
- B. Impairment of left ventricular contractility
- C. Inability of hemoglobin to bind oxygen
- D. Mismatch of pulmonary ventilation and perfusion
- E. Parallel pulmonary and systemic circuits

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- ✗ A. Impairment of alveolar-capillary gas diffusion (7%)
- B. Impairment of left ventricular contractility (5%)
- C. Inability of hemoglobin to bind oxygen (5%)
- D. Mismatch of pulmonary ventilation and perfusion (18%)
- ✓ E. Parallel pulmonary and systemic circuits (62%)

Incorrect
Correct answer
E

62%
Answered correctly

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Explanation

Transposition of the great arteries	
Anatomy	• Parallel (separate) systemic & pulmonary circuits ◦ Right ventricle → aorta ◦ Left ventricle → pulmonary artery • Deoxygenated blood is delivered to the systemic circulation • Mixing shunt (eg, VSD, PDA) must be present for viability

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Explanation

Transposition of the great arteries	

Anatomy	• Parallel (separate) systemic & pulmonary circuits
	  ◦ Right ventricle → aorta
	  ◦ Left ventricle → pulmonary artery
	• Deoxygenated blood is delivered to the systemic circulation
	• Mixing shunt (eg, VSD, PDA) must be present for viability

Clinical features	• Cyanosis at or shortly after birth • Tachypnea & tachycardia • Murmur typically absent at birth due to low velocity shunting
Diagnosis	• Chest x-ray: narrow mediastinum ± cardiomegaly ("egg on a string") • Hyperoxia test: little to no ↑ in arterial oxygenation with 100% O_2 • Echocardiography typically confirmatory

Initial management	• Promote mixing of oxygenated & deoxygenated blood
	  ◦ Prostaglandin infusion (maintains PDA)
	  ◦ Atrial septostomy (maintains PFO)

PDA = patent ductus arteriosus; **PFO** = patent foramen ovale; **VSD** = ventricular septal defect.	

This **cyanotic** newborn has severe hypoxemia that does not improve with oxygen administration. This finding suggests a congenital heart defect in which deoxygenated blood bypasses the pulmonary circulation and is delivered directly to the systemic circulation (ie, right-to-left shunt). In this case, the most likely etiology is **transposition of the great arteries** (TGA), in which the aorta originates from the right ventricle and the pulmonary artery from the left ventricle; the abnormal position of the aorta atop the pulmonary artery creates a **narrow-appearing mediastinum**.

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The anatomy of TGA results in **disconnected and parallel circuits** with delivery of deoxygenated blood from the right ventricle to the systemic circulation via the aorta (effectively a right-to-left shunt). Cyanosis occurs shortly after birth but does not improve with oxygen administration (ie, **hyperoxia test**) because the oxygenated blood cannot reach the systemic circulation; instead, it is cycled from the lungs to the left side of the heart and back to the lungs.

A mixing shunt (eg, patent ductus arteriosus, ventricular septal defect) that can deliver some oxygenated blood to the systemic circulation must be present for viability in TGA; an associated murmur can sometimes be present based on the size and type of mixing lesion, but many patients with TGA have **no murmur**.

(Choices A and D) Preterm newborns are at risk for neonatal respiratory distress syndrome (RDS) causing hypoxemia due to ventilation/perfusion mismatch and a modest reduction in diffusion capacity. However, hypoxemia due to ventilation/perfusion mismatch and/or diffusion impairment is expected to at least somewhat improve with oxygen administration. Furthermore, RDS is unlikely in a neonate born at term and affected patients have diffuse, ground-glass opacities on lung imaging.

(Choice B) Impaired left ventricular contractility (ie, heart failure) does not cause hypoxemia directly but can cause hypoxemia if it leads to pulmonary edema. This patient's clear lungs on auscultation and chest x-ray rule out significant pulmonary edema.

(Choice C) Congenital methemoglobinemia, or the inability to reduce methemoglobin back to hemoglobin, is a rare cause of neonatal cyanosis. Oxygen saturation fails to improve with oxygen administration, but

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(Choice B) Impaired left ventricular contractility (ie, heart failure) does not cause hypoxemia directly but can cause hypoxemia if it leads to pulmonary edema. This patient's clear lungs on auscultation and chest x-ray rule out significant pulmonary edema.

(Choice C) Congenital methemoglobinemia, or the inability to reduce methemoglobin back to hemoglobin, is a rare cause of neonatal cyanosis. Oxygen saturation fails to improve with oxygen administration, but methemoglobinemia does not explain a narrowed mediastinum on chest x-ray.

Educational objective:
Cyanotic congenital heart disease is characterized by hypoxemia that does not improve with oxygen administration. In transposition of the great arteries, deoxygenated blood from the right ventricle is delivered to the systemic circulation while oxygenated blood cycles through the pulmonary circulation (ie, parallel circuits).

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- Murmur typically absent at birth due to low velocity shunting

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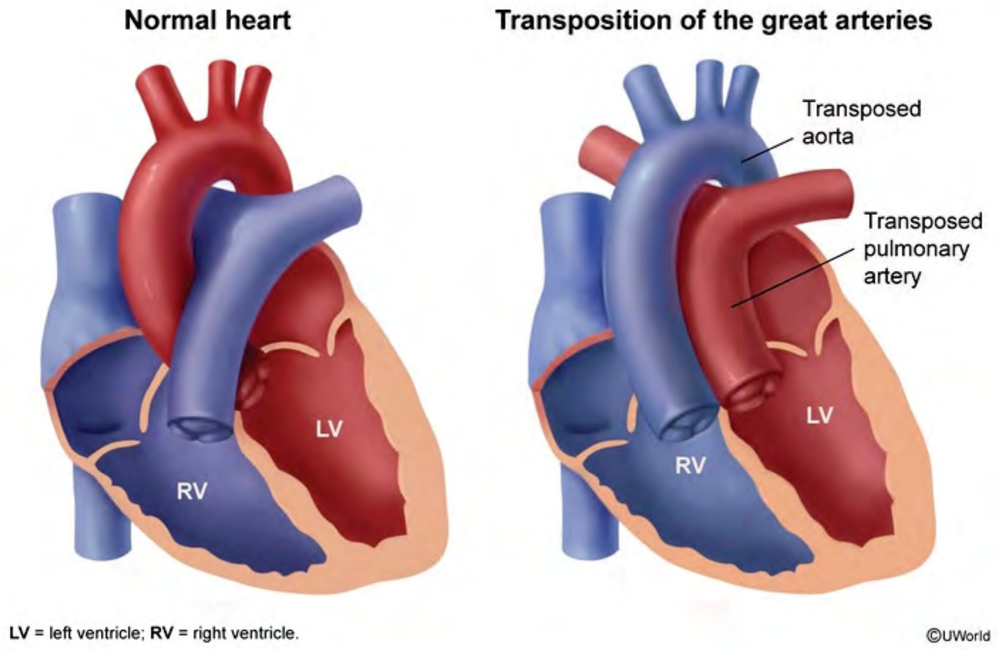

LV = left ventricle; RV = right ventricle.

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- Echocardiography typically confirmatory

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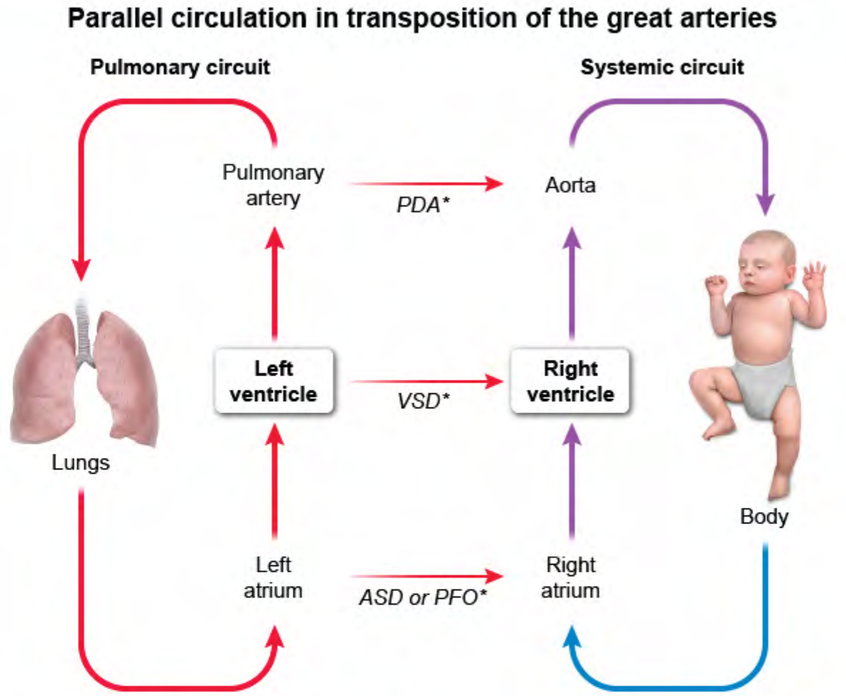

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A 55-year-old man comes to the office due to recurrent episodes of retrosternal chest pain. The episodes occur during physical activity, usually when he climbs stairs or walks uphill, and resolve after he rests for a few minutes. The patient has no known medical conditions and does not use tobacco, alcohol, or recreational drugs. He is prescribed sublingual nitroglycerin to take as needed when the chest pain occurs. This drug is most likely to relieve this patient's symptoms by causing which of the following hemodynamic changes?

- A. Decrease in heart rate
- B. Decrease in left ventricular end-diastolic volume
- C. Decrease in peripheral arteriolar resistance
- D. Increase in coronary blood flow
- E. Increase in coronary perfusion pressure

Submit

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- D. Increase in coronary blood flow
- E. Increase in coronary perfusion pressure

Incorrect
Correct answer
B

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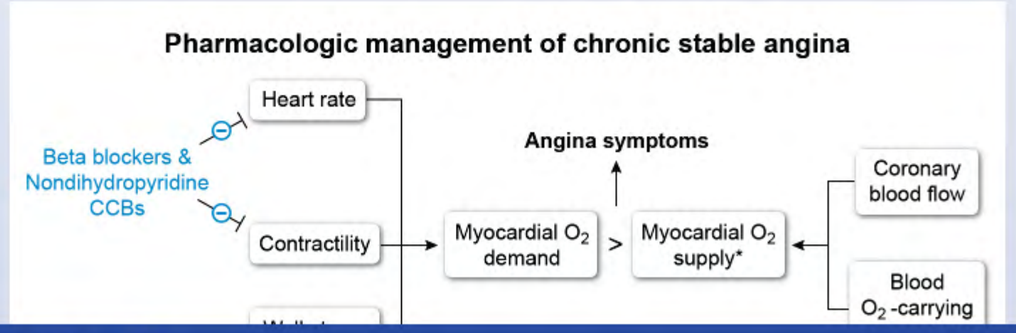
Explanation



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D. Increase in coronary blood flow

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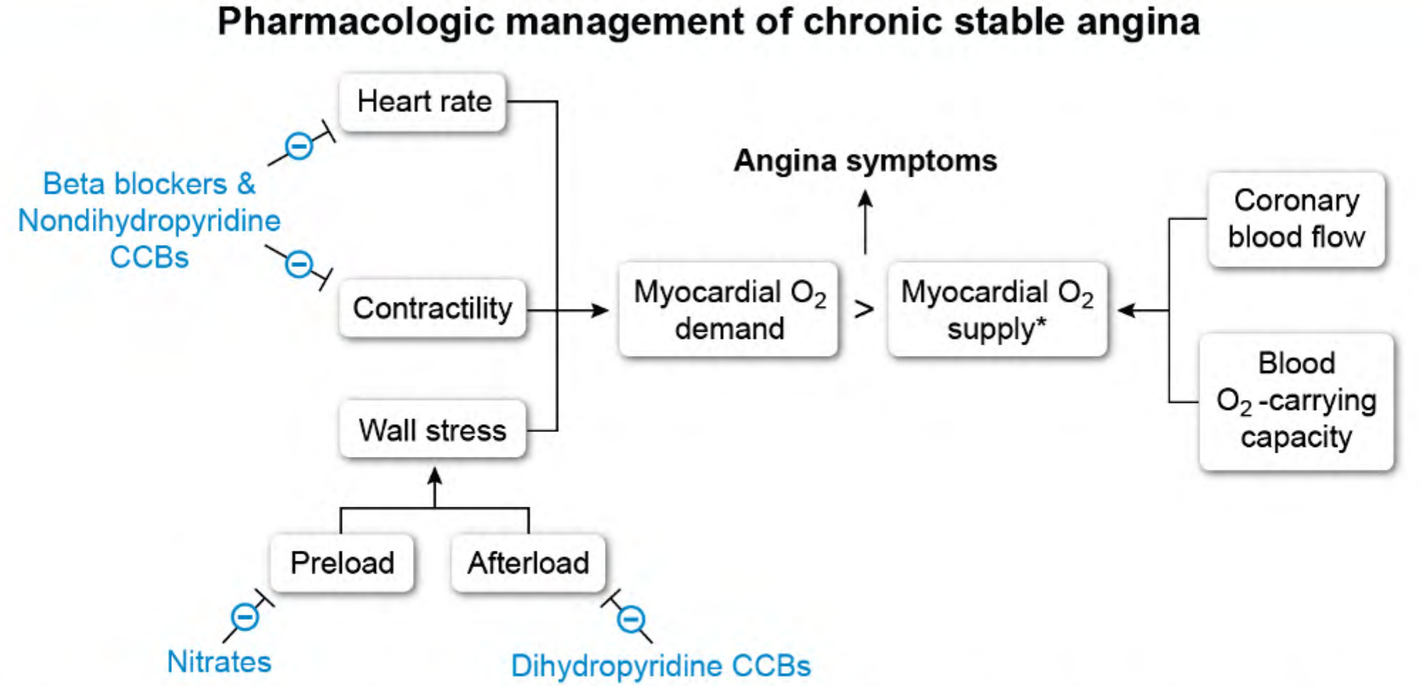

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flow (and oxygen supply) cannot be achieved to meet the increased myocardial oxygen demand of exercise.

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This patient's retrosternal chest pain brought on by exertion and relieved with rest is classic for **stable angina**. It is caused by atherosclerotic narrowing of the coronary arteries such that an adequate increase in myocardial blood flow (and oxygen supply) cannot be achieved to meet the increased myocardial oxygen demand of exercise. Medical therapy for stable angina is aimed at relieving this **myocardial oxygen supply/demand mismatch**.

Nitrates are commonly used to treat stable angina. Long-acting formulations (eg, isosorbide dinitrate) are used for prevention of stable angina, and short-acting formulations (eg, sublingual nitroglycerin) are used as an abortive agent when symptoms occur. The primary effect of nitrates is **venodilation**, which causes additional blood to remain in the capacitance veins and reduces venous return to the heart. The resulting **decrease** in left ventricular (**LV**) **end-diastolic volume** (ie, reduced preload) decreases **LV wall stress** to reduce myocardial **oxygen demand** and relieve angina symptoms.

(Choice A) Decreased heart rate reduces myocardial oxygen demand. Beta blockers (eg, metoprolol) and nondihydropyridine calcium channel blockers (eg, verapamil) treat stable angina in part via this mechanism, but nitrates have no direct effect on heart rate (and may cause a reflexive increase in heart rate).

(Choice C) Decreased peripheral arteriolar resistance decreases cardiac afterload to reduce LV wall stress and decrease myocardial oxygen demand. Dihydropyridine calcium channel blockers (eg, amlodipine) treat stable angina via this mechanism, but nitrates cause only slight peripheral arteriolar vasodilation.

(Choices D and E) Nitrates do stimulate some degree of coronary arteriolar vasodilation, but this mechanism has little effect on relieving angina symptoms. Coronary arterioles with substantial atherosclerotic disease are already nearly fully dilated at baseline such that the capacity for further dilation to recruit additional blood flow is minimal. Nitrates can also slightly augment the coronary perfusion pressure gradient (ie, aortic diastolic pressure − LV diastolic pressure) by *decreasing LV end-diastolic volume*. This may slightly increase coronary blood flow and myocardial oxygen supply, but the reduction in myocardial oxygen demand via decreased LV wall stress is a much larger contributor to relief of angina symptoms.

Educational objective:
Sublingual nitroglycerin is used for rapid symptom relief in patients with stable angina. The primary anti-ischemic

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Medical therapy for stable angina is aimed at relieving this **myocardial oxygen supply/demand mismatch**.

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Educational objective:
Sublingual nitroglycerin is used for rapid symptom relief in patients with stable angina. The primary anti-ischemic effect of nitrates is mediated by venodilation causing a decrease in left ventricular end-diastolic volume and wall stress, resulting in decreased myocardial oxygen demand.

Pharmacology Cardiovascular System Nitrates

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A 60-year-old man comes to the hospital with chest pain. He has had intermittent, squeezing substernal pain over the last 3 days that is now sustained. The patient has no associated palpitations, lightheadedness, or shortness of breath. Medical history is notable for moderate chronic obstructive pulmonary disease with a recent admission for an exacerbation. Blood pressure is 145/90 mm Hg and pulse is 93/min. Oxygen saturation by pulse oximetry is 98% on room air. Physical examination shows no heart murmurs, and lungs are clear to auscultation. ECG shows sinus rhythm with 2-mm anterior ST segment elevation. Cardiac troponin I levels are elevated. Which of the following is the most appropriate treatment for this patient?

- A. Ibutilide
- B. Metoprolol
- C. Nadolol
- D. Propranolol
- E. Sotalol

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A 60-year-old man comes to the hospital with chest pain. He has had intermittent, squeezing substernal pain over the last 3 days that is now sustained. The patient has no associated palpitations, lightheadedness, or shortness of breath. Medical history is notable for moderate chronic obstructive pulmonary disease with a recent admission for an exacerbation. Blood pressure is 145/90 mm Hg and pulse is 93/min. Oxygen saturation by pulse oximetry is 98% on room air. Physical examination shows no heart murmurs, and lungs are clear to auscultation. ECG shows sinus rhythm with 2-mm anterior ST segment elevation. Cardiac troponin I levels are elevated. Which of the following is the most appropriate treatment for this patient?

- ✗ A. Ibutilide (8%)
- ✓ B. Metoprolol (68%)
- C. Nadolol (3%)
- D. Propranolol (11%)
- E. Sotalol (7%)

Incorrect
Correct answer
B

68%
Answered correctly

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Time Spent

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Explanation

Effects of adrenergic blockade			
Receptor	**Target organ**	**Effect**	**Potential adverse effects**
B1-adrenergic	Heart	↓ Heart rate ↓ Contractility	Worsening of heart failure Bradycardia/heart block

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Effects of adrenergic blockade			
Receptor	**Target organ**	**Effect**	**Potential adverse effects**
B1-adrenergic blockade	Heart	↓ Heart rate ↓ Contractility ↓ O_2 consumption	Worsening of heart failure Bradycardia/heart block
	Kidney	↓ Renin release	
B2-adrenergic blockade	Muscle vasculature	Vasospasm	Exacerbation of peripheral artery disease
	Lungs	Bronchoconstriction	Worsening of obstructive lung disease
	Liver	↓ Glycogenolysis & gluconeogenesis	May cause hypoglycemia in diabetic patients
	Most tissues	↓ Potassium entry into cells	Hyperkalemia

This patient's clinical presentation—chest discomfort, ST elevation on ECG, elevated troponin I level—is consistent with **acute myocardial infarction** (AMI). Beta blockers are used in AMI to reduce myocardial oxygen demand by lowering heart rate, cardiac contractility, and afterload. They have been shown to reduce short-term morbidity (eg, recurrent symptoms or reinfarction), minimize infarct size, and improve long-term survival.

Contraindications to beta blockers include bradycardia or heart block, hypotension, and overt heart failure (eg, pulmonary edema). Also, **non-cardioselective beta blockers** (eg, propranolol, nadolol) can **trigger bronchospasm** in patients with underlying obstructive lung disease (asthma, **chronic obstructive pulmonary disease** [COPD]) due to beta-2 receptor blockade and should be avoided **(Choices C and D)**.

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blockade	Liver	↓ Glycogenolysis & gluconeogenesis	May cause hypoglycemia in diabetic patients
	Most tissues	↓ Potassium entry into cells	Hyperkalemia

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Cardioselective beta blockers with predominant action on beta-1 receptors (eg, **metoprolol**, atenolol, bisoprolol, nebivolol) are **safe** in patients with stable obstructive lung disease and are the beta blocker of choice in these patients. Combined beta and alpha receptor blockers (eg, carvedilol, labetalol) are also well tolerated and have been used safely in patients with COPD.

(Choice A) Ibutilide is a class III antiarrhythmic drug (potassium channel blocker) used for acute termination of atrial flutter or atrial fibrillation. Like other class III antiarrhythmics, ibutilide causes prolonged QT interval that can precipitate a deadly arrhythmia (torsade de pointes).

(Choice E) Sotalol is a class III antiarrhythmic drug with beta blocker properties and is primarily used for the treatment of atrial and ventricular arrhythmias. It would not be indicated in a patient with a recent myocardial infarction who is in sinus rhythm.

Educational objective:
Beta blockers are used in acute myocardial infarction to reduce morbidity and mortality by decreasing cardiac output and myocardial oxygen demand. Non-cardioselective beta blockers (eg, propranolol, nadolol) can trigger

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	Most tissues	↓ Potassium entry into cells	Hyperkalemia

This patient's clinical presentation—chest discomfort, ST elevation on ECG, elevated troponin I level—is consistent with **acute myocardial infarction** (AMI). Beta blockers are used in AMI to reduce myocardial oxygen demand by lowering heart rate, cardiac contractility, and afterload. They have been shown to reduce short-term morbidity (eg, recurrent symptoms or reinfarction), minimize infarct size, and improve long-term survival.

Contraindications to beta blockers include bradycardia or heart block, hypotension, and overt heart failure (eg, pulmonary edema). Also, **non-cardioselective beta blockers** (eg, propranolol, nadolol) can **trigger bronchospasm** in patients with underlying obstructive lung disease (asthma, **chronic obstructive pulmonary disease** [COPD]) due to beta-2 receptor blockade and should be avoided **(Choices C and D)**.

Cardioselective beta blockers with predominant action on beta-1 receptors (eg, **metoprolol**, atenolol, bisoprolol, nebivolol) are **safe** in patients with stable obstructive lung disease and are the beta blocker of choice in these patients. Combined beta and alpha receptor blockers (eg, carvedilol, labetalol) are also well tolerated and have been used safely in patients with COPD.

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(Choice E) Sotalol is a class III antiarrhythmic drug with beta blocker properties and is primarily used for the treatment of atrial and ventricular arrhythmias. It would not be indicated in a patient with a recent myocardial infarction who is in sinus rhythm.

Educational objective:
Beta blockers are used in acute myocardial infarction to reduce morbidity and mortality by decreasing cardiac output and myocardial oxygen demand. Non-cardioselective beta blockers (eg, propranolol, nadolol) can trigger bronchospasm in patients with underlying asthma or chronic obstructive pulmonary disease. Cardioselective beta blockers (eg, metoprolol) predominantly affect beta-1 receptors and are preferred in such patients.

References

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 12-month-old boy is brought to the pediatrician by his parents for an upper respiratory infection. Temperature is 38.3 C (101 F), blood pressure is 92/45 mm Hg, pulse is 110/min, and respirations are 25/min. Physical examination shows erythema and swelling of the nasal mucosa and nasal discharge. Cardiac auscultation findings at the left sternal border are given below. Pulses are equal in all 4 extremities. The remainder of the examination is unremarkable. Which of the following is the most likely diagnosis?

Play Media

- A. Aortic coarctation
- B. Ascending aortic aneurysm
- C. Atrial septal defect
- D. Mitral valve prolapse
- E. Patent ductus arteriosus

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 12-month-old boy is brought to the pediatrician by his parents for an upper respiratory infection. Temperature is 38.3 C (101 F), blood pressure is 92/45 mm Hg, pulse is 110/min, and respirations are 25/min. Physical examination shows erythema and swelling of the nasal mucosa and nasal discharge. Cardiac auscultation findings at the left sternal border are given below. Pulses are equal in all 4 extremities. The remainder of the examination is unremarkable. Which of the following is the most likely diagnosis?

Play Media

- A. Aortic coarctation (2%)
- B. Ascending aortic aneurysm (1%)
- C. Atrial septal defect (18%)
- D. Mitral valve prolapse (10%)
- ✓ E. Patent ductus arteriosus (67%)

Correct

67%
Answered correctly

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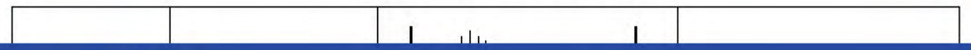
Explanation

Cardiac murmurs



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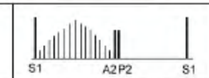
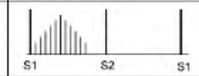
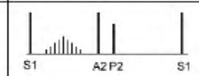
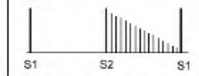
Cardiac murmurs

Systolic	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid-left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation*		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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Diastolic | Mitral | Opening snap followed

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This patient's auscultation findings reveal a continuous murmur with maximal intensity at S2 (when pressure in the aorta is at a peak), which is characteristic of a **patent ductus arteriosus** (PDA). The murmur is usually best heard at the left infraclavicular or posterior interscapular region. The symptoms caused by a PDA are variable depending on its size and resulting **degree of left-to-right shunting** from the aorta to the pulmonary arterial trunk. Because this patient was **asymptomatic** and the PDA was detected incidentally, he most likely has a **small PDA**. Larger PDAs typically present with respiratory distress, poor feeding, and/or retracted growth during the first year of life. Patients at greatest risk of PDA are those born prematurely and those with cyanotic congenital heart disease.

(Choice A) Aortic coarctation is usually associated with a systolic murmur best heard at the left infraclavicular area. It can sometimes cause a continuous murmur due to blood flow through large collateral vessels, but this is a much less common cause of continuous murmur than a PDA. In addition, equal pulses in the upper and lower extremities make aortic coarctation unlikely.

(Choice B) An ascending aortic aneurysm is often associated with aortic regurgitation, which causes a decrescendo early diastolic murmur as opposed to a continuous murmur. Aortic regurgitation resulting from aortic root (as opposed to valvular) pathology is typically best heard at the right upper sternal border.

(Choice C) An atrial septal defect typically causes a faint, crescendo-decrescendo systolic murmur (best heard at the left upper sternal border) caused by increased blood flow through the pulmonic valve (ie, pulmonic flow murmur). Splitting of S2 is wide and fixed.

(Choice D) Mitral valve prolapse classically causes a midsystolic click followed by a late systolic murmur of mitral regurgitation (best heard at the cardiac apex). The timing of the click (and murmur) varies with changes in left ventricular blood volume.

Educational objective:
A patent ductus arteriosus (PDA) causes a continuous murmur best heard at the left infraclavicular or posterior interscapular region. A small PDA is often asymptomatic and detected incidentally during routine cardiac auscultation. It most commonly occurs in patients born prematurely and those with cyanotic congenital heart

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Educational objective:
A patent ductus arteriosus (PDA) causes a continuous murmur best heard at the left infraclavicular or posterior interscapular region. A small PDA is often asymptomatic and detected incidentally during routine cardiac auscultation. It most commonly occurs in patients born prematurely and those with cyanotic congenital heart disease.

Pathophysiology	Cardiovascular System	Patent ductus arteriosus
Subject	System	Topic

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	arteriosus	S1 S2 S1	left infraclavicular area

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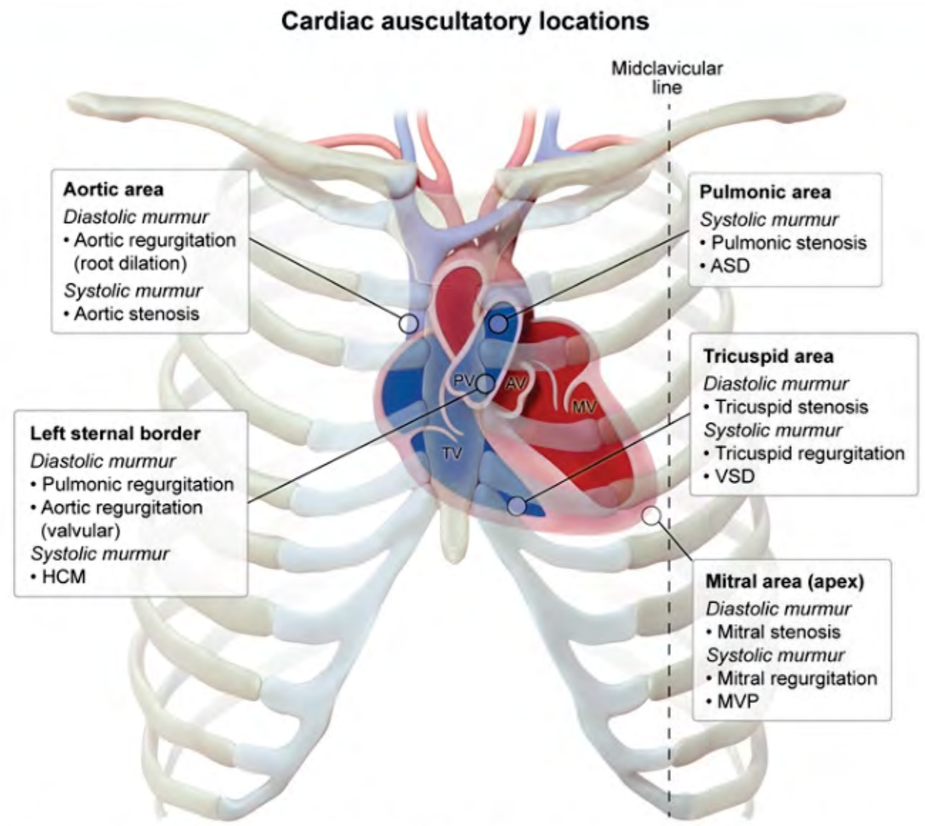

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A 42-year-old man comes to the emergency department due to severe chest pain that started abruptly 2 hours ago. The pain is midline, constant, and 10/10 in intensity. He has had no fever, cough, or shortness of breath. His only medical condition is hypertension. On examination, the patient is diaphoretic and is in severe distress due to pain. Temperature is 37 C (98.6 F), pulse is 116/min, and respirations are 24/min. Systolic blood pressure is 82 mm Hg in the right arm and 60 mm Hg in the left arm. Jugular veins are distended with an estimated pressure of 13 cm H_2O. The lungs are clear to auscultation. The point of maximal impulse is not palpable. The extremities are cold with no peripheral edema. The patient develops cardiac arrest and dies soon after arrival. Autopsy would most likely reveal which of the following findings?

- A. Air within the pleural space
- B. Blood in the pericardial space
- C. Ruptured posteromedial papillary muscle
- D. Thrombus at the pulmonary artery bifurcation
- E. Transmural necrosis of the anterolateral myocardium

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- A. Air within the pleural space (0%)
- ✓ B. Blood in the pericardial space (62%)
- C. Ruptured posteromedial papillary muscle (9%)
- D. Thrombus at the pulmonary artery bifurcation (14%)
- E. Transmural necrosis of the anterolateral myocardium (11%)

Correct

62% Answered correctly

06 secs Time Spent

2023 Version

Explanation

Acute aortic dissection	
Pathophysiology	• Intimal tear allows pressurized blood into aortic wall • Propagation of dissection creates false aortic lumen • Complications arise from extension into nearby structures

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Explanation

Acute aortic dissection	
Pathophysiology	• Intimal tear allows pressurized blood into aortic wall • Propagation of dissection creates false aortic lumen • Complications arise from extension into nearby structures
Risk factors	• Chronic hypertension (strongest overall risk factor) • Underlying aortopathy (eg, Marfan syndrome)
Clinical presentation	• Sudden-onset, severe chest and/or back pain • Hypertension usually present* • Often a >20 mm Hg variation in SBP between arms • Often a widened mediastinum on chest x-ray
*Hypotension on presentation suggests aortic rupture or other complication (eg, cardiac tamponade, acute aortic regurgitation). **SBP** = systolic blood pressure.	

This patient's presentation is most consistent with an **ascending aortic dissection** complicated by **cardiac tamponade**. Dissections can affect the ascending aorta (type A) or may involve only the descending aorta (type B). The most common symptom is sharp or tearing **chest and back pain** that is severe and sudden. If an ascending dissection extends proximally to the aortic root, it can affect the coronary ostia or aortic valve, resulting in myocardial ischemia or aortic regurgitation, respectively. Other signs and symptoms include **blood pressure asymmetry**, stroke, or paraplegia, depending on the vessels and structures involved (eg, subclavian artery, carotid artery, spinal arteries).

In addition, a dissection may extend proximally into the **pericardium**, causing blood to fill the pericardial space and leading to acute tamponade. As blood accumulates, the rising pericardial pressure can rapidly exceed right-sided

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In addition, a dissection may extend proximally into the **pericardium**, causing blood to fill the pericardial space and leading to acute tamponade. As blood accumulates, the rising pericardial pressure can rapidly exceed right-sided filling pressures and restrict venous return, resulting in a precipitous drop in cardiac output and **obstructive shock** that can lead to cardiac arrest. Signs of tamponade include jugular venous distension, muffled heart sounds, and hypotension (Beck triad), as well as reduced point of maximal impulse, cool extremities, and clear lungs.

(Choice A) Air in the pleural space is expected with tension pneumothorax, which can cause obstructive shock due to mediastinal shifting with vena cava collapse and impaired venous return to the heart. Although sudden-onset chest pain is common, shortness of breath and unilaterally absent breath sounds are expected.

(Choice C) A ruptured posteromedial papillary muscle causes acute mitral regurgitation, which can abruptly lead to cardiogenic shock. Patients are expected to have shortness of breath and lung crackles due to acute pulmonary edema.

(Choice D) A saddle pulmonary thromboembolism can cause obstructive shock with right ventricular failure and jugular venous distension. However, it would not account for upper extremity blood pressure asymmetry or an unpalpable point of maximal impulse.

(Choice E) Transmural necrosis of the anterolateral myocardium can cause left ventricular free wall rupture with patients developing acute tamponade as blood rapidly fills the pericardium. This etiology of tamponade would not explain upper extremity blood pressure asymmetry.

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artery, spinal arteries).

In addition, a dissection may extend proximally into the **pericardium**, causing blood to fill the pericardial space and leading to acute tamponade. As blood accumulates, the rising pericardial pressure can rapidly exceed right-sided filling pressures and restrict venous return, resulting in a precipitous drop in cardiac output and **obstructive shock** that can lead to cardiac arrest. Signs of tamponade include jugular venous distension, muffled heart sounds, and hypotension (Beck triad), as well as reduced point of maximal impulse, cool extremities, and clear lungs.

(Choice A) Air in the pleural space is expected with tension pneumothorax, which can cause obstructive shock due to mediastinal shifting with vena cava collapse and impaired venous return to the heart. Although sudden-onset chest pain is common, shortness of breath and unilaterally absent breath sounds are expected.

(Choice C) A ruptured posteromedial papillary muscle causes acute mitral regurgitation, which can abruptly lead to cardiogenic shock. Patients are expected to have shortness of breath and lung crackles due to acute pulmonary edema.

(Choice D) A saddle pulmonary thromboembolism can cause obstructive shock with right ventricular failure and jugular venous distension. However, it would not account for upper extremity blood pressure asymmetry or an unpalpable point of maximal impulse.

(Choice E) Transmural necrosis of the anterolateral myocardium can cause left ventricular free wall rupture with patients developing acute tamponade as blood rapidly fills the pericardium. This etiology of tamponade would not explain upper extremity blood pressure asymmetry.

Educational objective:
Acute aortic dissection typically presents with sudden-onset, severe chest pain that is sharp or tearing in nature. Upper extremity blood pressure asymmetry can result from extension of the dissection into the subclavian artery, and additional complications (eg, stroke, myocardial ischemia) can result from extension into other vessels. Extension into the pericardium can cause acute tamponade with reduced cardiac output and shock.

References

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*Hypotension on presentation suggests aortic rupture or other complication (eg, cardiac tamponade

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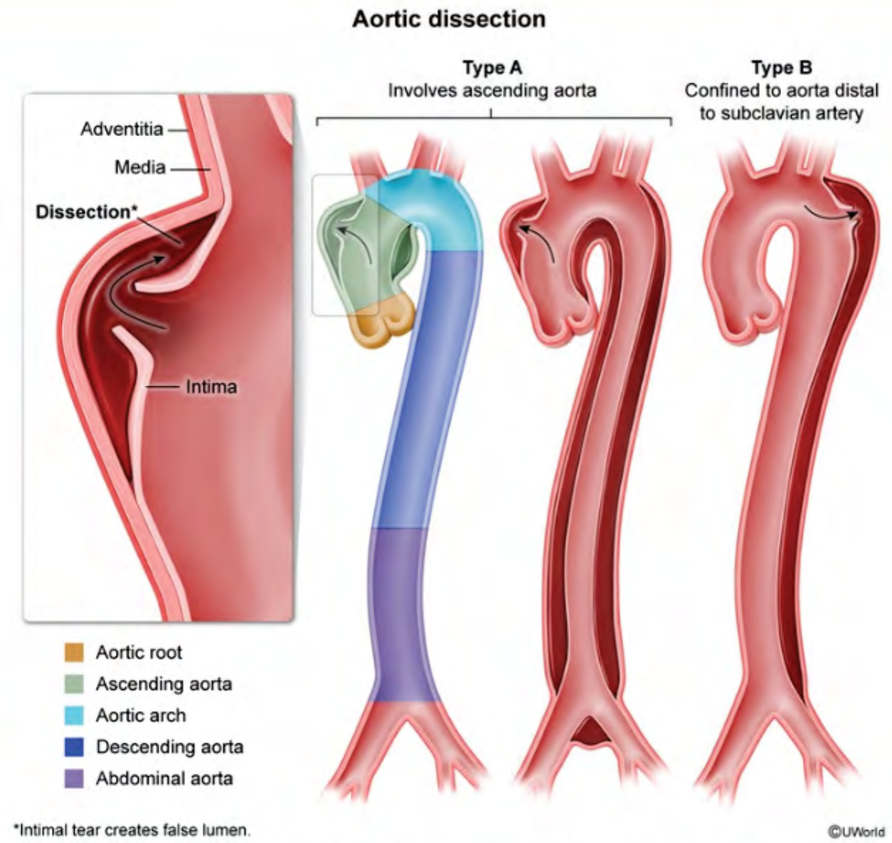

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*Hypotension on presentation suggests aortic rupture or other complication (eg, cardiac tamponade

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Complications due to extension of acute aortic dissection	
Complication (involved structure)	• Stroke (carotid artery) • Horner syndrome (carotid sympathetic plexus) • Acute aortic regurgitation (aortic root/valve) • Myocardial ischemia/infarction (coronary artery ostia) • Pericardial effusion/tamponade (pericardium) • Hemothorax (pleural cavity) • Renal infarction (renal arteries) • Intestinal ischemia (mesenteric arteries) • Lower extremity paralysis (spinal arteries)

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A 19-year-old man comes to the office due to difficulty seeing and blurred vision, which have worsened slowly over the past year. He is a second-year college student pursuing a degree in biochemistry. His grades are excellent, but he is concerned about the effect his poor vision has had on his classes this semester. The patient is also an avid swimmer. He weighs 71 kg (156.5 lb) and is 195 cm (6 ft 5 in) tall. Physical examination shows the findings seen in the exhibits. This patient is most likely to die from which of the following conditions?

- A. Aortic disease
- B. Liver disease
- C. Myocardial infarction
- D. Overwhelming infection
- E. Renal failure

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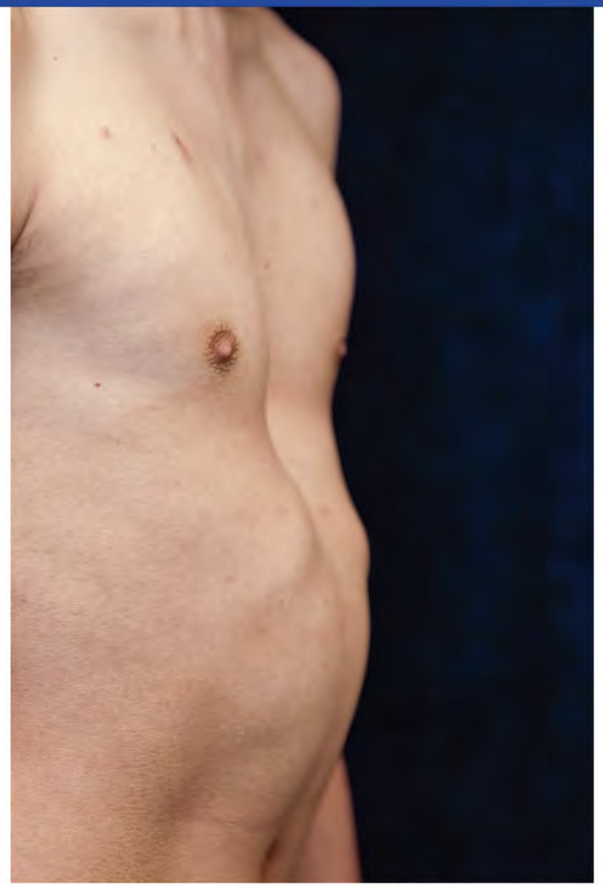

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- ✓ A. Aortic disease (88%)
- ✗ B. Liver disease (2%)
- C. Myocardial infarction (6%)
- D. Overwhelming infection (1%)
- E. Renal failure (1%)

Incorrect
Correct answer
A

88%
Answered correctly

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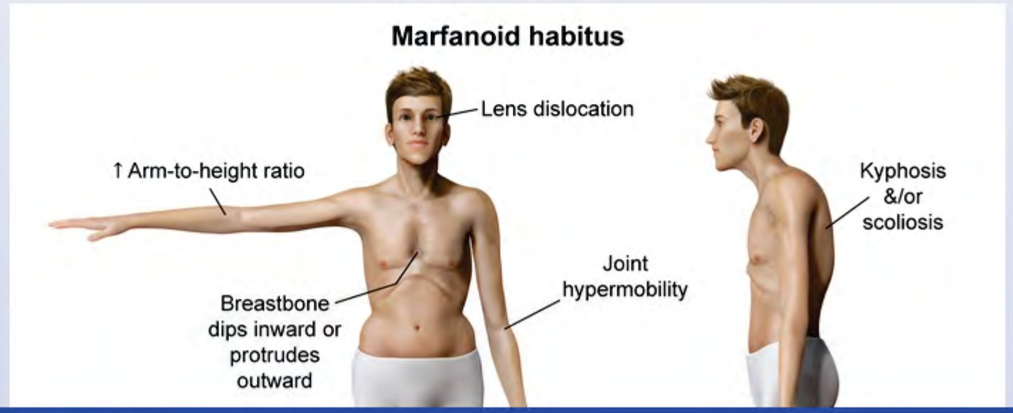
Explanation



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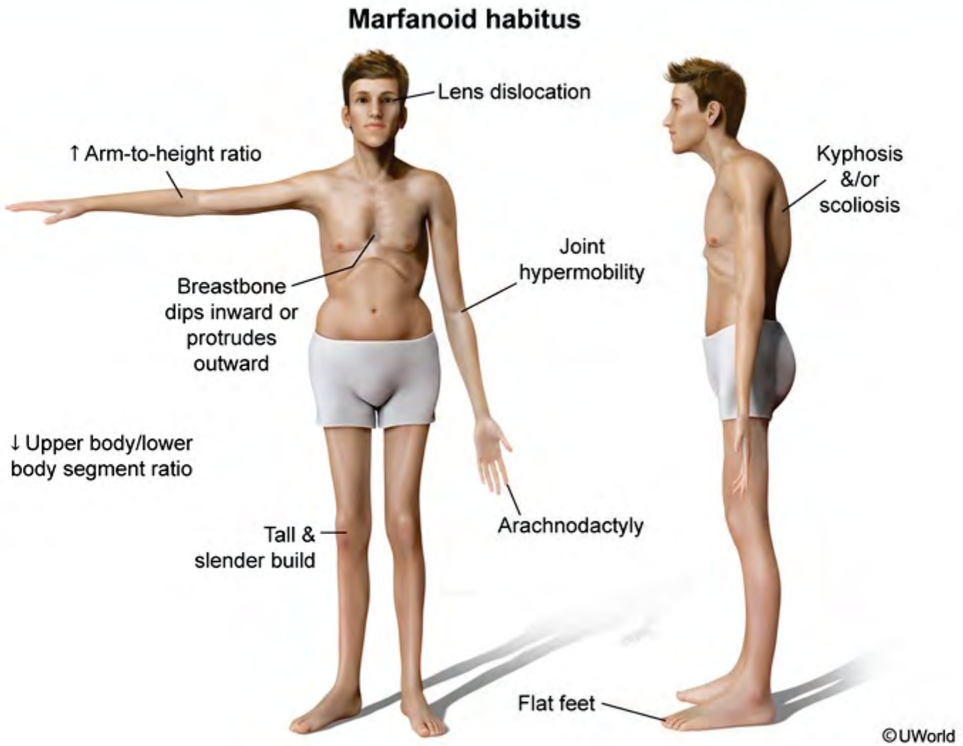

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Marfan syndrome (MFS) is one of the most common inherited connective tissue disorders. This condition is caused by a genetic defect in the glycoprotein **fibrillin-1** and results in abnormalities in the skeleton (eg, long extremities, scoliosis, pectus excavatum), eyes (eg, ectopia lentis), and cardiovascular system (eg, aortic root disease).

The 2 most common cardiac abnormalities seen in MFS patients are **mitral valve prolapse** and **cystic medial degeneration of the aorta**. In more than 75% of MFS patients, cystic medial degeneration of the aorta results in aneurysmal dilation. If untreated, this can cause **aortic dissection**, the most common cause of death in MFS patients. The second most common cause of death is cardiac failure secondary to mitral valve prolapse and/or aortic regurgitation. For these reasons, MFS patients should be followed regularly by a cardiologist.

(Choices B, D, and E) With careful cardiovascular management, patients with MFS can have a normal life expectancy. MFS patients are not at increased risk for any of these other conditions.

(Choice C) Although homocystinuria is also associated with a marfanoid body habitus, distinguishing features of homocystinuria include intellectual disability and increased risk of thrombotic events (eg, myocardial infarction, stroke). However, this patient has no intellectual dysfunction.

Educational objective:
Cardiovascular lesions are the most life-threatening complications associated with Marfan syndrome. Early-onset cystic medial degeneration of the aorta predisposes to aortic dissection, the most common cause of death in these patients.

Pathophysiology	Cardiovascular System	Marfan syndrome
Subject	System	Topic

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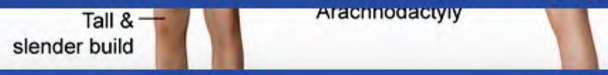

Exhibit Display

Differential diagnosis of Marfanoid body habitus		
Diagnosis	**Overlapping features**	**Distinguishing features**

Marfan syndrome	• Pectus deformity
	• Tall stature
	  ◦ ↑ Arm: height ratio
	  ◦ ↓ Upper: lower segment ratio
	• Arachnodactyly
	• Joint hyperlaxity
	• Skin hyperelasticity
	• Scoliosis
		• Autosomal dominant
		• Normal intellect
		• Aortic root dilation
		• Upward lens dislocation

Homocystinuria		• Autosomal recessive • Intellectual disability • Thrombosis • Downward lens dislocation • Megaloblastic anemia • Fair complexion

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A 71-year-old woman comes to the office for follow-up. She has a history of systolic heart failure and was hospitalized 2 weeks ago for an exacerbation, which was treated with intravenous diuretics. Since discharge the patient has been taking oral diuretics and says her shortness of breath has improved, but she is now experiencing muscle weakness and cramping. Blood pressure is 128/72 mm Hg and pulse is 80/min. Physical examination shows mildly decreased muscle strength in the lower extremities. Laboratory evaluation reveals a serum potassium level of 2.2 mEq/L. The patient receives potassium supplements, which improve the weakness. Another diuretic is added to her medical regimen to help prevent this adverse effect in the future. This additional medication predominantly acts on which of the following nephron segments?

- A. A
- B. B
- C. C
- D. D
- E. E
- F. F

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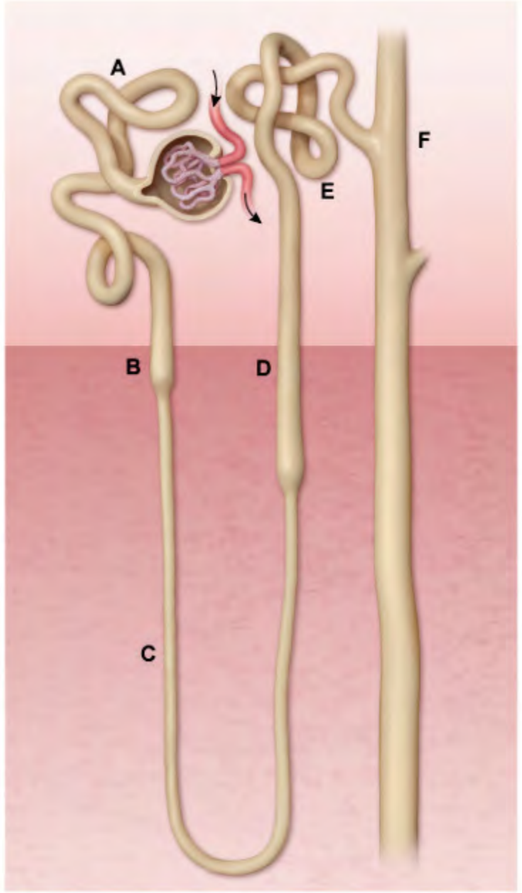

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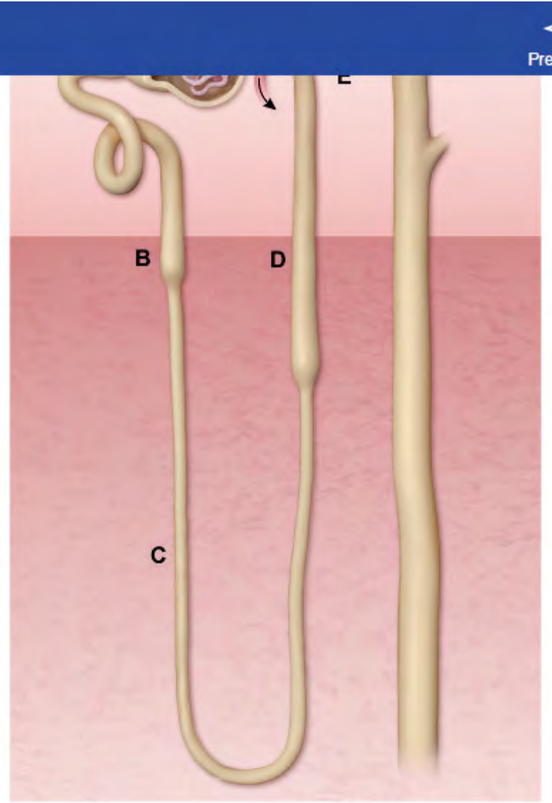

- A. A
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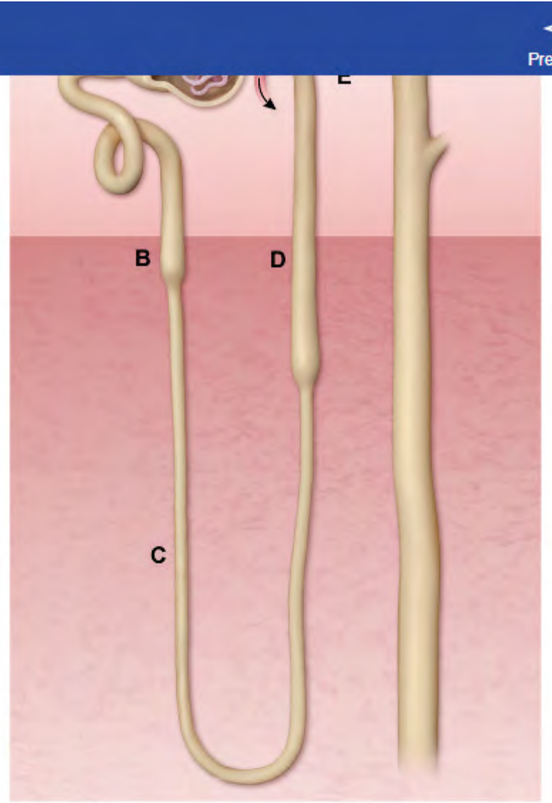

- A. A (1%)
- B. B (0%)
- ✗ C. C (1%)
- D. D (4%)
- E. E (10%)
- ✓ F. F (82%)

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Correct answer
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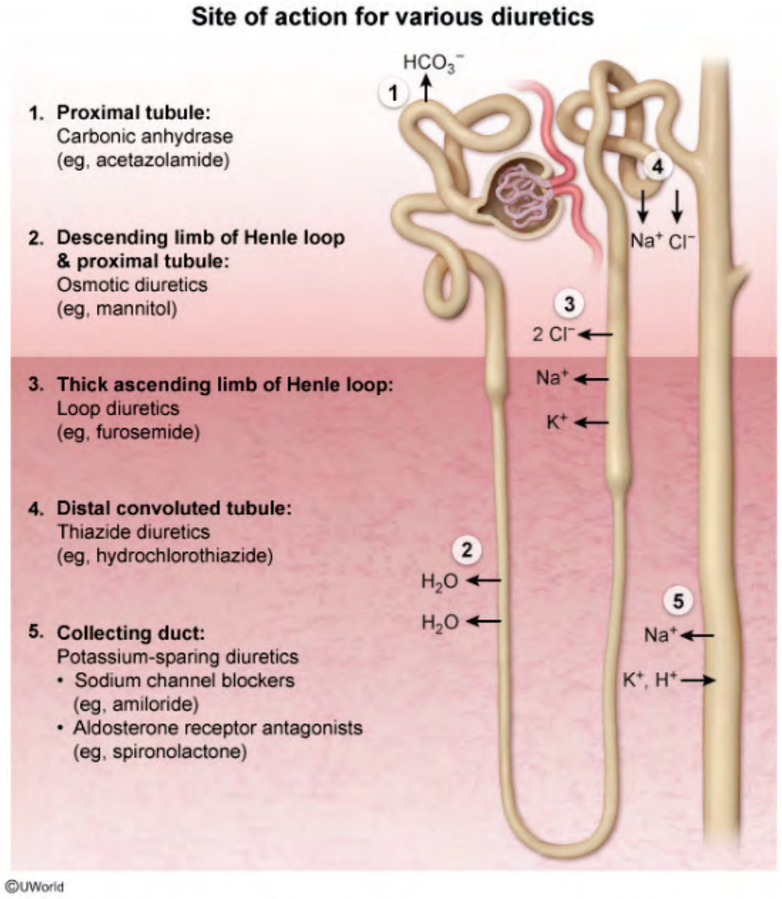

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reduce the symptoms of heart failure (eg, dyspnea, edema). The physician most likely added a **potassium-sparing diuretic** to this patient's treatment regimen to minimize potassium loss caused by loop diuretic use. All

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This patient has symptomatic hypokalemia, likely due to treatment with loop diuretics (eg, furosemide) used to reduce the symptoms of heart failure (eg, dyspnea, edema). The physician most likely added a **potassium-sparing diuretic** to this patient's treatment regimen to minimize potassium loss caused by loop diuretic use. All diuretics except for the potassium-sparing class cause potassium loss by increasing sodium delivery to the **late distal tubule** and **cortical collecting duct**, where aldosterone-induced sodium reabsorption occurs at the expense of potassium. Potassium-sparing diuretics act primarily by antagonizing this effect of aldosterone.

Some potassium-sparing diuretics, such as amiloride and triamterene, function by directly blocking the epithelial sodium channels (ENaCs) in the apical membrane of principal cells. Others, including spironolactone and eplerenone, competitively inhibit the aldosterone receptor, causing downregulation of both the apical ENaCs and basolateral Na^+/K^+ pumps. Spironolactone and eplerenone (a more specific receptor antagonist than spironolactone with fewer side effects) are preferred in patients with heart failure because they can also prevent the deleterious effects of **aldosterone-induced cardiac remodeling**.

(Choice A) Carbonic anhydrase inhibitors (eg, acetazolamide, dorzolamide) block sodium and bicarbonate reabsorption in the proximal convoluted tubule. These weak diuretics are typically used to treat altitude sickness or glaucoma.

(Choices B and C) Osmotic diuretics (eg, mannitol) function by preventing sodium and water reabsorption, mainly in the proximal tubule and the descending limb of the loop of Henle. These are typically used for cerebral edema with increased intracranial pressure.

(Choice D) Loop diuretics (eg, furosemide, bumetanide, torsemide) inhibit the Na-K-2Cl symporter in the thick ascending limb of the loop of Henle. This patient is likely already receiving this medication for symptomatic treatment of her heart failure; further dosage increase would worsen hypokalemia.

(Choice E) Thiazide diuretics (eg, hydrochlorothiazide, chlorthalidone) act in the early distal convoluted tubule. These drugs are commonly used in the treatment of hypertension and are sometimes added to loop diuretics in patients with refractory edema, but they would likely worsen hypokalemia.

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Educational objective:
All diuretics except for the potassium-sparing class cause potassium loss by increasing sodium delivery to the late distal tubule and cortical collecting duct, where aldosterone-induced sodium reabsorption occurs at the expense of potassium. Potassium-sparing diuretics (eg, spironolactone, amiloride) act on the late distal tubule and cortical collecting duct to antagonize the effects of aldosterone.

Pharmacology Cardiovascular System Aldosterone antagonists

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A 23-year-old man comes to the emergency department with sudden onset of heart palpitations that started while he was at his desk at work. The patient has no known medical problems and does not use tobacco or illicit drugs. He drinks alcohol occasionally on the weekends. Initial blood pressure is 110/70 mm Hg and pulse is 160/min and regular. Gentle neck massage just below the angle of the right mandible provides immediate improvement of his condition. His blood pressure is now 120/80 mm Hg and pulse is 75/min. Which of the following mechanisms is responsible for improvement of this patient's condition?

- A. Decreased baroreceptor firing rate
- B. Increased sympathetic output to the sinoatrial node
- C. Increased systemic vascular resistance
- D. Prolonged atrioventricular node refractory period
- E. Prolonged ventricular myocardium refractory period

Submit

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A 23-year-old man comes to the emergency department with sudden onset of heart palpitations that started while he was at his desk at work. The patient has no known medical problems and does not use tobacco or illicit drugs. He drinks alcohol occasionally on the weekends. Initial blood pressure is 110/70 mm Hg and pulse is 160/min and regular. Gentle neck massage just below the angle of the right mandible provides immediate improvement of his condition. His blood pressure is now 120/80 mm Hg and pulse is 75/min. Which of the following mechanisms is responsible for improvement of this patient's condition?

- ✗ A. Decreased baroreceptor firing rate (34%)
- B. Increased sympathetic output to the sinoatrial node (5%)
- C. Increased systemic vascular resistance (4%)
- ✓ D. Prolonged atrioventricular node refractory period (53%)
- E. Prolonged ventricular myocardium refractory period (1%)

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Correct answer
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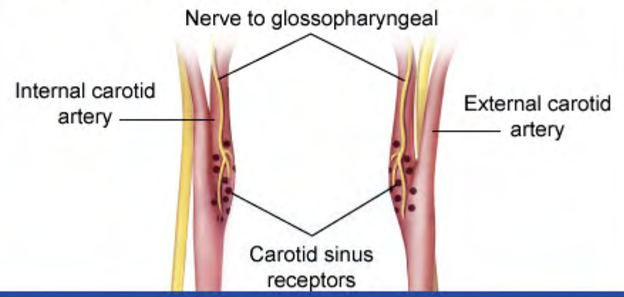
Explanation

Carotid sinus & aortic arch baroreceptors



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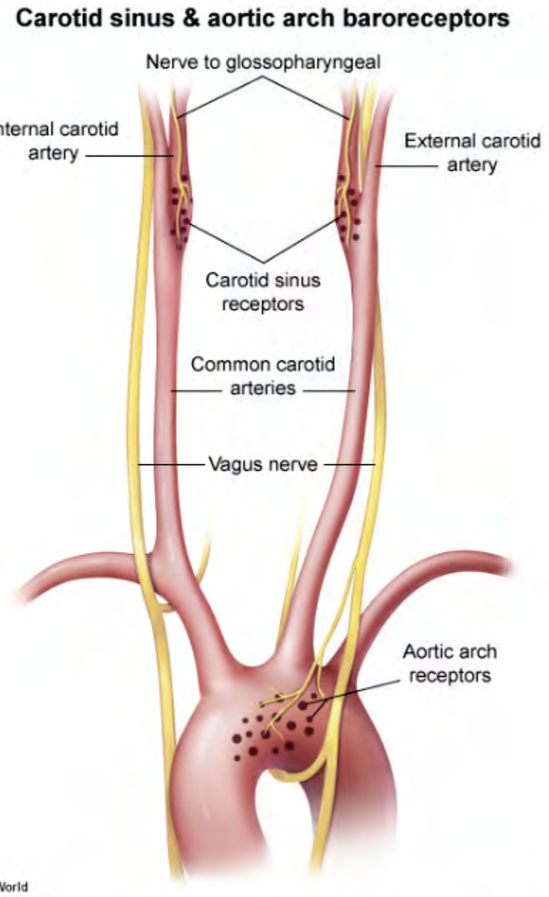

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Carotid sinus receptors

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This patient's presentation with sudden onset of palpitations and rapid regular tachycardia is consistent with **paroxysmal supraventricular tachycardia** (PSVT), which is most often due to a reentrant impulse traveling circularly between the slowly and rapidly conducting segments of the atrioventricular (AV) node. **Vagal maneuvers** such as carotid sinus massage, Valsalva, and cold water immersion can be used to acutely terminate PSVT.

The carotid sinuses are bulges in the internal carotid artery with baroreceptors located immediately distal to the bifurcation of the common carotid artery. The carotid sinus reflex has an afferent limb that arises from the baroreceptors in the sinus and travels to the vagal nucleus and medullary centers via the glossopharyngeal nerve (cranial nerve [CN] IX), whereas the efferent limb carries parasympathetic impulses to the sinoatrial (SA) and AV nodes via the vagus nerve (CN X). Carotid sinus massage leads to increased afferent firing from the carotid sinus, which in turn, **increases vagal parasympathetic tone**. This **slows conduction through the AV node** and **prolongs** the AV node **refractory period**, helping to terminate the reentrant tachycardia.

(Choices A, B, and C) Carotid sinus massage stimulates the baroreceptors and increases the firing rate from the carotid sinus, leading to an increase in parasympathetic output and withdrawal of sympathetic output to the heart and peripheral vasculature. This leads to slowing of the heart rate and AV conduction, along with a decrease in systemic vascular resistance.

(Choice E) Vagal efferent nerves only sparsely innervate ventricular myocardium, and carotid sinus massage does not cause any significant change in the ventricular refractory period.

Educational objective:
Carotid sinus massage leads to an increase in parasympathetic tone causing temporary inhibition of sinoatrial node activity, slowing of conduction through the atrioventricular (AV) node, and prolongation of the AV node refractory period. It is a useful vagal maneuver for termination of paroxysmal supraventricular tachycardia.

References

- Vagal techniques for termination of paroxysmal supraventricular tachycardia.

Physiology	Cardiovascular System	Supraventricular tachycardia
Subject	System	Topic

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A 72-year-old man comes to the emergency department due to sudden-onset, severe abdominal pain accompanied by nausea. He has a history of coronary artery disease and recently underwent cardiac angiography. The patient is a current smoker with a 30-pack-year history. After initial evaluation, he undergoes emergency laparotomy. A segment of small intestine is resected. On microscopic examination, there are several large-caliber vessels with needle-shaped cholesterol clefts in their lumens, as shown in the image below.

Given these findings, the patient is at highest risk for which of the following complications?

- A. Kidney injury
- B. Myelosuppression
- C. Nephrolithiasis
- D. Pulmonary infarction
- E. Septic arthritis

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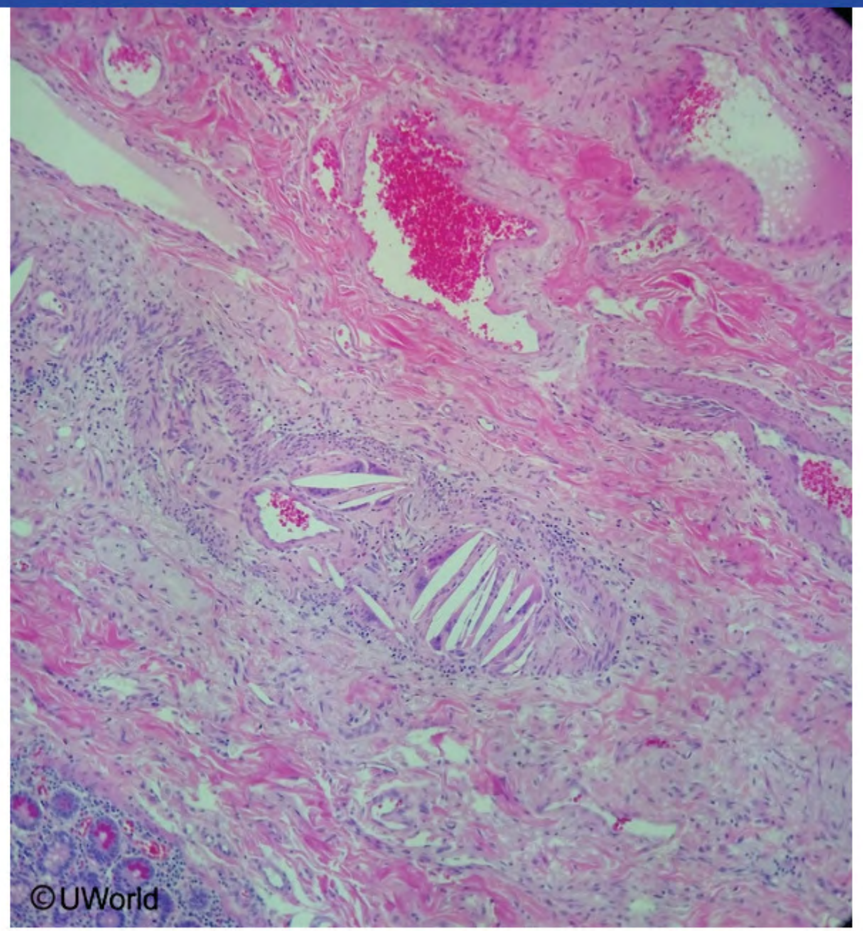

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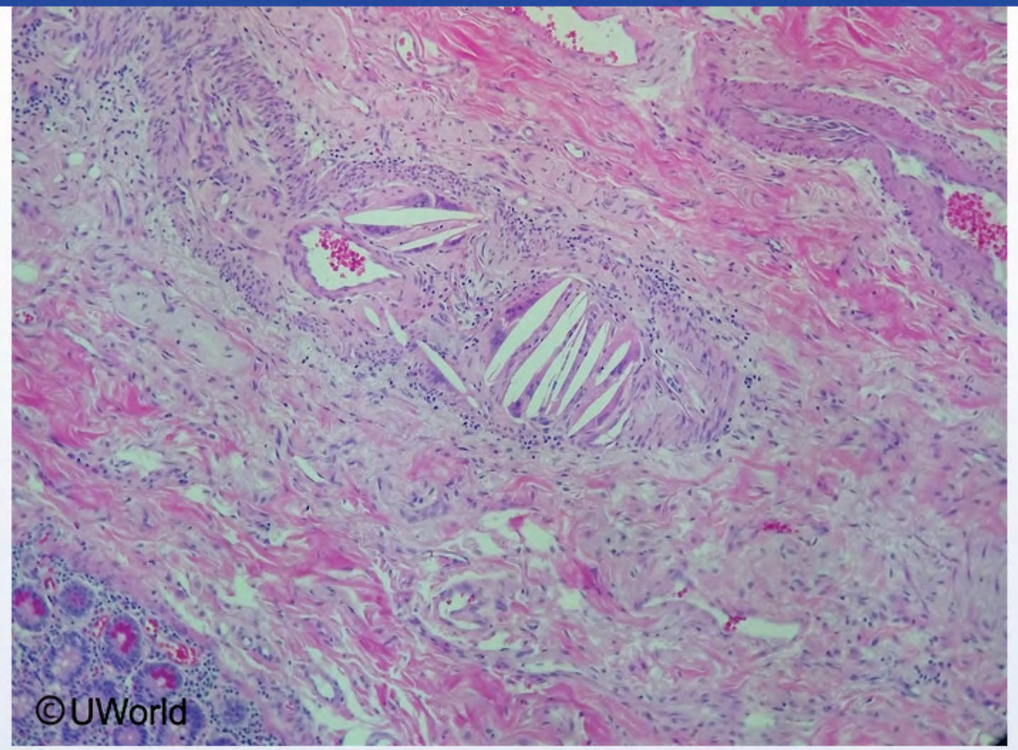

Given these findings, the patient is at highest risk for which of the following complications?

- A. Kidney injury
- B. Myelosuppression
- C. Nephrolithiasis
- D. Pulmonary infarction
- E. Septic arthritis

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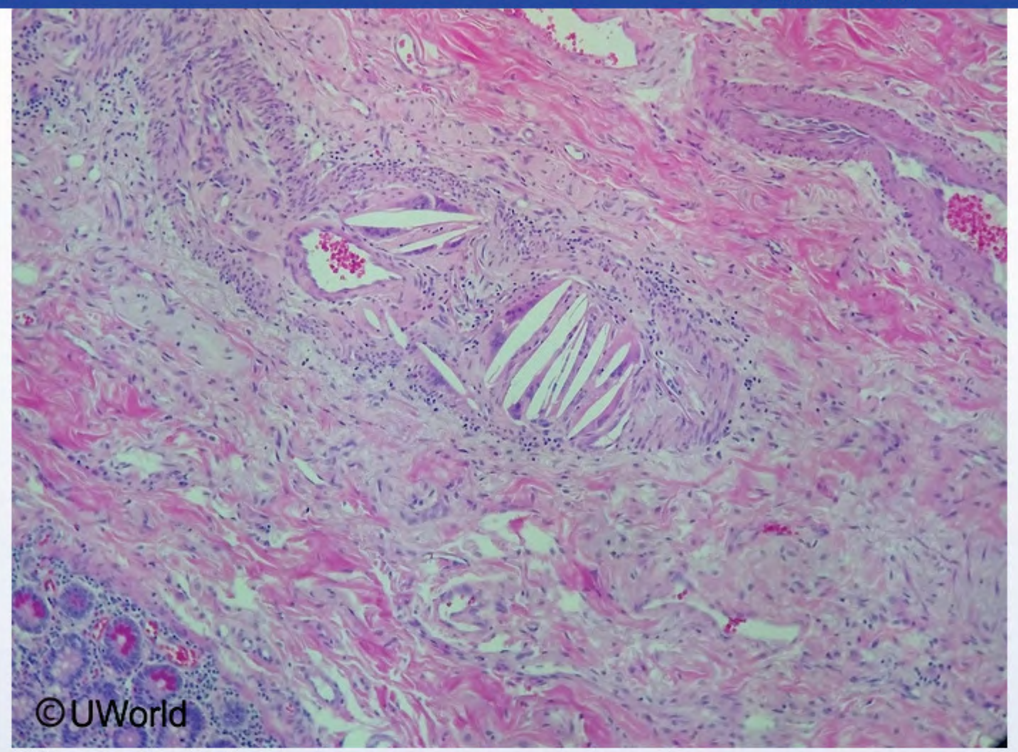

Given these findings, the patient is at highest risk for which of the following complications?

- ✓ A. Kidney injury (65%)
- ✗ B. Myelosuppression (0%)
- C. Nephrolithiasis (9%)
- D. Pulmonary infarction (23%)
- E. Septic arthritis (1%)

Incorrect
Correct answer
A

65%
Answered correctly

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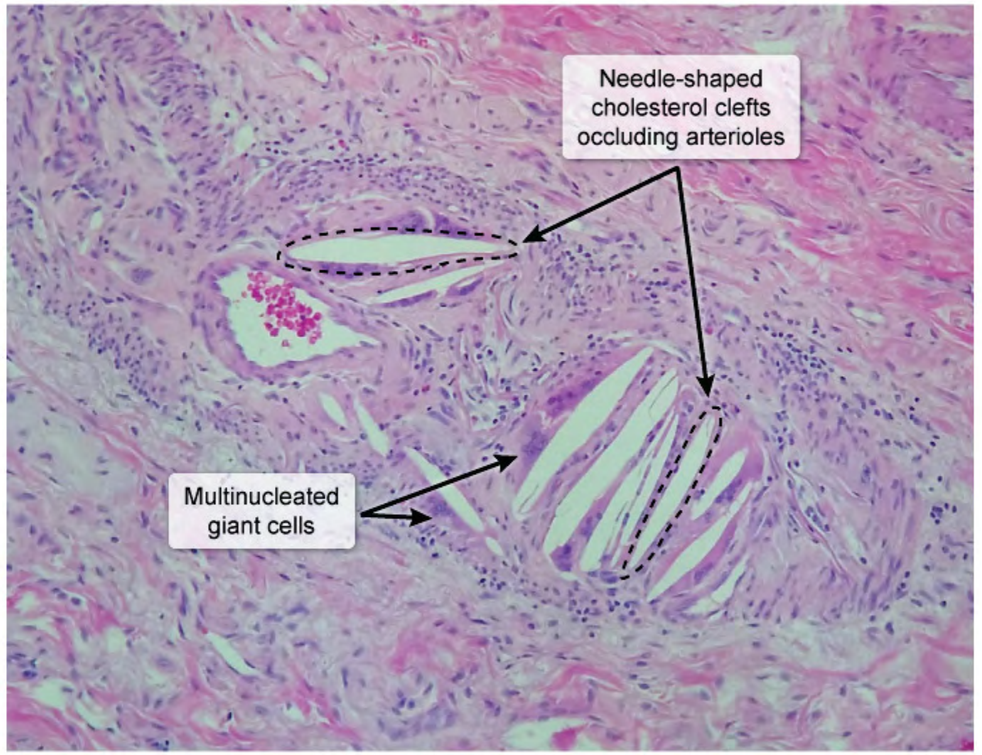
Explanation

Exhibit Display

Atheroembolization



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mechanically dislodged. This leads to showering of cholesterol-rich microemboli into the circulation with resultant

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This patient developed acute abdominal pain after recent coronary angiography. Small bowel histology demonstrates **needle-shaped intravascular cholesterol clefts**, a characteristic finding in **atheroembolic disease** (ie, cholesterol crystal embolism). Cholesterol is dissolved during tissue processing, leaving empty clefts on histologic evaluation. Atheroembolic disease typically occurs after an **invasive vascular procedure** (eg, coronary angiography, aortic surgery) in which atherosclerotic plaques on large vessels (eg, aorta) are mechanically dislodged. This leads to showering of cholesterol-rich microemboli into the circulation with resultant obstruction of distal arterioles and tissue ischemia.

Clinical manifestations depend on the location of the embolic scatter. Patients frequently **develop acute kidney injury** due to partial or complete occlusion of the arcuate or interlobular arteries. Other commonly involved organ systems include the skin (eg, blue toe syndrome, livedo reticularis), the gastrointestinal tract (eg, bleeding; intestinal infarction, as in this patient), and the CNS (eg, stroke, amaurosis fugax).

(Choice B) Heparin anticoagulants given during angiography can cause heparin-induced thrombocytopenia (HIT), which typically presents with low platelet counts and thrombotic complications. However, the destruction of platelets in HIT is due to immune-mediated platelet aggregation, rather than bone marrow suppression.

(Choice C) Gout causes needle-shaped crystals seen on microscopy. However, uric acid crystals are negatively birefringent (not empty clefts) and are located within tophi or synovial fluid. In addition, gout does not cause embolic disease.

(Choice D) Pulmonary infarction can also occur in atheroembolic disease; however, this is an extremely rare manifestation and occurs much less commonly than acute kidney injury.

(Choice E) Infectious endocarditis also causes embolic showering and can cause both small bowel involvement and septic arthritis. However, endocarditis is more common in drug users, and patients typically develop fever, arthralgias, and night sweats. In addition, bacteria, not cholesterol clefts, would be visualized on microscopic examination.

Educational objective:

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coronary angiography, aortic surgery) in which atherosclerotic plaques on large vessels (eg, aorta) are mechanically dislodged. This leads to showering of cholesterol-rich microemboli into the circulation with resultant obstruction of distal arterioles and tissue ischemia.

Clinical manifestations depend on the location of the embolic scatter. Patients frequently **develop acute kidney injury** due to partial or complete occlusion of the arcuate or interlobular arteries. Other commonly involved organ systems include the skin (eg, blue toe syndrome, livedo reticularis), the gastrointestinal tract (eg, bleeding; intestinal infarction, as in this patient), and the CNS (eg, stroke, amaurosis fugax).

(Choice B) Heparin anticoagulants given during angiography can cause heparin-induced thrombocytopenia (HIT), which typically presents with low platelet counts and thrombotic complications. However, the destruction of platelets in HIT is due to immune-mediated platelet aggregation, rather than bone marrow suppression.

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(Choice E) Infectious endocarditis also causes embolic showering and can cause both small bowel involvement and septic arthritis. However, endocarditis is more common in drug users, and patients typically develop fever, arthralgias, and night sweats. In addition, bacteria, not cholesterol clefts, would be visualized on microscopic examination.

Educational objective:
Atheroembolic disease typically occurs after an invasive vascular procedure due to mechanical dislodgement of atherosclerotic plaque, resulting in the showering of cholesterol-rich microemboli into the circulation. Needle-shaped cholesterol clefts in affected vessels are diagnostic. Commonly involved organs include the kidneys (eg, acute kidney injury), skin (eg, blue toe syndrome, livedo reticularis), gastrointestinal tract (eg, bleeding, infarction), and CNS (eg, stroke, amaurosis fugax).

Pathophysiology Cardiovascular System Atheroembolism

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A 56-year-old woman comes to clinic for follow-up of heart failure with reduced ejection fraction. Over the last 3 months, the patient has been hospitalized twice due to acute decompensation of heart failure that required treatment with intravenous furosemide. Since her most recent discharge, she has been taking high doses of oral furosemide, but she continues to have progressive lower extremity edema and weight gain. Serum creatinine is 1.2 mg/dL and serum potassium is 4.1 mEq/L. The patient is started on metolazone. The addition of metolazone is likely to assist in treating this patient because of which of the following effects of furosemide?

- A. Increased calcium delivery to the distal tubules
- B. Increased sodium delivery to the distal tubules
- C. Increased tubular reabsorption of bicarbonate
- D. Increased tubular reabsorption of uric acid
- E. Reduced tubular reabsorption of glucose
- F. Reduced tubular reabsorption of potassium

Submit

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- A. Increased calcium delivery to the distal tubules (9%)
- ✓ B. Increased sodium delivery to the distal tubules (64%)
- C. Increased tubular reabsorption of bicarbonate (3%)
- D. Increased tubular reabsorption of uric acid (2%)
- E. Reduced tubular reabsorption of glucose (3%)
- F. Reduced tubular reabsorption of potassium (17%)

Correct

64% Answered correctly

03 secs Time Spent

2023 Version

Explanation

Diuretic effects on total body electrolyte levels

Diuretic type	Na^+	K^+	HCO_3^-	Ca^{2+}	Uric acid
Loop (eg, furosemide)	↓↓↓	↓↓	↑↑	↓	↑
Thiazide					

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Diuretic effects on total body electrolyte levels					
Diuretic type	**Na^+**	**K^+**	**HCO_3^-**	**Ca^{2+}**	**Uric acid**
Loop (eg, furosemide)	↓↓↓	↓↓	↑↑	↓	↑
Thiazide (eg, HCTZ, metolazone)	↓↓	↓	↑	↑	↑
Potassium sparing (eg, spironolactone, amiloride)	↓	↑	↓	—	—
Carbonic anhydrase inhibitor (eg, acetazolamide)	↓	↓	↓	↓	—
HCTZ = hydrochlorothiazide.					

This patient has **decompensated heart failure** with ongoing volume retention despite high doses of oral furosemide (ie, loop diuretic) therapy. **Thiazide diuretics**, especially metolazone, **potentiate the diuretic effect of loop diuretics** and can be helpful in treating refractory volume overload.

Sodium is the major determinant of **volume status**. Loop diuretics function by inhibiting the Na-K-2Cl cotransporter in the ascending limb of the loop of Henle to block sodium absorption and encourage the excretion of sodium and water in the urine. However, the sodium excretion caused by loop diuretics is limited by the reabsorption of sodium in the distal convoluted tubule; the Na-Cl cotransporter in the distal tubule counteracts some of the effect of loop diuretics by absorbing much of the sodium that is not absorbed in the loop of Henle. **Inhibition of the Na-Cl cotransporter** with metolazone **prevents reabsorption** of the **increased sodium delivered to the distal tubule**, significantly increasing total sodium excretion.

(Choice A) Loop diuretics inhibit calcium absorption in the loop of Henle, leading to increased calcium delivery to

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Sodium is the major determinant of **volume status**. Loop diuretics function by inhibiting the Na-K-2Cl cotransporter in the ascending limb of the loop of Henle to block sodium absorption and encourage the excretion of sodium and water in the urine. However, the sodium excretion caused by loop diuretics is limited by the reabsorption of sodium in the distal convoluted tubule; the Na-Cl cotransporter in the distal tubule counteracts some of the effect of loop diuretics by absorbing much of the sodium that is not absorbed in the loop of Henle. **Inhibition of the Na-Cl cotransporter** with metolazone **prevents reabsorption** of the **increased sodium delivered to the distal tubule**, significantly increasing total sodium excretion.

(Choice A) Loop diuretics inhibit calcium absorption in the loop of Henle, leading to increased calcium delivery to the distal tubule. However, calcium has minimal effect on volume status. Thiazide diuretics increase calcium absorption in the distal tubule, making up for some of the calcium loss caused by loop diuretics.

(Choice C) Loop diuretics stimulate hydrogen ion excretion and bicarbonate reabsorption, creating a tendency toward metabolic alkalosis. Carbonic anhydrase inhibitors (eg, acetazolamide) block bicarbonate absorption in the proximal tubule and are sometimes used to minimize the metabolic alkalosis caused by loop diuretics.

(Choice D) Both loop and thiazide diuretics increase the reabsorption of uric acid and, therefore, may increase the risk of gout attacks.

(Choice E) Both loop and thiazide diuretics have minimal effect on tubular glucose reabsorption. Sodium-glucose cotransporter-2 inhibitors (eg, empagliflozin) block glucose resorption in the proximal tubule to lower serum glucose levels while also stimulating an osmotic diuresis.

(Choice F) The increased distal tubular delivery of sodium caused by loop diuretics stimulates increased potassium secretion by the principal cells in the late distal tubule and collecting duct. This potassium loss is amplified with the addition of a thiazide diuretic. Potassium-sparing diuretics (eg, spironolactone) can help offset the potassium loss caused by loop and thiazide diuretics.

Educational objective:
Thiazide diuretics, especially metolazone, potentiate the diuretic effect of loop diuretics (eg, furosemide) by blocking the reabsorption of the increased distal tubular sodium delivery caused by loop diuretics.

References

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Potassium sparing					

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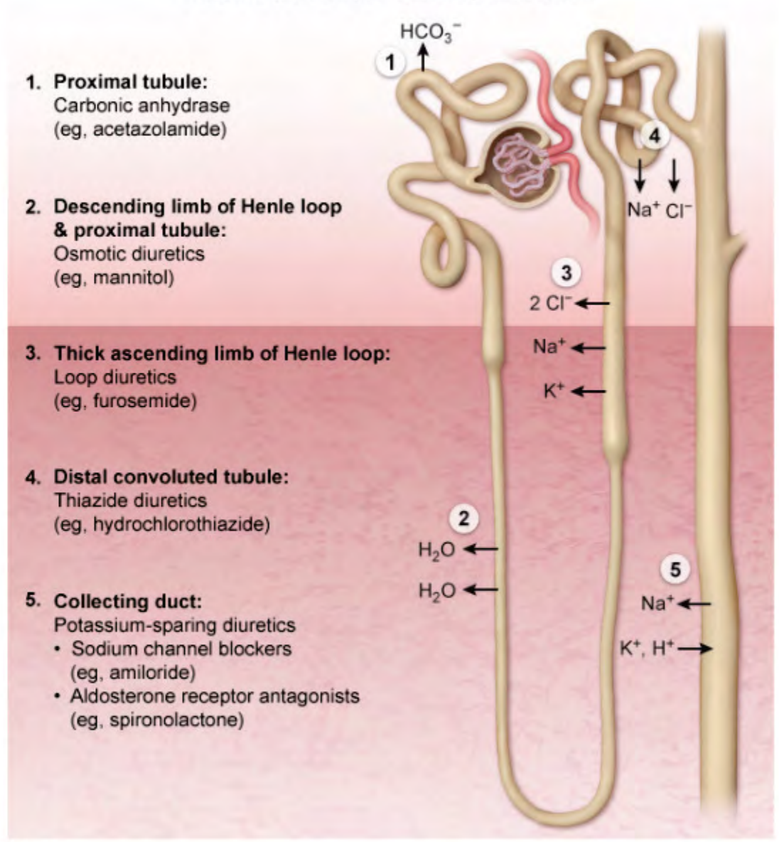
Site of action for various diuretics



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cotransporter-2 inhibitors (eg, empagliflozin) block glucose resorption in the proximal tubule to lower serum

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A 71-year-old man comes to the hospital due to 3 hours of persistent retrosternal chest pain and dyspnea. After prompt evaluation, he is diagnosed with acute myocardial infarction and undergoes cardiac catheterization. During cannulation of the right common femoral artery, the arterial wall is penetrated superior to the right inguinal ligament. Percutaneous coronary intervention is then performed, after which firm pressure is applied to achieve hemostasis. Soon after the procedure, the patient becomes cold, clammy, and hypotensive. Physical examination shows a 5cm region of ecchymosis surrounding the femoral puncture site. Internal bleeding is suspected. Which of the following is the most likely location of the blood collection?

- A. Pelvic cavity
- B. Retroperitoneal space
- C. Right paracolic gutter
- D. Subcutaneous tissue
- E. Thigh muscles

Submit

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- A. Pelvic cavity (36%)
- ✓ B. Retroperitoneal space (27%)
- C. Right paracolic gutter (16%)
- D. Subcutaneous tissue (10%)
- E. Thigh muscles (8%)

Correct

27% Answered correctly

03 secs Time Spent

2023 Version

Explanation

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A 71-year-old man comes to the hospital due to 3 hours of persistent retrosternal chest pain and dyspnea. After
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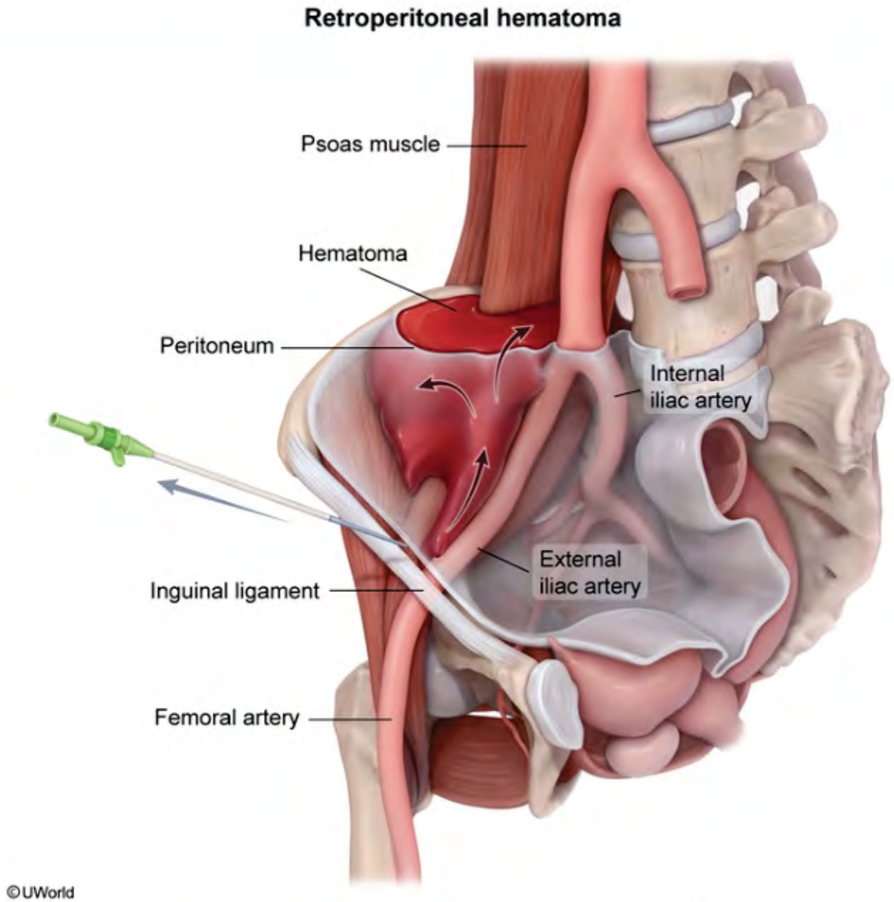

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Hematoma

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Vascular access during **cardiac catheterization** is typically obtained through either the **common femoral artery** or radial artery. The common femoral artery is the continuation of the external iliac artery as it crosses the inguinal ligament. Arterial **puncture above the inguinal ligament** increases the risk of **retroperitoneal hemorrhage**, as this portion of the vessel lies directly inferior to the peritoneum. Accidental puncture of the posterior wall can cause blood to track along the loose connective tissue surrounding the vessel and accumulate within the interfascial planes of the retroperitoneum.

Bleeding in the retroperitoneal space **cannot be controlled** with external compression and can lead to life-threatening hemorrhage. Affected patients typically develop **hemodynamic instability** with significant hypotension, a drop in hemoglobin, and ipsilateral flank pain.

(Choice A) Bleeding into the pelvic cavity is frequently due to trauma (eg, pelvic fracture) or gynecologic hemorrhage (eg, ruptured ectopic pregnancy). Injury to the common femoral artery is less likely to cause intraperitoneal bleeding as the external iliac artery courses underneath the peritoneum.

(Choice C) The right paracolic gutter is a peritoneal recess found between the ascending colon and abdominal wall. Fluid (bile, pus, or blood) typically accumulates there from pathology involving the gastrointestinal organs, particularly the appendix and gall bladder.

(Choice D) Subcutaneous tissue hemorrhage from an arterial source typically presents with a large area of ecchymosis and a palpable hematoma. Development of significant hypotension is less likely with subcutaneous bleeding, as the potential space for blood accumulation is smaller than with peritoneal or retroperitoneal bleeding.

(Choice E) Hemorrhage into the thigh muscles would present with significant pain and edema in the thigh and possibly lead to compartment syndrome (severe pain, absent distal pulses).

Educational objective:
The optimal site for obtaining vascular access in the lower extremity during cardiac catheterization is the common femoral artery below the inguinal ligament. Cannulation above the inguinal ligament can significantly increase the risk of retroperitoneal hemorrhage.

References

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A 24-year-old woman participates in a study of changes in blood pressure during aerobic exercise. She is healthy and takes no medications. During the study she is asked to run on a treadmill at incrementally increasing speed until her heart rate reaches 90% of her expected maximum heart rate. Compared to at rest, which of the following changes in blood pressure are expected during this time?

	Systolic blood pressure	Diastolic blood pressure	Pulse pressure
A.	Decreased	Decreased	Unchanged
B.	Unchanged	Increased	Decreased
C.	Increased	Unchanged	Increased
D.	Increased	Increased	Unchanged
E.	Increased	Increased	Increased

Submit

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	Systolic blood pressure	Diastolic blood pressure	Pulse pressure	
✗ A.	Decreased	Decreased	Unchanged	(5%)
B.	Unchanged	Increased	Decreased	(2%)
✓ C.	Increased	Unchanged	Increased	(37%)
D.	Increased	Increased	Unchanged	(30%)
E.	Increased	Increased	Increased	(25%)

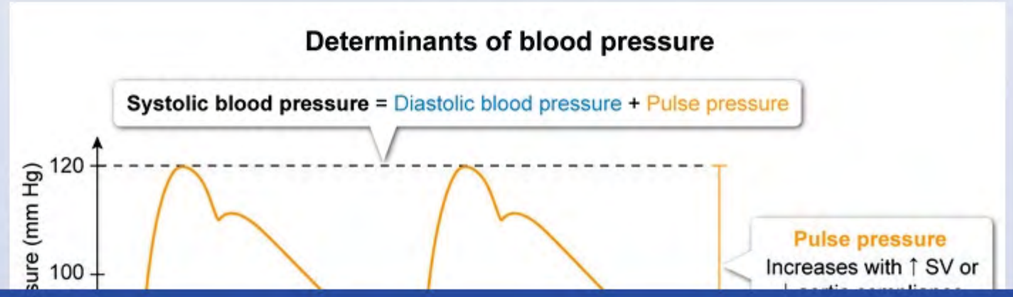
Incorrect
Correct answer
C

37%
Answered correctly

01 min, 50 secs
Time Spent

2023
Version

Explanation



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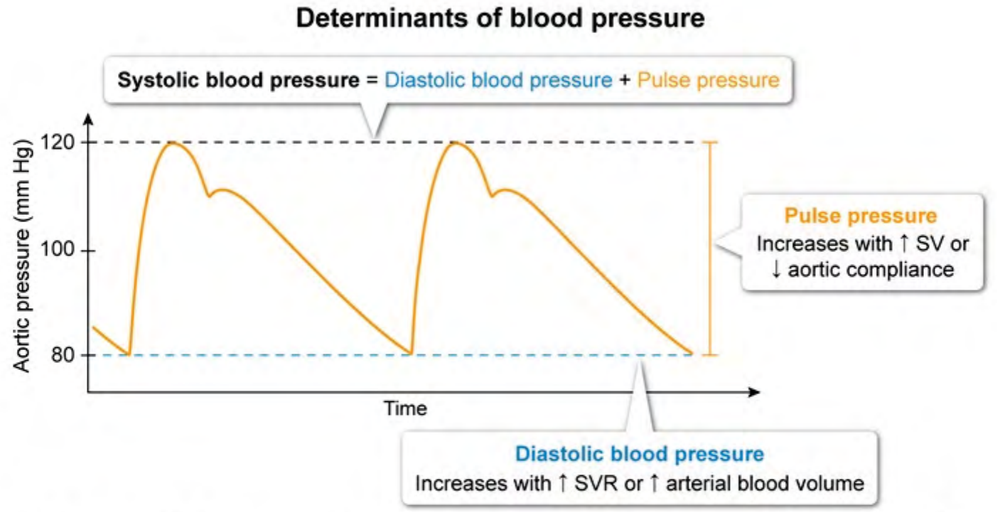

SV = stroke volume; **SVR** = systemic vascular resistance.

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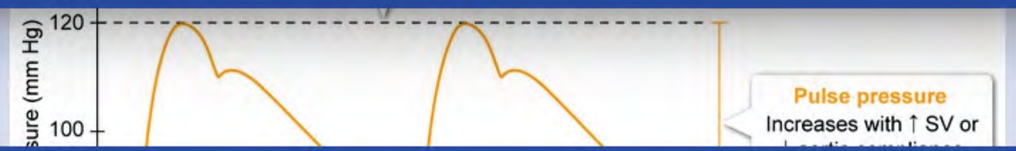

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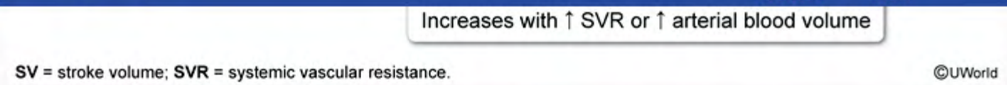

SV = stroke volume; **SVR** = systemic vascular resistance. ©UWorld

Diastolic blood pressure (DBP) is the baseline hydrostatic pressure in the arterial system and is **directly related to systemic vascular resistance** (SVR) and arterial blood volume. **Pulse pressure** (PP) is the amount that arterial pressure increases above DBP during left ventricular contraction; it is **directly related to stroke volume** and inversely related to aortic compliance (which is generally constant but decreases with advancing age). Systolic blood pressure (SBP) is the summation of DBP and PP.

During **aerobic exercise**, there is vasoconstriction of splanchnic arterioles and a larger degree of vasodilation of skeletal muscle arterioles, which shunts blood to exercising muscle and leads to overall **reduced SVR**. In addition, venoconstriction of capacitance veins mobilizes stored blood to increase venous return (ie, **increased preload**), and **myocardial contractility** is also increased. Both the increased preload and increased myocardial contractility **increase stroke volume**, which in combination with increased heart rate, markedly increases cardiac output.

The reduced SVR during aerobic exercise facilitates decreased DBP but it is somewhat offset by mobilization of a higher percentage of blood volume from the venous to the arterial circulation (due to venoconstriction and increased heart rate), resulting in **slightly decreased or unchanged DBP**. The increased stroke volume leads to **increased PP**, and consequently **increased SBP**.

(Choice A) Decreased DBP and unchanged PP results in decreased SBP. Such changes are expected with reduced SVR and no change in stroke volume.

(Choice B) Increased DBP and decreased PP can result in unchanged SBP. Such changes are expected with increased SVR and decreased stroke volume.

(Choice D) Increased DBP and unchanged PP results in increased SBP. Such changes are expected with increased SVR and no change in stroke volume.

(Choice E) Increased DBP and increased PP results in greatly increased SBP. Such changes occur with resistance exercise (eg, heavy weightlifting) as SVR is increased due to arteriolar compression by vigorously contracting muscles, and stroke volume is increased due to increased venous return and myocardial contractility.

Educational objective:

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Systolic blood pressure (SBP) is the summation of DBP and PP.

During **aerobic exercise**, there is vasoconstriction of splanchnic arterioles and a larger degree of vasodilation of skeletal muscle arterioles, which shunts blood to exercising muscle and leads to overall **reduced SVR**. In addition, venoconstriction of capacitance veins mobilizes stored blood to increase venous return (ie, **increased preload**), and **myocardial contractility** is also increased. Both the increased preload and increased myocardial contractility **increase stroke volume**, which in combination with increased heart rate, markedly increases cardiac output.

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(Choice E) Increased DBP and increased PP results in greatly increased SBP. Such changes occur with resistance exercise (eg, heavy weightlifting) as SVR is increased due to arteriolar compression by vigorously contracting muscles, and stroke volume is increased due to increased venous return and myocardial contractility.

Educational objective:
Diastolic blood pressure (DBP) is largely determined by systemic vascular resistance (SVR), and pulse pressure is largely determined by stroke volume. During aerobic exercise, reduced SVR leads to slightly decreased or unchanged DBP, and increased stroke volume leads to increased pulse pressure and systolic blood pressure.

References

- Physiology, pulse pressure.

Physiology Cardiovascular System Physical exercise

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A 66-year-old man comes to the emergency department due to several episodes of lightheadedness and a recent fall. The symptoms are especially pronounced in the morning when he gets up from bed. The patient takes medications for hypertension, stable angina pectoris, gout, and benign prostate hyperplasia. He also has osteoarthritis of the right knee and takes occasional nonsteroidal anti-inflammatory drugs. His blood pressure is 120/80 mm Hg and pulse is 70/min in the supine position, and 90/60 mm Hg and 87/min on standing, respectively. Cardiopulmonary examination is normal. Blockade of which of the following receptors is most likely contributing to this patient's current condition?

- A. α1-adrenergic
- B. α2-adrenergic
- C. β1-adrenergic
- D. β2-adrenergic
- E. D1-dopaminergic

Submit

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- ✓ A. α1-adrenergic (82%)
- B. α2-adrenergic (4%)
- C. β1-adrenergic (8%)
- D. β2-adrenergic (4%)
- E. D1-dopaminergic (0%)

Correct

82% Answered correctly

04 secs Time Spent

2023 Version

Explanation

Cardiovascular effects of adrenergic drugs				
Drug class	**Examples**	**Heart rate**	**Cardiac contractility**	**Systemic vascular resistance**
α1 agonist	• Midodrine • Phenylephrine	↓ (reflex)	↓ (reflex)	↑
		↓ (via central		

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Cardiovascular effects of adrenergic drugs				
Drug class	**Examples**	**Heart rate**	**Cardiac contractility**	**Systemic vascular resistance**
α1 agonist	• Midodrine • Phenylephrine	↓ (reflex)	↓ (reflex)	↑
α2 agonist	• Clonidine	↓ (via central receptors)		↓ (via central receptors)
α1 antagonist	• Doxazosin • Terazosin	↑ (reflex)		↓
α1/ α2 antagonist	• Phenoxybenzamine	↑ (reflex)		↓
β agonist	• Isoproterenol	↑	↑	↓ (via β2 agonist effect)
β1 antagonist	• Metoprolol • Atenolol	↓	↓	

This patient has **orthostatic (postural) hypotension**, which is characterized by a drop in blood pressure on **standing** from the supine position (≥20 mm Hg systolic and/or ≥10 mm Hg diastolic). Standing causes pooling of a significant amount of blood (500-1000 mL) into the veins below the heart. This initiates the following sequence of events in otherwise healthy individuals:

1. Decreased venous return to the heart
2. Decreased ventricular filling and subsequent drop in cardiac output
3. Drop in blood pressure that evokes compensatory baroreceptor reflex
4. Increased sympathetic tone that increases peripheral vascular resistance (α1-adrenoreceptors) as well as heart rate and myocardial contractility (β1-adrenoreceptors)

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1. Decreased venous return to the heart
2. Decreased ventricular filling and subsequent drop in cardiac output
3. Drop in blood pressure that evokes compensatory baroreceptor reflex
4. Increased sympathetic tone that increases peripheral vascular resistance (α1-adrenoreceptors) as well as heart rate and myocardial contractility (β1-adrenoreceptors)

The **α1-adrenergic antagonists** (terazosin, doxazosin) are commonly used for symptomatic benign prostate hyperplasia and are often associated with orthostatic hypotension, resulting in **lightheadedness** and **syncope** due to cerebral hypoperfusion upon standing. Other etiologies of orthostatic hypotension include volume depletion (hypovolemia due to hemorrhage, hyperglycemia, or diuretics) and autonomic dysfunction (Parkinson disease, diabetes).

(Choice B) Blockade of presynaptic α2-adrenergic receptors results in increased release of norepinephrine from peripheral nerve terminals. This effect results in increased blood pressure and heart rate.

(Choice C) The β1-adrenergic receptors are found predominantly in the heart. Their stimulation increases heart rate, contractility, and conduction. Blockade of β1-adrenoreceptors would prevent this patient's heart rate from increasing appropriately upon standing (normally about 10-25/min).

(Choice D) Stimulation of β2-adrenergic receptors causes bronchodilation, vasodilation in skeletal muscles, and uterine relaxation. Blockade of β2-adrenoreceptors is associated with peripheral vasoconstriction and bronchoconstriction and would not cause orthostatic hypotension.

(Choice E) D1-adrenergic (dopaminergic) receptors are located in the walls of renal, splanchnic, and mesenteric blood vessels. Stimulation of these receptors causes vasodilation and increased renal and splanchnic blood flow. Blockade of D1 receptors does not result in orthostatic hypotension.

Educational objective:
Orthostatic hypotension is a frequent cause of lightheadedness and syncope and is defined as a decrease in systolic (≥20 mm Hg) or diastolic (≥10 mm Hg) blood pressure on standing from the supine position. Medications (α1-adrenergic antagonists, diuretics), volume depletion, and autonomic dysfunction are common causes of orthostatic hypotension.

References

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A 54-year-old man comes to the emergency department with retrosternal chest pain of 30 minutes duration. The patient also complains of sweating and mild dyspnea. A single tablet of nitroglycerin is delivered sublingually, and the patient's pain decreases significantly. The patient has experienced several similar episodes of pain over the last 12 hours, all of which resolved spontaneously. Which of the following ultrastructural changes would most likely indicate irreversible myocardial cell injury in this patient?

- A. Myofibril relaxation
- B. Disaggregation of polysomes
- C. Mitochondrial vacuolization
- D. Disaggregation of nuclear granules
- E. Triglyceride droplet accumulation
- F. Glycogen loss

Submit

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- A. Myofibril relaxation (3%)
- ✗ B. Disaggregation of polysomes (3%)
- ✓ C. Mitochondrial vacuolization (59%)
- D. Disaggregation of nuclear granules (28%)
- E. Triglyceride droplet accumulation (2%)
- F. Glycogen loss (2%)

Incorrect
Correct answer
C

59%
Answered correctly

04 secs
Time Spent

2023
Version

Explanation

The appearance of vacuoles and phospholipid-containing amorphous densities within mitochondria generally signifies irreversible injury, and implies a permanent inability to generate further ATP via oxidative phosphorylation. When the mitochondria are injured irreversibly, the cell cannot recover. Simple mitochondrial swelling may be associated with reversible cellular injury, however.

(Choice A) Myofibril relaxation is an early sign of *reversible* injury in cardiac myocytes, which occurs within the first 30 minutes of severe ischemia. Myofibril relaxation corresponds with intracellular ATP depletion and lactate accumulation due to anaerobic glycolysis during this period.

(Choice B) Disaggregation of polysomes denotes the dissociation of rRNA from mRNA in *reversible*

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The appearance of vacuoles and phospholipid-containing amorphous densities within mitochondria generally signifies irreversible injury, and implies a permanent inability to generate further ATP via oxidative phosphorylation. When the mitochondria are injured irreversibly, the cell cannot recover. Simple mitochondrial swelling may be associated with reversible cellular injury, however.

(Choice A) Myofibril relaxation is an early sign of *reversible* injury in cardiac myocytes, which occurs within the first 30 minutes of severe ischemia. Myofibril relaxation corresponds with intracellular ATP depletion and lactate accumulation due to anaerobic glycolysis during this period.

(Choice B) Disaggregation of polysomes denotes the dissociation of rRNA from mRNA in *reversible* ischemic/hypoxic injury. Depletion of intracellular ATP is thought to promote the dissolution of polysomes into monosomes as well as the detachment of ribosomes from the rough endoplasmic reticulum. Disaggregation of polysomes results in impaired protein synthesis.

(Choice D) Disaggregation of granular and fibrillar elements of the nucleus is associated with *reversible* cell injury. Another common nuclear change associated with reversible cell injury is clumping of nuclear chromatin, perhaps secondary to a decrease in intracellular pH.

(Choice E) Triglyceride droplet accumulation is characteristic of *reversible* cell injury, especially in hepatocytes, and also in striated muscle cells and renal cells. This fatty change may result from the decreased synthesis of intracellular proteins that occurs with cell injury. In the hepatocyte, decreased production of lipid acceptor proteins prevents the normal incorporation of triglycerides into lipoproteins. Since triglycerides cannot be rapidly exported from the cell in the form of lipoproteins, they accumulate intracellularly.

(Choice F) Glycogen loss is another early and *reversible* cellular response to injury. As a result of lowered mitochondrial ATP production, ATP must be supplied to the cell via anaerobic glycolysis of glucose derived from the cell's glycogen stores. Myocardial glycogen stores may be completely depleted within 30 minutes of the onset of severe ischemia.

Educational Objective:
Mitochondrial vacuolization is typically a sign of irreversible cell injury, signifying that the involved mitochondria are permanently unable to generate ATP.

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In experiments, transient myocardial ischemia causes myocardial cells to increase in size. This effect is due in part to which of the following?

- A. Intracellular K^+ accumulation
- B. Intracellular Ca^{2+} accumulation
- C. High cellular HCO_3^- content
- D. Cascade protein phosphorylation
- E. Net cellular solute loss

Submit

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In experiments, transient myocardial ischemia causes myocardial cells to increase in size. This effect is due in part to which of the following?

- A. Intracellular K^+ accumulation (21%)
- ✓ B. Intracellular Ca^{2+} accumulation (54%)
- C. High cellular HCO_3^- content (5%)
- D. Cascade protein phosphorylation (11%)
- E. Net cellular solute loss (6%)

Correct

54% Answered correctly

04 secs Time Spent

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Explanation

When blood flow cannot meet myocardial demands, cardiac myocytes transition from aerobic to anaerobic metabolism. However, anaerobic metabolism cannot maintain proper intracellular ATP levels, and ADP, AMP and adenosine accumulate. Without ATP, the membrane Na^+/K^+-ATPases and the sarcoplasmic reticulum Ca^{2+}-ATPases fail, leading to increased intracellular Na^+ and Ca^{2+} and increased intramitochondrial Ca^{2+} concentrations. These increased concentrations attract free water, causing cellular and mitochondrial swelling. Failure of the sarcoplasmic reticulum to resequester Ca^{2+} leads to cessation of contraction within ischemic zones of myocardium.

(Choice A) The cell membrane Na^+/K^+-ATPase pump normally brings K^+ into the cell in exchange for Na+. Thus, in ischemic tissue, intracellular K^+ is decreased.

(Choice C) Cellular HCO_3^- is not elevated in cardiac ischemia. Anaerobic metabolism leads to lactic acid production and a decrease in pH. Tissue CO_2, the conjugate acid of HCO_3^-, is thus elevated in ischemic myocardium.

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Explanation

When blood flow cannot meet myocardial demands, cardiac myocytes transition from aerobic to anaerobic metabolism. However, anaerobic metabolism cannot maintain proper intracellular ATP levels, and ADP, AMP and adenosine accumulate. Without ATP, the membrane Na^+/K^+-ATPases and the sarcoplasmic reticulum Ca^{2+}-ATPases fail, leading to increased intracellular Na^+ and Ca^{2+} and increased intramitochondrial Ca^{2+} concentrations. These increased concentrations attract free water, causing cellular and mitochondrial swelling. Failure of the sarcoplasmic reticulum to resequester Ca^{2+} leads to cessation of contraction within ischemic zones of myocardium.

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(Choice D) Protein phosphorylation cascades play a role in a variety of processes including second messenger signaling and regulation of apoptosis. The cell's response to ischemia does involve initiating certain metabolic processes through protein phosphorylation, but this does not cause cell swelling.

(Choice E) Ischemia causes a net solute gain within cardiac myocytes. Elevated concentrations of Na^+ and Ca^{2+} draw free water into the cells, causing edema.

Educational Objective:
Ion pump failure due to ATP deficiency during cardiac ischemia causes intracellular accumulation of Na^+ and Ca^{2+}. The increased intracellular solute concentration draws free water into the cell, causing the cellular and mitochondrial swelling that is observed histologically.

Pathology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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A 63-year-old woman with a prolonged history of hypertension is brought to the emergency department after a syncopal episode. The patient was at church when she experienced sudden-onset, severe chest pain radiating to her back and collapsed to the floor. She regained consciousness spontaneously but has had persistent chest pain. On arrival at the emergency department, the patient is confused and agitated. Blood pressure is 85/60 mm Hg and pulse is 110/min. The jugular veins are distended. The lungs are clear on auscultation. An intraarterial catheter in the radial artery shows significant variation in systolic blood pressure related to the respiratory cycle. Chest x-ray reveals widening of the mediastinum. Which of the following is the most likely cause of this patient's syncope?

- A. Cardiac tachyarrhythmia
- B. Intravascular volume depletion
- C. Papillary muscle dysfunction
- D. Pericardial fluid accumulation
- E. Severe aortic valve narrowing

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- ✗ A. Cardiac tachyarrhythmia (2%)
- B. Intravascular volume depletion (16%)
- C. Papillary muscle dysfunction (3%)
- ✓ D. Pericardial fluid accumulation (67%)
- E. Severe aortic valve narrowing (7%)

Incorrect
Correct answer
D

67%
Answered correctly

03 secs
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Explanation

Acute aortic dissection	
Pathophysiology	• Intimal tear allows pressurized blood into aortic wall • Propagation of dissection creates false aortic lumen • Complications arise from extension into nearby structures
Risk factors	• Chronic hypertension (strongest overall risk factor)

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Explanation

Acute aortic dissection	
Pathophysiology	• Intimal tear allows pressurized blood into aortic wall • Propagation of dissection creates false aortic lumen • Complications arise from extension into nearby structures
Risk factors	• Chronic hypertension (strongest overall risk factor) • Underlying aortopathy (eg, Marfan syndrome)
Clinical presentation	• Sudden-onset, severe chest and/or back pain • Hypertension usually present* • Often a >20 mm Hg variation in SBP between arms • Often a widened mediastinum on chest x-ray
*Hypotension on presentation suggests aortic rupture or other complication (eg, cardiac tamponade, acute aortic regurgitation). **SBP** = systolic blood pressure.	

This patient with a history of **chronic hypertension** has sudden-onset, severe chest pain and a widened mediastinum on chest x-ray, which suggest an acute **aortic dissection**. The hypotension and syncope are likely due to **cardiac tamponade** (ie, pericardial fluid accumulation), a potential complication of ascending (type A) aortic dissection.

In aortic dissection, an intimal tear allows pressurized blood to enter the aortic wall, creating a split that becomes a false aortic lumen. Complications arise from the extension of the dissection into nearby structures; a proximal extension can involve the pericardium and cause aortic rupture into the pericardial sac, resulting in **hemopericardium**. The abrupt pericardial accumulation of even small amounts of blood can significantly raise pericardial pressure, causing compression of the right-sided cardiac chambers with **restricted diastolic filling** and jugular venous distension. Less blood is then pumped through the lungs to the left side of the heart, causing reduced cardiac output and hypotension (and sometimes syncope). The lungs remain clear.

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Pulsus paradoxus is a sensitive (ie, usually present) finding in cardiac tamponade that involves an exaggerated drop in systolic blood pressure during inspiration. It results from the restricted right ventricle bowing into the left ventricle during inspiration, causing a transient further decrease in cardiac output.

(Choice A) Cardiac tachyarrhythmias, most commonly ventricular tachycardia, can cause syncope due to rapid heart rate with impaired diastolic filling and reduced cardiac output. However, cardiac tachyarrhythmia would not explain pulsus paradoxus and a widened mediastinum on chest x-ray.

(Choice B) Intravascular volume depletion can occur with acute blood loss or dehydration and may cause orthostatic syncope (ie, syncope upon standing). In patients with significant intravascular volume depletion, reduced jugular venous pressure (ie, flat neck veins) is expected.

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(Choice B) Intravascular volume depletion can occur with acute blood loss or dehydration and may cause orthostatic syncope (ie, syncope upon standing). In patients with significant intravascular volume depletion, reduced jugular venous pressure (ie, flat neck veins) is expected.

(Choice C) Papillary muscle dysfunction is a potential complication of myocardial infarction that leads to acute mitral regurgitation. Sudden onset hypotension is typical and syncope may occur, but pulmonary edema is expected and pulsus paradoxus is not present.

(Choice E) Syncope can occur with severe aortic stenosis, but it is usually exertional. Physical examination reveals *pulsus parvus et tardus* (weak and delayed pulses) rather than pulsus paradoxus, and a widened mediastinum is not expected.

Educational objective:
Cardiac tamponade can occur as a complication of acute aortic dissection. It should be suspected in patients with sudden-onset, severe chest pain in combination with hypotension, jugular venous distension, and pulsus paradoxus.

Pathophysiology	Cardiovascular System	Aortic dissection
Subject	System	Topic

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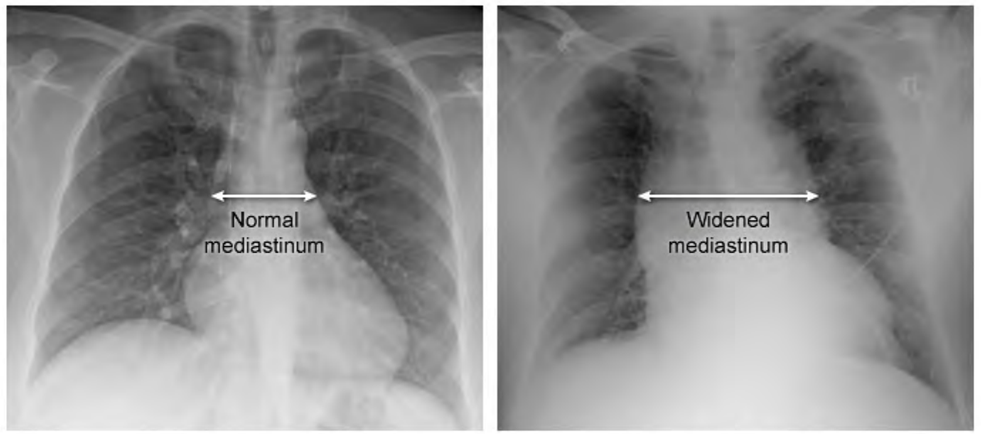
- Sudden-onset, severe chest and/or back pain
- Hypertension usually present*

Exhibit Display

Aortic dissection



Normal chest x-ray

Enlarged aortic shadow suggesting false aortic lumen

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- Sudden-onset, severe chest and/or back pain
- Hypertension usually present*

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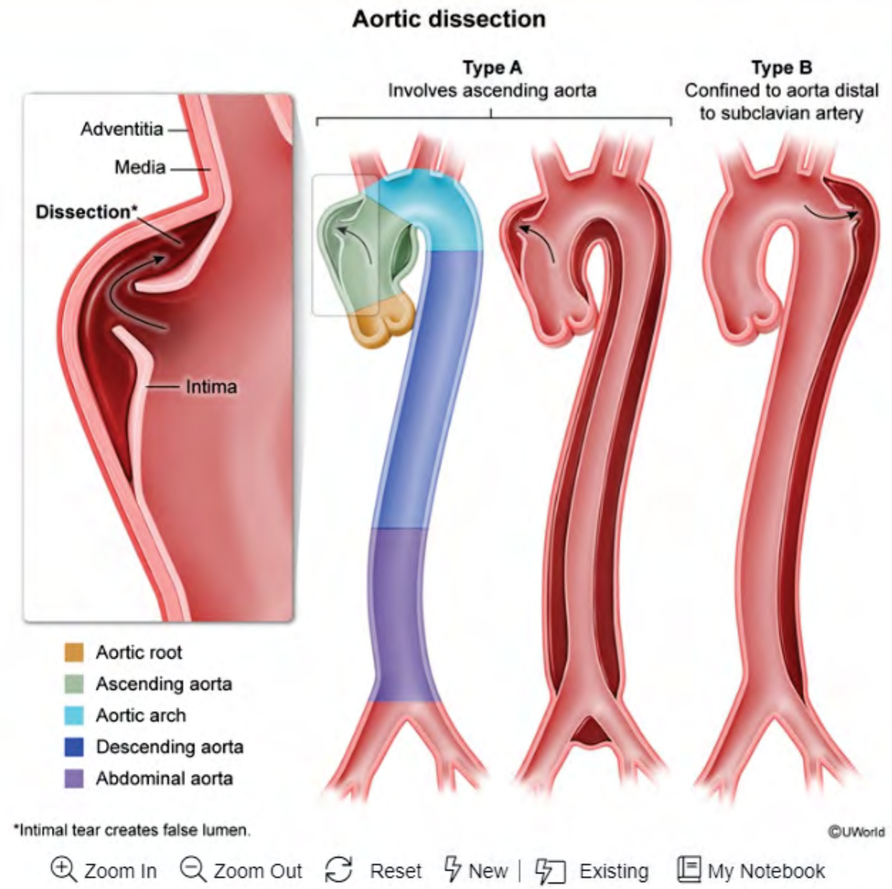

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(Choice B) Intravascular volume depletion can occur with acute blood loss or dehydration and may cause orthostatic syncope (ie, syncope upon standing). In patients with significant intravascular volume depletion, reduced jugular venous pressure (ie, flat neck veins) is expected.

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- Sudden-onset, severe chest and/or back pain
- Hypertension usually present*

Exhibit Display

Complications due to extension of acute aortic dissection	
Complication (involved structure)	• Stroke (carotid artery) • Horner syndrome (carotid sympathetic plexus) • Acute aortic regurgitation (aortic root/valve) • Myocardial ischemia/infarction (coronary artery ostia) • Pericardial effusion/tamponade (pericardium) • Hemothorax (pleural cavity) • Renal infarction (renal arteries) • Intestinal ischemia (mesenteric arteries) • Lower extremity paralysis (spinal arteries)

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*Hypotension on presentation suggests aortic rupture or other complication (eg, cardiac tamponade

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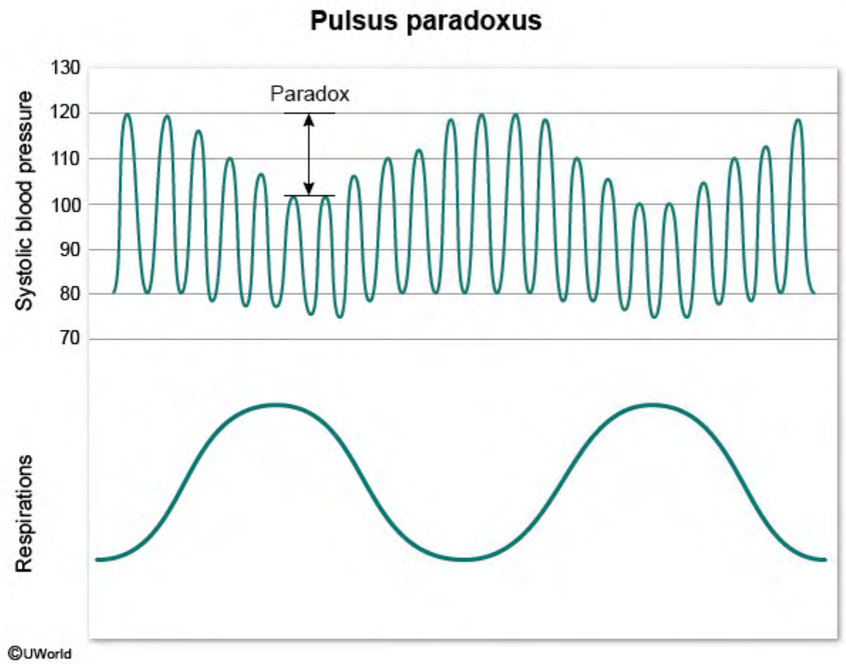

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(Choice C) Papillary muscle dysfunction is a potential complication of myocardial infarction that leads to acute mitral regurgitation. Sudden onset hypotension is typical and syncope may occur, but pulmonary edema is expected and pulsus paradoxus is not present.

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A 43-year-old woman comes to the office due to occasional chest discomfort over the past year. She describes the pain as a midline pressure or squeezing sensation that lasts 10-15 minutes and is sometimes accompanied by sweating. The patient has no history of hypertension or diabetes mellitus but is a smoker. Ambulatory ECG monitoring shows transient ST-segment elevations in the anteroseptal leads during an episode of chest pain at night. Coronary angiography reveals no atherosclerotic stenosis, but the administration of acetylcholine elicits similar chest pain and ECG changes. Which of the following best explains the coronary intervention findings in this patient?

- A. Decreased systemic resistance
- B. Endothelial dysfunction
- C. Increased venous return
- D. Positive inotropic effect
- E. Reflex tachycardia

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- ✗ A. Decreased systemic resistance (6%)
- ✓ B. Endothelial dysfunction (52%)
- C. Increased venous return (6%)
- D. Positive inotropic effect (15%)
- E. Reflex tachycardia (19%)

Incorrect
Correct answer
B

52%
Answered correctly

04 secs
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Explanation

Vasospastic angina	
Pathogenesis	• Hyperreactivity of coronary smooth muscle due to endothelial dysfunction & autonomic imbalance
	• Young age (<50) • Minimal CAD risk factors (other than

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Vasospastic angina	
Pathogenesis	• Hyperreactivity of coronary smooth muscle due to endothelial dysfunction & autonomic imbalance

Clinical presentation	• Young age (<50)
	• Minimal CAD risk factors (other than smoking)
	• Recurrent chest discomfort
	  ◦ At rest or during sleep
	  ◦ Spontaneous resolution ≤15 min

Diagnosis	• ECG: ST-segment elevation during episode • Coronary angiography: no CAD
CAD = coronary artery disease.	

This patient with episodes of **recurrent chest discomfort** that spontaneously **resolve within 10-15 minutes** most likely has **vasospastic angina** (formerly called Prinzmetal angina). It results from coronary **endothelial dysfunction** and autonomic imbalance that lead to **intermittent coronary vasospasm** and **myocardial ischemia**. Affected patients are usually young (eg, age <50) and lack typical risk factors for coronary artery disease (CAD) (eg, hypertension, diabetes mellitus); however, **smoking** is a strong risk factor, likely because it contributes to endothelial dysfunction.

Symptoms of vasospastic angina are thought to be triggered by **excess vagal tone**, and they occur most commonly **at night** when vagal tone is at a peak. The diagnosis is made by ambulatory ECG demonstrating ST-segment elevation during an episode of chest discomfort and coronary angiography revealing an absence of obstructive atherosclerotic CAD. Because of the pathophysiology of the disorder, both **acetylcholine** and **ergot alkaloids** (eg, dihydroergotamine) provoke symptoms and may also aid in diagnosis, as follows:

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Symptoms of vasospastic angina are thought to be triggered by **excess vagal tone**, and they occur most commonly **at night** when vagal tone is at a peak. The diagnosis is made by ambulatory ECG demonstrating ST-segment elevation during an episode of chest discomfort and coronary angiography revealing an absence of obstructive atherosclerotic CAD. Because of the pathophysiology of the disorder, both **acetylcholine** and **ergot alkaloids** (eg, dihydroergotamine) provoke symptoms and may also aid in diagnosis, as follows:

- Acetylcholine normally stimulates endothelial muscarinic receptors to cause vasodilation via increased release of nitric oxide. However, a **deficiency of endothelial nitric oxide** in affected patients causes increased vagal tone to instead trigger **vasoconstriction** and precipitate vasospastic symptoms.
- Ergot alkaloids activate 5-HT_2 serotonergic receptors to cause vasoconstriction. Normally, the vasoconstriction is somewhat offset by endothelial release of vasodilatory prostaglandins, but this response is lacking in affected patients due to endothelial dysfunction.

(Choices A and C) Peripheral endothelial function is typically intact in patients with vasospastic angina. As a result, acetylcholine is expected to cause peripheral vasodilation, which decreases venous return (due to venodilation). Systemic vascular resistance is also decreased, which can lower systemic blood pressure and somewhat reduce coronary artery perfusion pressure. However, this is unlikely to cause myocardial ischemia in the absence of obstructive CAD.

(Choice D) Acetylcholine has a negative inotropic effect, reducing cardiac contractility and myocardial oxygen demand.

(Choice E) Increased heart rate can increase myocardial oxygen demand and may exacerbate angina symptoms in patients with obstructive CAD, but it is unlikely to cause myocardial ischemia in the absence of CAD. In addition, acetylcholine directly slows heart rate, counteracting any reflex tachycardia caused by peripheral vasodilation.

Educational objective:
Vasospastic angina is the result of intermittent coronary vasospasm caused by endothelial dysfunction and autonomic imbalance. Although acetylcholine normally stimulates vasodilation, it triggers coronary vasospasm in affected patients due to a deficiency of vasodilatory endothelial nitric oxide.

References

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A 65-year-old man dies while hospitalized for severe breathing difficulty. The patient had several prior episodes of dyspnea and cough requiring hospitalization. He had a history of hypertension and chronic kidney disease. The patient smoked a pack of cigarettes daily for 38 years and immigrated to the United States 20 years ago. Autopsy is performed, and microscopic examination of the lungs reveals alveolar macrophages containing aggregates of golden-brown cytoplasmic granules that turn dark blue with Prussian blue staining. Which of the following conditions is most likely associated with this patient's microscopic findings?

- A. Airway hyperreactivity
- B. Coal particle inhalation
- C. Granulomatous microbial infection
- D. Left ventricular dysfunction
- E. Pulmonary arterial hypertension
- F. Pulmonary thromboembolism

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- A. Airway hyperreactivity (3%)
- B. Coal particle inhalation (11%)
- ✗ C. Granulomatous microbial infection (6%)
- ✓ D. Left ventricular dysfunction (64%)
- E. Pulmonary arterial hypertension (10%)
- F. Pulmonary thromboembolism (2%)

Incorrect
Correct answer
D

64%
Answered correctly

03 secs
Time Spent

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Explanation

This patient most likely had heart failure due to **left ventricular dysfunction**. Impaired forward pumping by the left ventricle causes increased pulmonary capillary pressure that can lead to pulmonary edema and dyspnea. The rise in hydrostatic pressure also disrupts the integrity of the pulmonary capillaries, leading to **extravasation of red blood cells** and alveolar hemorrhage. The red blood cells are eventually **phagocytosed by macrophages**, and the iron from hemoglobin is converted to hemosiderin.

Prussian blue stain detects ferric iron stores (eg, ferritin and hemosiderin). In the Prussian blue reaction, colorless potassium ferrocyanide is converted by iron to blue-black ferric ferrocyanide. Macrophages containing

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(Choice A) Airway hyperreactivity occurs in asthma or chronic obstructive pulmonary disease, neither of which is associated with hemosiderin-laden macrophages.

(Choice B) Coal worker's pneumoconiosis results from chronic inhalation of coal dust and is associated with alveolar macrophages containing black intracytoplasmic aggregates, so-called carbon-laden macrophages. These carbon aggregates do not stain blue with Prussian blue staining.

(Choice C) A number of infectious organisms (eg, *Mycobacterium tuberculosis*, *Histoplasma capsulatum*) can survive within macrophages and produce a granulomatous response in the lungs; however, these organisms are typically visualized using other special staining methods (eg, acid-fast staining) and would not stain blue with Prussian blue staining.

(Choices E and F) Pulmonary arterial hypertension (primary pulmonary hypertension) and pulmonary thromboembolism both involve an elevation in pulmonary arterial pressure; however, pulmonary edema with extravasation of red blood cells does not occur as pulmonary capillary and pulmonary venous pressures are not elevated.

Educational objective:
Alveolar hemosiderin-laden macrophages indicate alveolar hemorrhage. They most commonly result from chronic

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Educational objective:
Alveolar hemosiderin-laden macrophages indicate alveolar hemorrhage. They most commonly result from chronic elevation of pulmonary capillary hydrostatic pressure in the setting of left-sided heart failure.

Pathology	Cardiovascular System	Chronic heart failure
Subject	System	Topic

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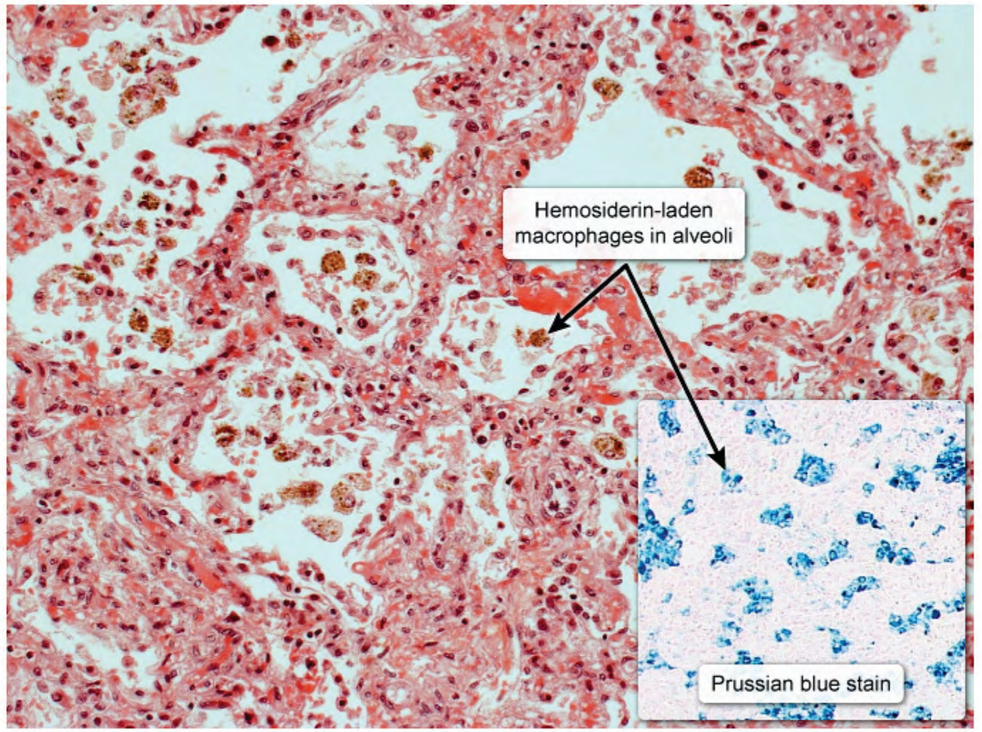
Exhibit Display

Chronic pulmonary congestion



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A 45-year-old woman collapses and dies despite appropriate resuscitation efforts. Prior to the episode, she reported substernal chest pressure brought on by exertion, as well as episodes of palpitations and light-headedness. An autopsy is performed, and myocardial histology is shown in the image below:

Which of the following is the most likely cause of this woman's condition?

- A. Genetically mediated abnormality in cell junctions
- B. Genetically mediated abnormality in ion channels
- C. Genetically mediated abnormality in sarcomeres
- D. Infiltrative disease due to deposition of amyloid fibrils
- E. Infiltrative disease due to deposition of granulomas

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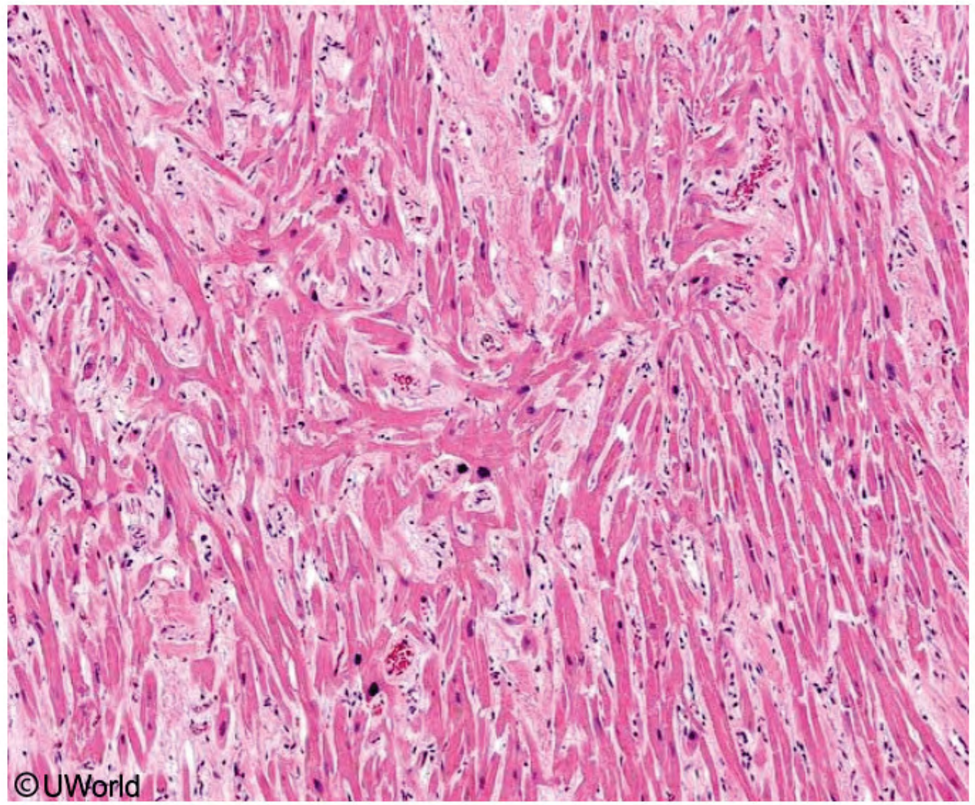

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Which

C. Genetically mediated abnormality in sarcomeres

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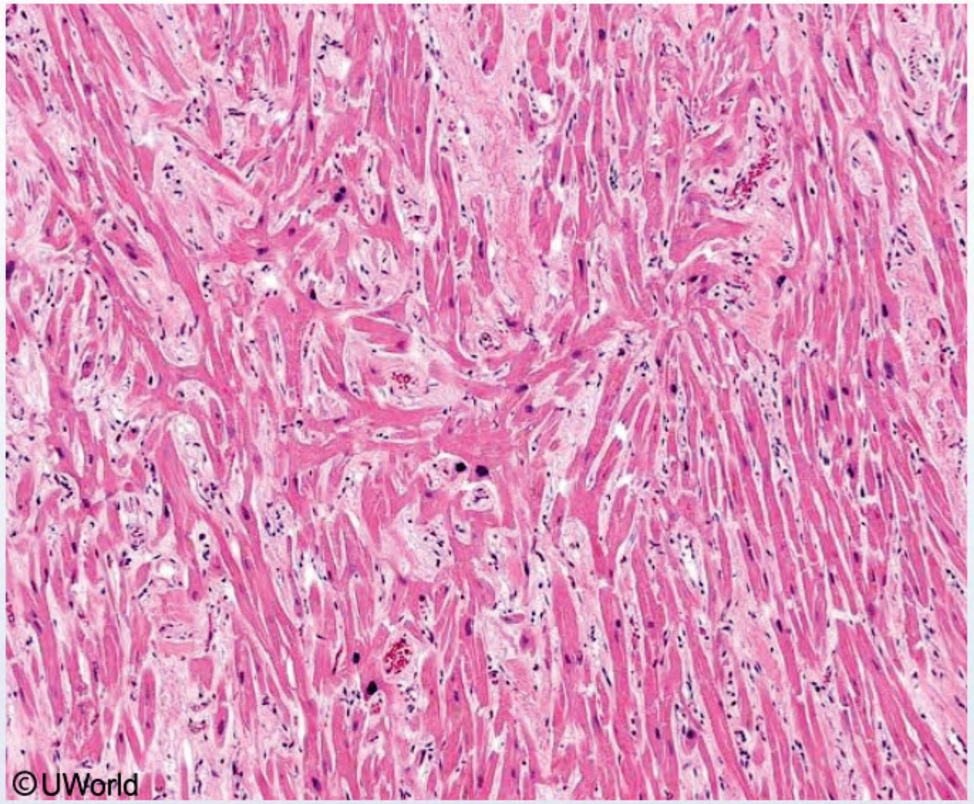

Which of the following is the most likely cause of this woman's condition?

- A. Genetically mediated abnormality in cell junctions
- B. Genetically mediated abnormality in ion channels
- C. Genetically mediated abnormality in sarcomeres
- D. Infiltrative disease due to deposition of amyloid fibrils
- E. Infiltrative disease due to deposition of granulomas

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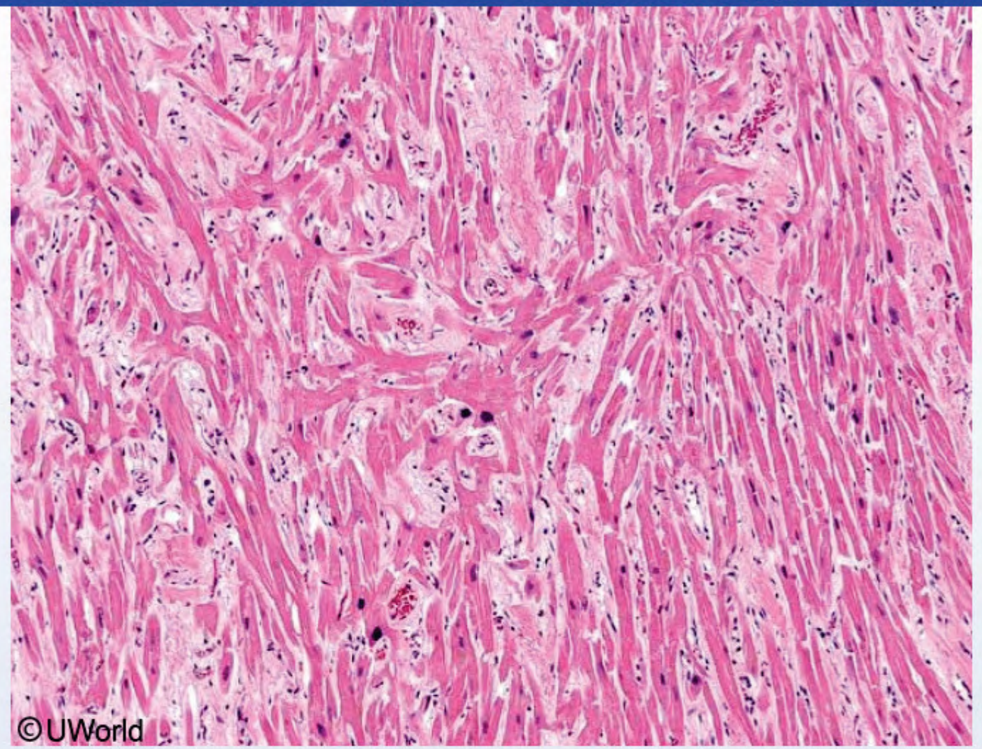

Which of the following is the most likely cause of this woman's condition?

- ✗ A. Genetically mediated abnormality in cell junctions (5%)
- B. Genetically mediated abnormality in ion channels (3%)
- ✓ C. Genetically mediated abnormality in sarcomeres (58%)
- D. Infiltrative disease due to deposition of amyloid fibrils (28%)
- E. Infiltrative disease due to deposition of granulomas (4%)

Incorrect
Correct answer
C

58%
Answered correctly

12 secs
Time Spent

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Explanation

Histologic examination of this patient's myocardium is consistent with **hypertrophic cardiomyopathy** (HCM), which is characterized by **cardiomyocyte hypertrophy** with **haphazard** cellular arrangement and prominent interstitial spacing with fibrosis. HCM is caused by genetic mutations affecting structural proteins of the **cardiac sarcomere**, most commonly beta-myosin heavy chain and myosin-binding protein C. Approximately 50% of cases result from recognized familial mutations with autosomal dominant inheritance and variable expression.

HCM is one of the most common causes of **sudden cardiac death** (SCD) in young adults. Although SCD due to HCM often occurs with exertion, it can also occur at rest. It is believed that cardiomyocyte disarray and fibrosis alter the spatial relationship of intercalated discs (the primary mediators of organized cardiac conduction), increasing susceptibility to **ventricular arrhythmia** (eg, ventricular tachycardia, ventricular fibrillation). Chronic myocardial ischemia, which can occur due to microvascular dysfunction and insufficient blood supply to hypertrophied myocardium, also contributes to interstitial fibrosis and the risk of arrhythmia.

(Choice A) Arrhythmogenic right ventricular cardiomyopathy results from mutations affecting proteins that make up desmosome cell junctions (eg, plakoglobin, desmoplakin). There is fibrosis and scarring of the right ventricle that predisposes to ventricular arrhythmias and SCD; however, histology does not demonstrate the cardiomyocyte hypertrophy or marked cellular disarray seen in HCM.

(Choice B) Long QT syndrome and Brugada syndrome result from mutations affecting cardiac ion channels. Both disorders can lead to ventricular arrhythmias and SCD, but they do not involve structural derangement of the myocardium.

(Choices D and E) Infiltrative disease of the myocardium, including the deposition of amyloid fibrils (ie, amyloidosis) and granulomas (eg, sarcoidosis), can predispose to ventricular arrhythmias and SCD; however, the infiltrative deposits are apparent on histopathology, and marked cellular disarray is not.

Educational objective:
Hypertrophic cardiomyopathy is caused by genetic mutations affecting structural proteins of cardiac sarcomeres (eg, beta-myosin heavy chain, myosin-binding protein C) and is one of the most common causes of sudden cardiac death in young adults. Histologically, it is characterized by cardiomyocyte hypertrophy with haphazard cellular

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which is characterized by **cardiomyocyte hypertrophy** with **haphazard** cellular arrangement and prominent interstitial spacing with fibrosis. HCM is caused by genetic mutations affecting structural proteins of the **cardiac sarcomere**, most commonly beta-myosin heavy chain and myosin-binding protein C. Approximately 50% of cases result from recognized familial mutations with autosomal dominant inheritance and variable expression.

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(Choice A) Arrhythmogenic right ventricular cardiomyopathy results from mutations affecting proteins that make up desmosome cell junctions (eg, plakoglobin, desmoplakin). There is fibrosis and scarring of the right ventricle that predisposes to ventricular arrhythmias and SCD; however, histology does not demonstrate the cardiomyocyte hypertrophy or marked cellular disarray seen in HCM.

(Choice B) Long QT syndrome and Brugada syndrome result from mutations affecting cardiac ion channels. Both disorders can lead to ventricular arrhythmias and SCD, but they do not involve structural derangement of the myocardium.

(Choices D and E) Infiltrative disease of the myocardium, including the deposition of amyloid fibrils (ie, amyloidosis) and granulomas (eg, sarcoidosis), can predispose to ventricular arrhythmias and SCD; however, the infiltrative deposits are apparent on histopathology, and marked cellular disarray is not.

Educational objective:
Hypertrophic cardiomyopathy is caused by genetic mutations affecting structural proteins of cardiac sarcomeres (eg, beta-myosin heavy chain, myosin-binding protein C) and is one of the most common causes of sudden cardiac death in young adults. Histologically, it is characterized by cardiomyocyte hypertrophy with haphazard cellular arrangement and interstitial fibrosis.

References

- Sudden cardiac death in hypertrophic cardiomyopathy.

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D. Infiltrative disease due to deposition of amyloid fibrils (28%)

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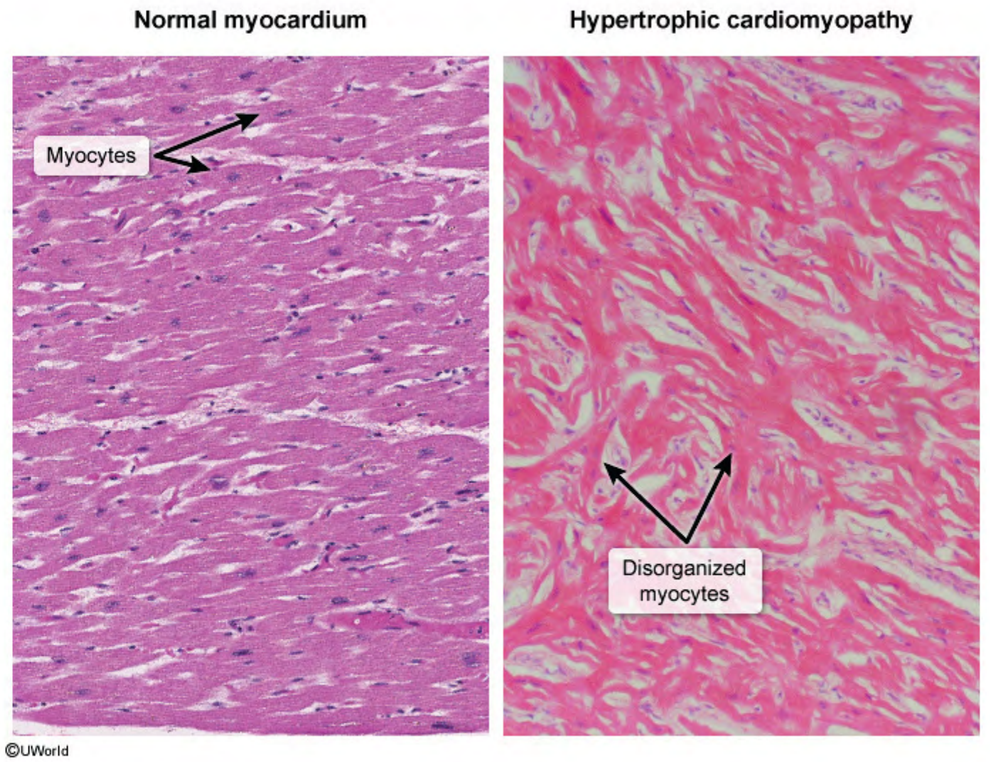

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D. Infiltrative disease due to deposition of amyloid fibrils (28%)

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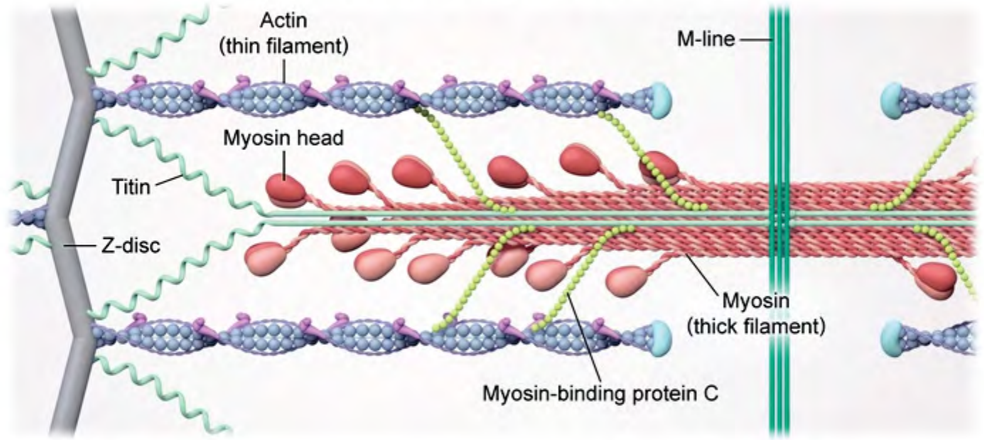
Cardiac sarcomere protein structure



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D. Infiltrative disease due to deposition of amyloid fibrils (28%)

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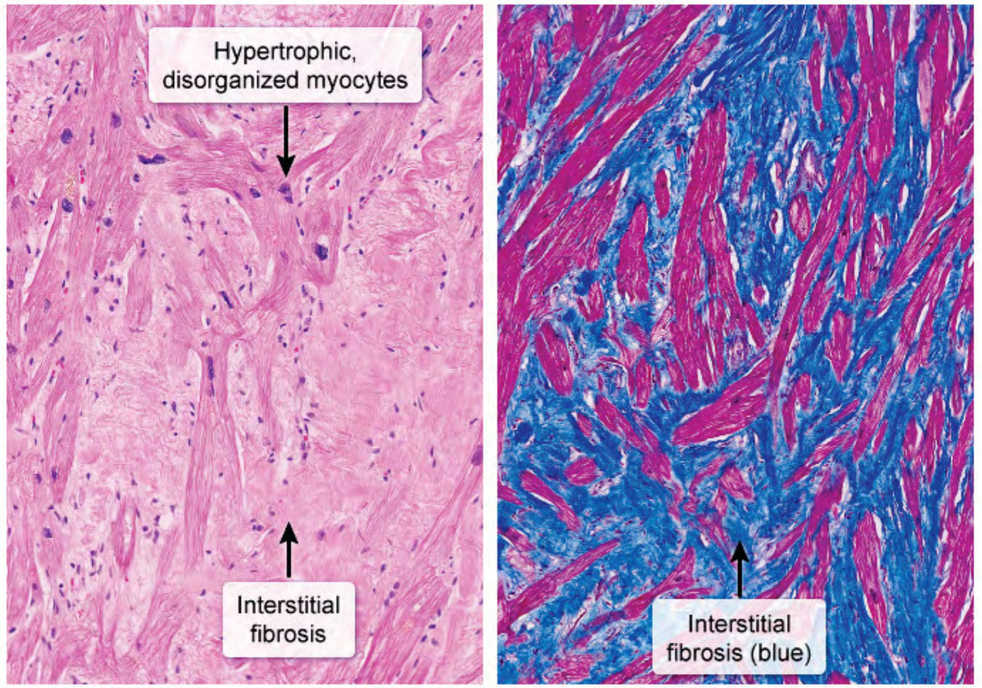
Hypertrophic cardiomyopathy



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Hematoxylin & eosin stain

Trichrome stain

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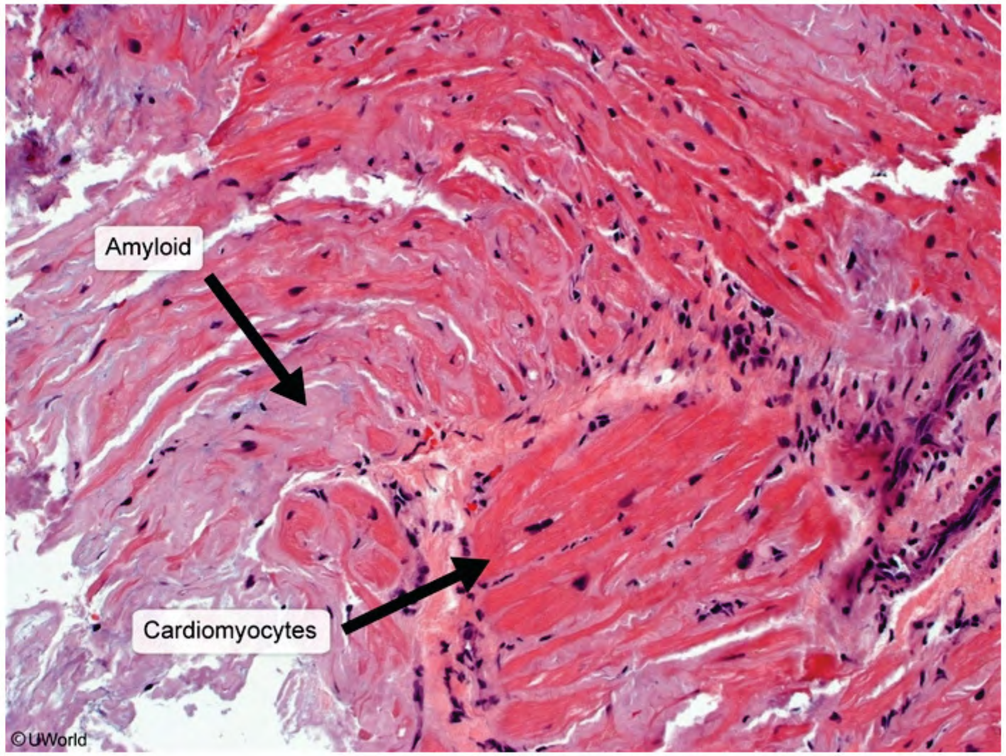

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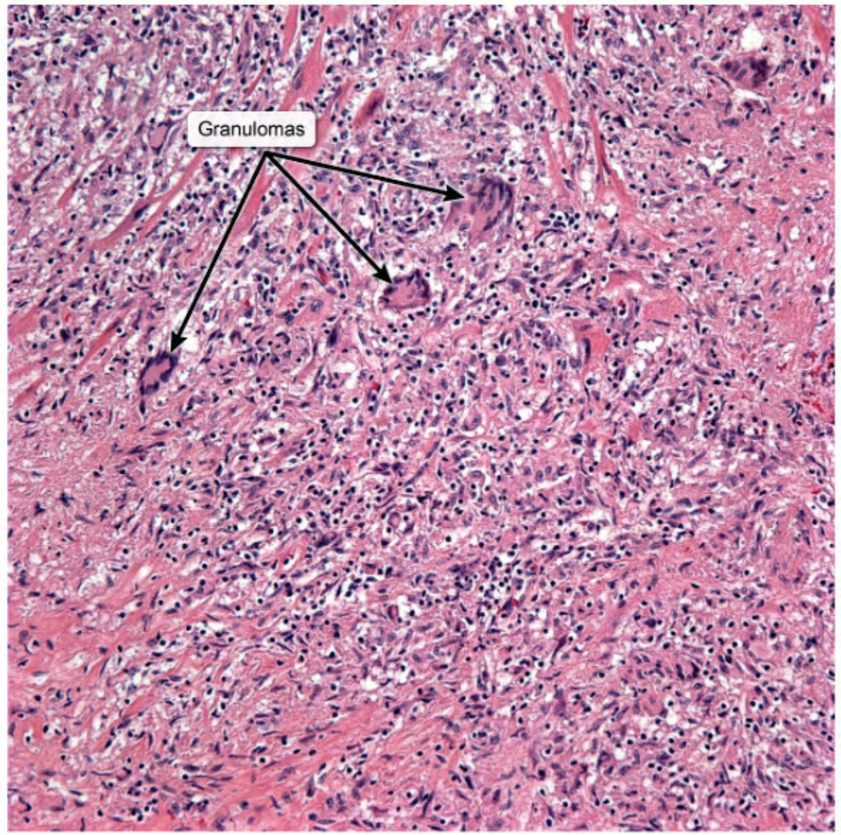
Explanation

Exhibit Display

Cardiac sarcoidosis



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A 32-year-old man dies suddenly in his sleep. An autopsy is performed and the heart is examined for signs of structural cardiac disease. The heart shows a left ventricle with a large area of apical thinning composed of fibrotic scar tissue. The mid and basal left ventricular segments show normal myocardial thickness. The coronary arteries show no evidence of obstructive atherosclerosis. The right ventricle appears normal in size. No apparent valve damage is present. Dilation and wall-thickening of the esophagus is also noted. Which of the following factors in this patient's medical history would be most helpful in determining the cause of death?

- A. Alcohol consumption
- B. Location of residence
- C. Childhood vaccinations
- D. Family history of cardiomyopathy
- E. Occupational chemical exposure

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A 32-year-old man dies suddenly in his sleep. An autopsy is performed and the heart is examined for signs of structural cardiac disease. The heart shows a left ventricle with a large area of apical thinning composed of fibrotic scar tissue. The mid and basal left ventricular segments show normal myocardial thickness. The coronary arteries show no evidence of obstructive atherosclerosis. The right ventricle appears normal in size. No apparent valve damage is present. Dilation and wall-thickening of the esophagus is also noted. Which of the following factors in this patient's medical history would be most helpful in determining the cause of death?

- ✗ A. Alcohol consumption (20%)
- ✓ B. Location of residence (46%)
- C. Childhood vaccinations (5%)
- D. Family history of cardiomyopathy (19%)
- E. Occupational chemical exposure (8%)

Incorrect
Correct answer
B

46%
Answered correctly

33 secs
Time Spent

2023
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Explanation

Chagas disease	
Epidemiology	• Caused by protozoan *Trypanosoma cruzi* • Vector: triatomine ("kissing") bug • Endemic in Central & South America
Cardiac manifestations	• Dilated cardiomyopathy with biventricular failure • Apical wall thinning with aneurysm ± mural thrombus • Ventricular arrhythmias

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Chagas disease	
Epidemiology	• Caused by protozoan *Trypanosoma cruzi* • Vector: triatomine ("kissing") bug • Endemic in Central & South America
Cardiac manifestations	• Dilated cardiomyopathy with biventricular failure • Apical wall thinning with aneurysm ± mural thrombus • Ventricular arrhythmias
Gastrointestinal manifestations	• Megaesophagus (secondary achalasia) • Megacolon

This man with a large area of localized cardiac apical wall thinning likely died of chronic **Chagas cardiomyopathy**. Chagas disease is caused by infection with the protozoan *Trypanosoma cruzi*, which is transmitted by the triatomine "kissing" bug (Reduviid family) found throughout the Americas as far north as the southern United States. Infection is rare in developed regions, but the disease is endemic throughout **Central and South America** where widespread open-air and thatched-roof housing exposes individuals to routine contact with the triatomine vector.

Years following initial infection, some individuals develop serious end-organ damage affecting the cardiac, gastrointestinal, and/or central nervous systems. Cardiac disease results from a chronic parasite-induced and immune-mediated myocarditis that leads to **dilated cardiomyopathy** (DCM). Chronic Chagas cardiomyopathy is relatively unique among DCMs in that there is characteristically localized **apical wall thinning** with the development of a large apical **aneurysm**. In addition to impairing ventricular systolic function, the aneurysm can harbor **mural thrombus** that may systemically embolize and cause stroke. Damage to the cardiac conduction system can also trigger **ventricular arrhythmias** (eg, ventricular tachycardia, ventricular fibrillation) that can lead to sudden cardiac death (the likely cause of death in this patient).

The gastrointestinal manifestations of Chagas disease result from destruction of the myenteric plexus, which can lead to progressive dilation and dysfunction of the esophagus and, less commonly, the colon.

(Choices A, C, D, and E) Excessive alcohol consumption can lead to DCM. Measles, mumps, and rubella are

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transmitted by the triatomine "kissing" bug (Reduviid family) found throughout the Americas as far north as the southern United States. Infection is rare in developed regions, but the disease is endemic throughout **Central and South America** where widespread open-air and thatched-roof housing exposes individuals to routine contact with the triatomine vector.

Years following initial infection, some individuals develop serious end-organ damage affecting the cardiac, gastrointestinal, and/or central nervous systems. Cardiac disease results from a chronic parasite-induced and immune-mediated myocarditis that leads to **dilated cardiomyopathy** (DCM). Chronic Chagas cardiomyopathy is relatively unique among DCMs in that there is characteristically localized **apical wall thinning** with the development of a large apical **aneurysm**. In addition to impairing ventricular systolic function, the aneurysm can harbor **mural thrombus** that may systemically embolize and cause stroke. Damage to the cardiac conduction system can also trigger **ventricular arrhythmias** (eg, ventricular tachycardia, ventricular fibrillation) that can lead to sudden cardiac death (the likely cause of death in this patient).

The gastrointestinal manifestations of Chagas disease result from destruction of the myenteric plexus, which can lead to progressive dilation and dysfunction of the esophagus and, less commonly, the colon.

(Choices A, C, D, and E) Excessive alcohol consumption can lead to DCM. Measles, mumps, and rubella are typically vaccinated against in childhood and are potential causes of viral DCM. Genetic mutations of the gene encoding for the cardiac sarcomere protein titin can lead to familial DCM. Miners and metal workers who are exposed to high levels of cobalt can develop DCM. These DCMs typically involve diffuse left ventricular wall thinning, unlike the characteristic localized apical wall thinning and aneurysm development that occurs in chronic Chagas cardiomyopathy. DCM due to ischemic heart disease may sometimes involve localized wall thinning with aneurysm development, but it is unlikely in this young man with no evidence of atherosclerotic coronary artery disease. Esophageal enlargement is also not expected with these other etiologies of DCM.

Educational objective:
Chagas disease is endemic to Central and South America and can lead to chronic Chagas cardiomyopathy, a dilated cardiomyopathy. Unlike the diffuse ventricular wall thinning that occurs in most dilated cardiomyopathies, chronic Chagas cardiomyopathy is characterized by localized apical wall thinning with large apical aneurysm development.

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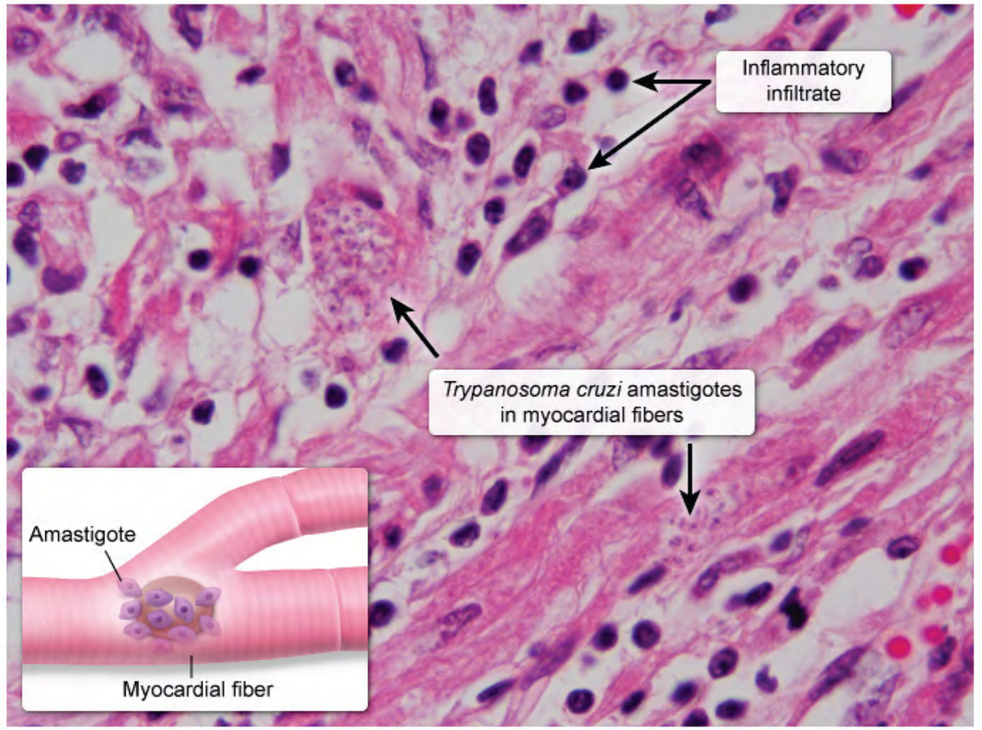
Cardiac manifestations	• Apical wall thinning with aneurysm ± mural thrombus • Ventricular arrhythmias

Exhibit Display

Chagas disease



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aneurysm development, but it is unlikely in this young man with no evidence of atherosclerotic coronary artery

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A 56-year-old man comes to the emergency department due to palpitations that started several hours ago. He has not had similar symptoms before. The patient has had no chest pain, shortness of breath, lightheadedness, or syncope. He drinks alcohol on weekends and does not use illicit drugs. He has no family history of heart disease. Blood pressure is 145/78 mm Hg. BMI is 40 kg/m^2. ECG is shown in the exhibit. The patient is treated medically in the emergency department. He is then taken to the electrophysiology laboratory for radiofrequency catheter ablation to terminate his arrhythmia. The ablation procedure should create a conduction block through which of the following areas?

- A. Accessory pathway bypassing the atrioventricular node
- B. Area between the tricuspid valve and inferior vena cava in the right atrium
- C. Area in the right ventricular outflow tract
- D. Ostia of the pulmonary veins in the left atrium
- E. Slow pathway of the atrioventricular node

Submit

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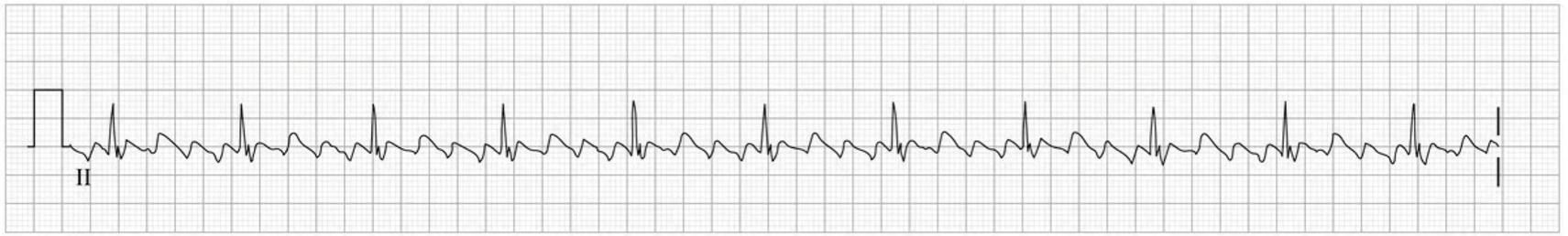

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- A. Accessory pathway bypassing the atrioventricular node (23%)
- ✓ B. Area between the tricuspid valve and inferior vena cava in the right atrium (45%)
- C. Area in the right ventricular outflow tract (3%)
- D. Ostia of the pulmonary veins in the left atrium (17%)
- E. Slow pathway of the atrioventricular node (11%)

Correct

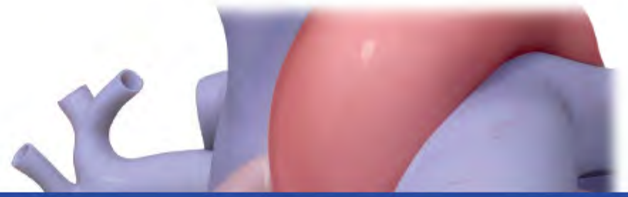
45% Answered correctly

29 secs Time Spent

2023 Version

Explanation

Reentrant circuit in atrial flutter



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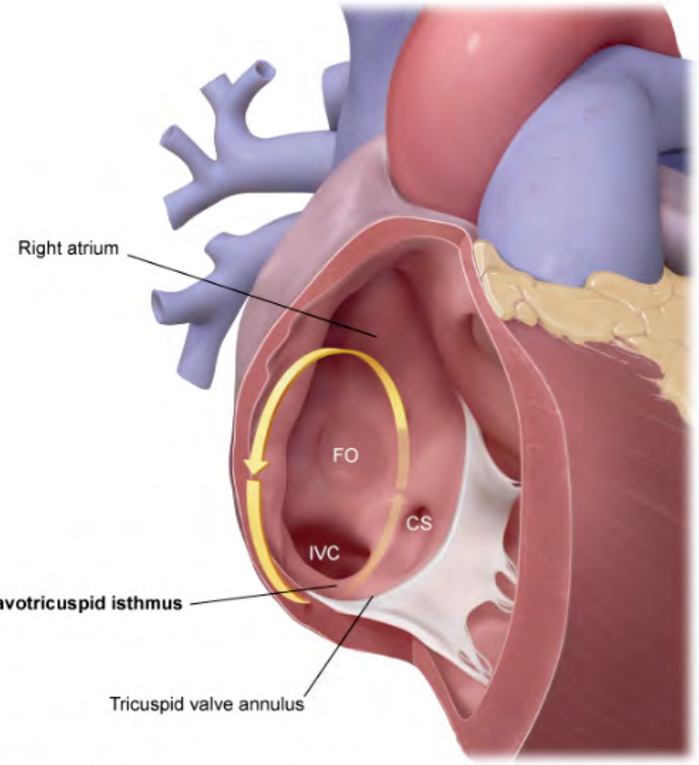
following areas?

Exhibit Display

Reentrant circuit in atrial flutter



CS = coronary sinus; **FO** = fossa ovalis; **IVC** = inferior vena cava.
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CS = coronary sinus; **FO** = fossa ovalis; **IVC** = inferior vena cava.
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This patient's ECG strip shows rapid and regular atrial activity in a **saw-toothed** pattern (flutter or F waves), consistent with **atrial flutter**. Narrow QRS complexes are typically present and the ventricular rhythm appears regular. Because the **atrial rate** is approximately **300/min**, typical ventricular rates in atrial flutter are 150/min, 100/min, or 75/min due to 2:1, 3:1, or 4:1 conduction through the atrioventricular node, respectively.

Atrial flutter shares many of the same triggers as atrial fibrillation; underlying comorbidities that cause atrial dilation (eg, heart failure) are often present and alcohol intake may contribute. Typical atrial flutter is caused by a **large reentrant circuit** that traverses the **cavotricuspid isthmus**, the region of right atrial tissue between the inferior vena cava and the tricuspid valve annulus. This region is identified during electrophysiologic study and is the target for radiofrequency ablation to interrupt the reentrant circuit and abolish atrial flutter.

(Choice A) An accessory pathway that bypasses the atrioventricular node is present in atrioventricular reentrant tachycardia, also known as Wolff-Parkinson-White syndrome. ECG shows the triad of short PR interval, wide QRS complex, and slurred upstroke of the QRS complex (delta wave).

(Choice C) The right ventricular outflow tract is the most common origination site of idiopathic ventricular tachycardia, a repetitive monomorphic ventricular tachycardia that occurs in the absence of structural heart disease. ECG shows a wide QRS complex with uniform QRS morphology and a regular rhythm.

(Choice D) The pulmonary vein ostia are often the origination site of atrial fibrillation, and catheter ablation of pulmonary vein trigger sites (pulmonary vein isolation) may be used to treat symptomatic patients. ECG reveals an irregularly irregular rhythm with varying R-R intervals and absence of P waves.

(Choice E) Dual electrical pathways (a slow and fast pathway) within or near the atrioventricular node are implicated in atrioventricular nodal reentrant tachycardia; the slow pathway is targeted for radiofrequency ablation. ECG shows narrow QRS complexes with a rapid rate and regular rhythm, and sometimes retrograde P waves.

Educational objective:
Atrial flutter demonstrates rapid and regular atrial activity in a saw-toothed pattern (flutter or F waves) on ECG. Typical atrial flutter is caused by a large reentrant circuit that traverses the cavotricuspid isthmus of the right atrium,

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consistent with **atrial flutter**. Narrow QRS complexes are typically present and the ventricular rhythm appears regular. Because the **atrial rate** is approximately **300/min**, typical ventricular rates in atrial flutter are 150/min, 100/min, or 75/min due to 2:1, 3:1, or 4:1 conduction through the atrioventricular node, respectively.

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Educational objective:
Atrial flutter demonstrates rapid and regular atrial activity in a saw-toothed pattern (flutter or F waves) on ECG. Typical atrial flutter is caused by a large reentrant circuit that traverses the cavotricuspid isthmus of the right atrium, which is the target site for radiofrequency ablation.

References

- Electrophysiologic studies in atrial flutter.

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Cavotricuspid isthmus

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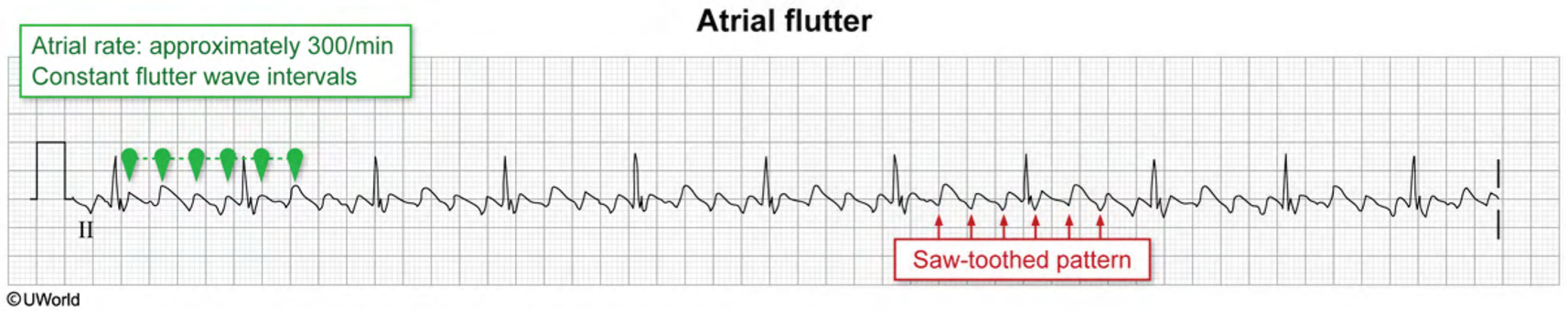

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ECG shows narrow QRS complexes with a rapid rate and regular rhythm, and sometimes retrograde P waves.

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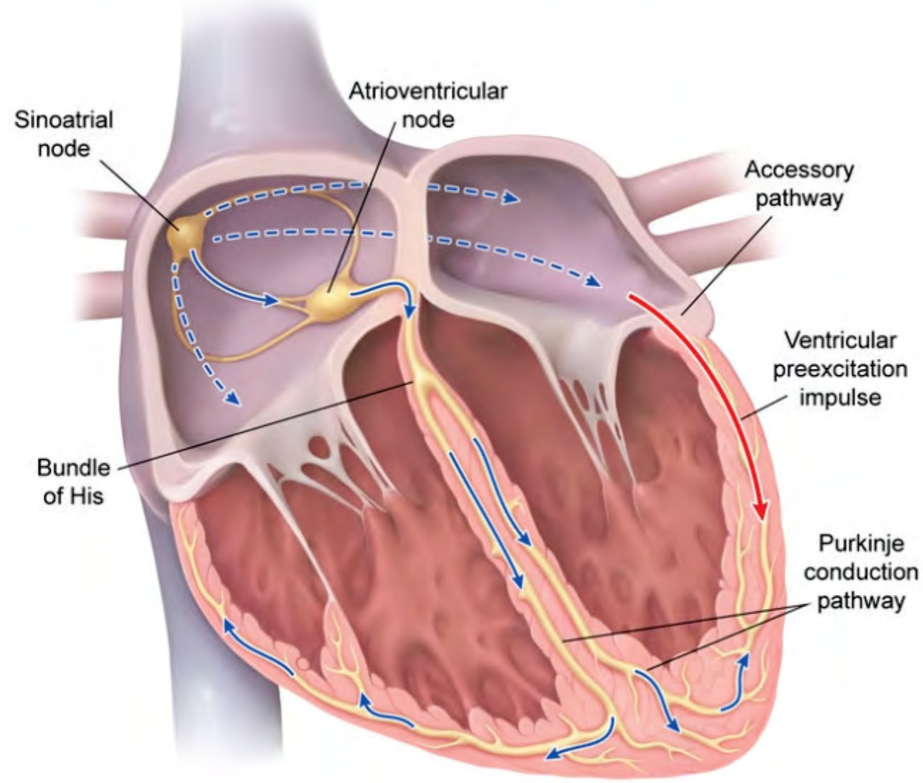
Cavotricuspid isthmus

Exhibit Display

Wolff-Parkinson-White ventricular preexcitation



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Cavotricuspid isthmus

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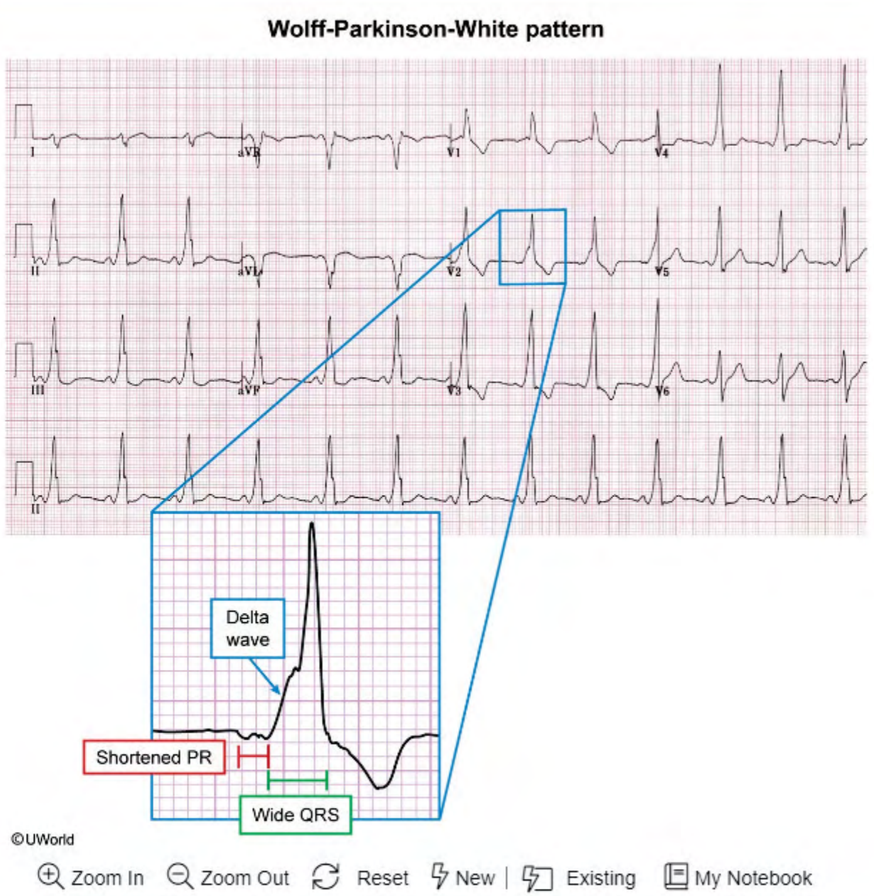

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(Choice E) Dual electrical pathways (a slow and fast pathway) within or near the atrioventricular node are implicated in atrioventricular nodal reentrant tachycardia; the slow pathway is targeted for radiofrequency ablation. ECG shows narrow QRS complexes with a rapid rate and regular rhythm, and sometimes retrograde P waves.

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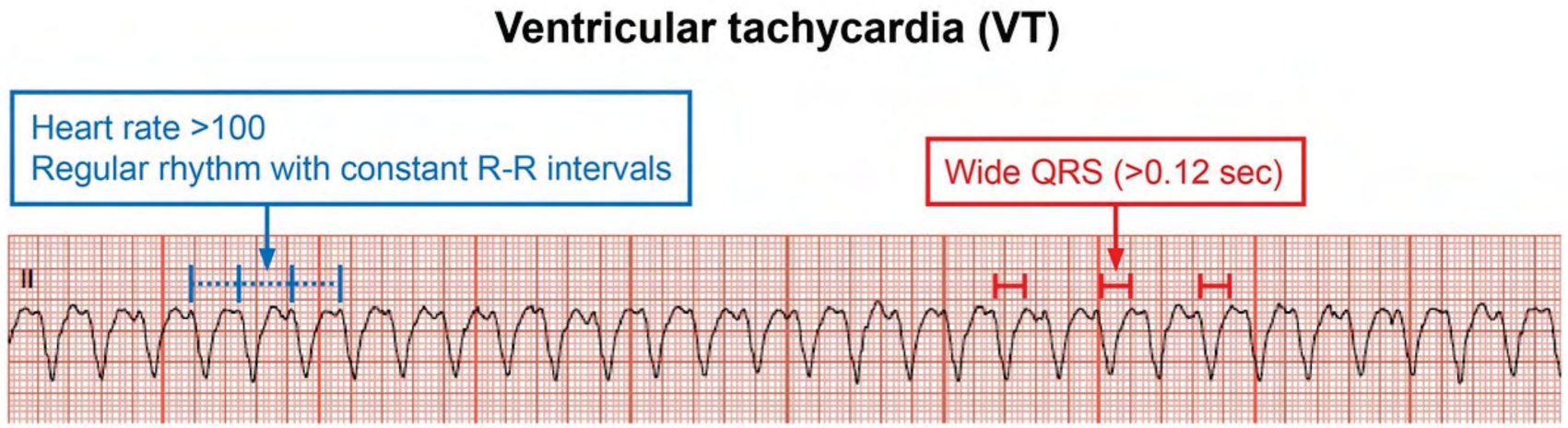

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Atrial flutter demonstrates rapid and regular atrial activity in a saw-toothed pattern (flutter or F waves) on ECG.
Typical atrial flutter is caused by a large reentrant circuit that traverses the cavotricuspid isthmus of the right atrium,

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consistent with **atrial flutter**. Narrow QRS complexes are typically present and the ventricular rhythm appears

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- Electrophysiologic studies in atrial flutter.

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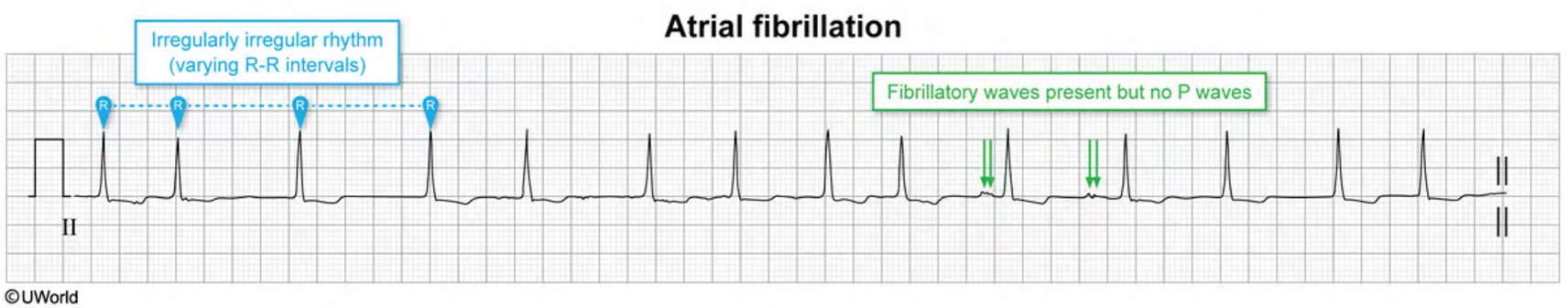

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Atrial flutter shares many of the same triggers as atrial fibrillation; underlying comorbidities that cause atrial dilation

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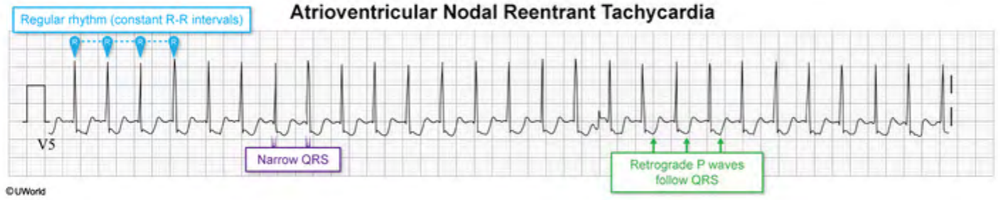

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A 34-year-old woman is being evaluated for fatigue and progressive exertional dyspnea. She goes mountain biking with friends a few times a month and recently noticed that she has to stop and rest more frequently when traveling uphill. The patient has no significant medical history and takes no medications. She occasionally drinks alcohol but does not use tobacco or illicit drugs. Her father died of a myocardial infarction at age 72. Lung auscultation is normal. An echocardiogram shows an enlarged coronary sinus. Which of the following is the most likely cause of the observed finding in this patient?

- A. Aortic dissection
- B. Coronary artery disease
- C. Hypertrophic cardiomyopathy
- D. Pericardial effusion
- E. Pulmonary hypertension

Submit

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A 34-year-old woman is being evaluated for fatigue and progressive exertional dyspnea. She goes mountain biking with friends a few times a month and recently noticed that she has to stop and rest more frequently when traveling uphill. The patient has no significant medical history and takes no medications. She occasionally drinks alcohol but does not use tobacco or illicit drugs. Her father died of a myocardial infarction at age 72. Lung auscultation is normal. An echocardiogram shows an enlarged coronary sinus. Which of the following is the most likely cause of the observed finding in this patient?

- ✗ A. Aortic dissection (2%)
- B. Coronary artery disease (16%)
- C. Hypertrophic cardiomyopathy (23%)
- D. Pericardial effusion (5%)
- ✓ E. Pulmonary hypertension (53%)

Incorrect
Correct answer
E

53%
Answered correctly

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Time Spent

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Version

Explanation

Most of the venous drainage from the myocardium traverses the **coronary sinus** (CS), which delivers deoxygenated blood to the right atrium. The CS runs transversely in the left atrioventricular groove on the posterior aspect of the heart, and opens into the right atrium between the inferior vena cava and tricuspid valve at the CS orifice. Because the CS communicates freely with the right atrium, it will become dilated by any factor that causes dilation of the right atrium. The most common cause of **coronary sinus dilation** evident on echocardiography is **elevated right-sided heart pressure** secondary to **pulmonary hypertension**. CS dilation also occurs in patients with anomalous venous drainage into the CS, including persistent left superior vena cava and total anomalous pulmonary venous return.

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Most of the venous drainage from the myocardium traverses the **coronary sinus** (CS), which delivers deoxygenated blood to the right atrium. The CS runs transversely in the left atrioventricular groove on the posterior aspect of the heart, and opens into the right atrium between the inferior vena cava and tricuspid valve at the CS orifice. Because the CS communicates freely with the right atrium, it will become dilated by any factor that causes dilation of the right atrium. The most common cause of **coronary sinus dilation** evident on echocardiography is **elevated right-sided heart pressure** secondary to **pulmonary hypertension**. CS dilation also occurs in patients with anomalous venous drainage into the CS, including persistent left superior vena cava and total anomalous pulmonary venous return.

(Choice A) Aortic dissection can occur in patients with severe hypertension. Such patients might have elevated left heart pressures, but elevated right-sided heart pressures with dilation of the CS would be unlikely.

(Choice B) The presence of asymptomatic coronary artery disease usually does not change the size of the CS. Long-standing left ventricular systolic dysfunction due to prior myocardial infarction and ischemic cardiomyopathy can lead to secondary pulmonary hypertension, with the possibility of CS dilation over time.

(Choice C) Hypertrophic cardiomyopathy usually manifests as asymmetric septal hypertrophy with or without left ventricular outflow tract obstruction. CS dilation should not occur unless there is superimposed biventricular heart failure.

(Choice D) Pericardial effusion can cause extrinsic compression of the CS.

Educational objective:
The coronary sinus communicates freely with the right atrium and will become dilated secondary to any factor that causes increased right atrial pressure. The most common cause is pulmonary hypertension, leading to elevated right heart pressures.

References

- Dilation of the coronary sinus on echocardiogram: Prevalence and significance in patients with chronic pulmonary hypertension.

Pathophysiology	Cardiovascular System	Coronary blood flow
Subject	System	Topic

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A 33-year-old man comes to the office due to fatigue and decreased exercise tolerance over the past 6 months. The patient was diagnosed with infective endocarditis in the setting of intravenous drug use a year ago and completed a course of antibiotic therapy. He has not used drugs since then. The patient has no other medical problems and takes no medications. A cardiac murmur is heard on auscultation. Both carotid arteries demonstrate a rapid rise and fall of the arterial pulse. Partial compression of the femoral arteries by the stethoscope elicits a systolic-diastolic bruit. Which of the following sets of pressure findings is most likely to be observed during cardiac catheterization of this patient?

	Aortic pressure	Left ventricular pressure
○ A.	110/75	160/20
○ B.	120/80	120/10
○ C.	120/75	120/30
○ D.	160/60	160/20
○ E.	160/95	160/10

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A 33-year-old man comes to the office due to fatigue and decreased exercise tolerance over the past 6 months. The patient was diagnosed with infective endocarditis in the setting of intravenous drug use a year ago and completed a course of antibiotic therapy. He has not used drugs since then. The patient has no other medical problems and takes no medications. A cardiac murmur is heard on auscultation. Both carotid arteries demonstrate a rapid rise and fall of the arterial pulse. Partial compression of the femoral arteries by the stethoscope elicits a systolic-diastolic bruit. Which of the following sets of pressure findings is most likely to be observed during cardiac catheterization of this patient?

		Aortic pressure	Left ventricular pressure	
✗	A.	110/75	160/20	(28%)
	B.	120/80	120/10	(4%)
	C.	120/75	120/30	(7%)
✓	D.	160/60	160/20	(48%)
	E.	160/95	160/10	(10%)

Incorrect
Correct answer
D

48%
Answered correctly

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Explanation

Aortic regurgitation pressure findings

Normal

Aortic regurgitation

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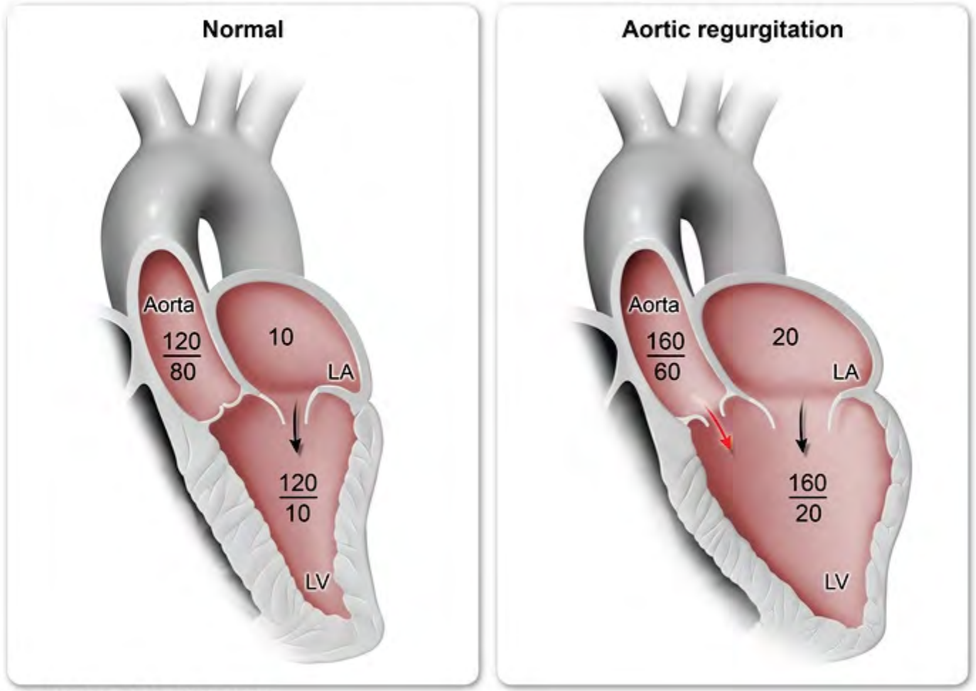
Aortic regurgitation pressure findings



LA = left atrium; **LV** = left ventricle.
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This patient's presentation is consistent with **aortic regurgitation** (AR), which likely developed as a complication of infective endocarditis. AR is characterized by a rapid **loss of aortic pressure** during diastole due to backflow of blood into the left ventricle. The left ventricle becomes dilated with an increase in end-diastolic volume and undergoes eccentric hypertrophy that allows for a compensatory **increase in stroke volume** and cardiac output. The result of these effects is a characteristic high-amplitude, **rapid rise-rapid fall pulsation** with each ventricular contraction that manifests as widened pulse pressure, rapid distension and collapse of the carotid arteries (Corrigan sign), and a "to-and-fro" bruit appreciated over the femoral arteries (Duroziez sign).

The hemodynamic changes in AR are evident on cardiac catheterization and include **reduced aortic diastolic pressure** and **elevated left ventricular (LV) diastolic pressure** due to backflow of blood during diastole. In addition, **aortic and LV systolic pressures are increased** due to high stroke volume and a compensatory increase in cardiac contractility.

(Choice A) LV systolic pressure that is higher than aortic systolic pressure is consistent with aortic stenosis. Patients with aortic stenosis have weak and delayed pulses (pulsus parvus et tardus) and pulse pressure may be reduced due to restricted stroke volume. The LV often undergoes concentric hypertrophy (wall-thickening) due to pressure overload.

(Choice B) These are normal LV and aortic pressure findings on cardiac catheterization.

(Choice C) Normal LV systolic and aortic pressures but elevated LV end-diastolic pressure is consistent with compensated heart failure. The left ventricle often undergoes eccentric hypertrophy due to volume overload.

(Choice E) Elevated aortic pressures with no systolic pressure gradient across the aortic valve and normal LV diastolic pressure is consistent with systemic hypertension. Concentric hypertrophy may develop due to pressure overload.

Educational objective:
Aortic regurgitation causes a rapid fall in aortic pressure during diastole with an increase in left ventricular end-diastolic volume and a compensatory increase in stroke volume. These hemodynamic changes create characteristic pressure changes, including reduced aortic diastolic pressure, increased aortic systolic pressure, and increased left ventricular diastolic and systolic pressures.

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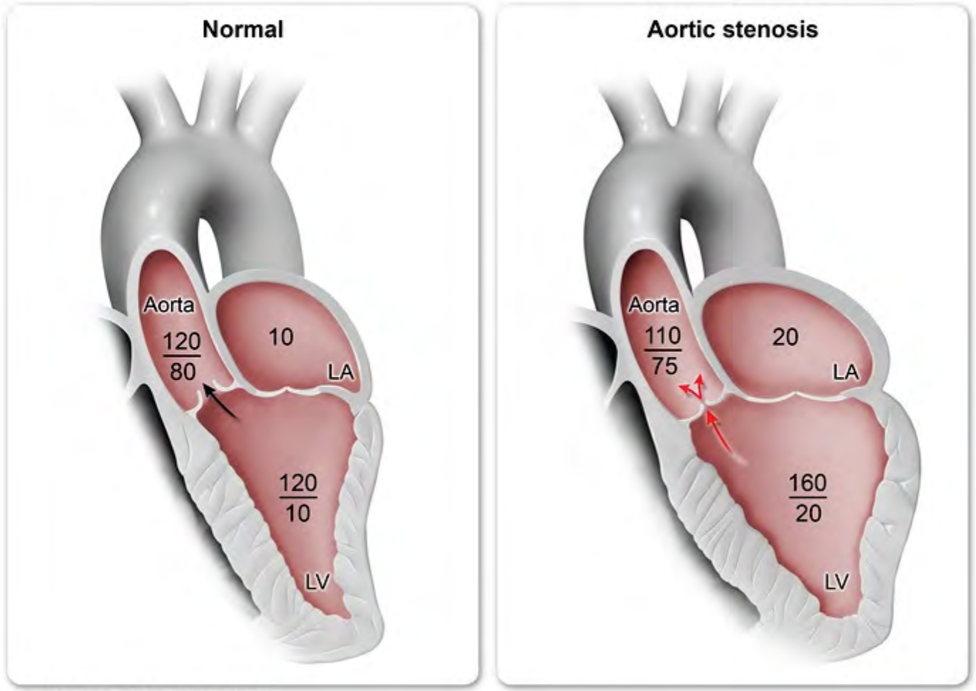
Aortic stenosis pressure findings



LA = left atrium; **LV** = left ventricle.

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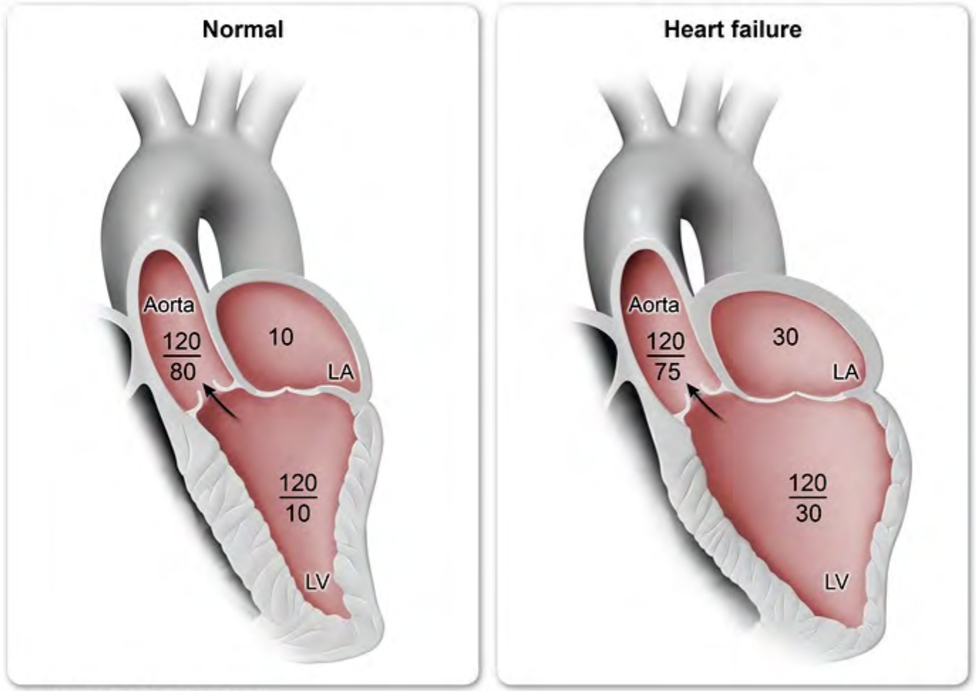
Heart failure pressure findings



LA = left atrium; **LV** = left ventricle.
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This patient's presentation is consistent with **aortic regurgitation** (AR), which likely developed as a complication of infecti... AR is characterized by a rapid **loss of aortic pressure** during diastole due to backflow of blood ... underg... The re... contra... (Corrig...

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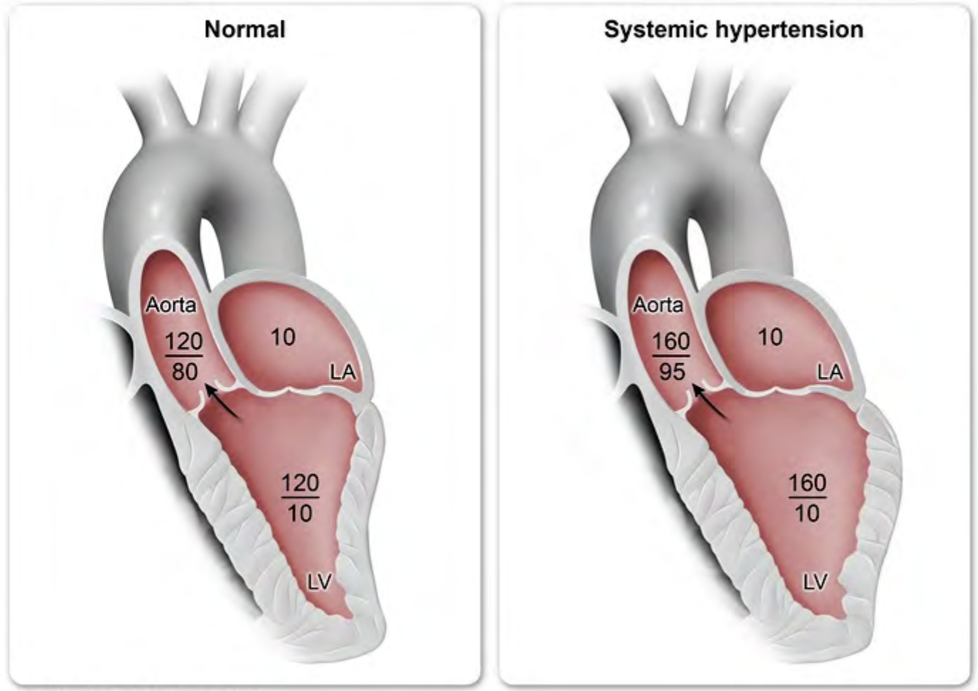
Systemic hypertension pressure findings



LA = left atrium; **LV** = left ventricle.

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A 33-year-old, previously healthy woman comes to the emergency department due to sudden-onset shortness of breath and left-sided chest pain. The symptoms began an hour ago while she was doing yard work, and she nearly passed out at symptom onset. The patient takes no medications or supplements other than ibuprofen for occasional headaches. She does not use tobacco, alcohol, or illicit drugs. Temperature is 37.1 C (98.8 F), blood pressure is 84/58 mm Hg, pulse is 122/min, and respirations are 24/min. Pulse oximetry is 86% on room air. On physical examination, the patient is in moderate distress. The jugular veins are distended. Heart sounds are rapid and regular with no murmurs. Lungs are clear to auscultation bilaterally with no crackles or wheezes. Arterial blood gas results are pH 7.52, $PaCO_2$ 28 mm Hg, and PaO_2 54 mm Hg. Which of the following is most likely to be seen on bedside echocardiography?

- A. Enlarged left ventricular cavity
- B. Enlarged right ventricular cavity
- C. Left atrial enlargement
- D. Left ventricular wall thickening
- E. Right ventricular wall thickening

Submit

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- A. Enlarged left ventricular cavity (5%)
- ✓ B. Enlarged right ventricular cavity (40%)
- C. Left atrial enlargement (7%)
- D. Left ventricular wall thickening (8%)
- E. Right ventricular wall thickening (37%)

Correct

40%
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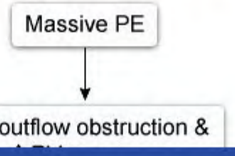
Explanation

Pathophysiology of massive pulmonary embolism



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and regular with no murmurs. Lungs are clear to auscultation bilaterally with no crackles or wheezes. Arterial blood gas results are pH 7.52, PaCO2 28 mm Hg, and PaO2 54 mm Hg. Which of the following is most likely to be seen

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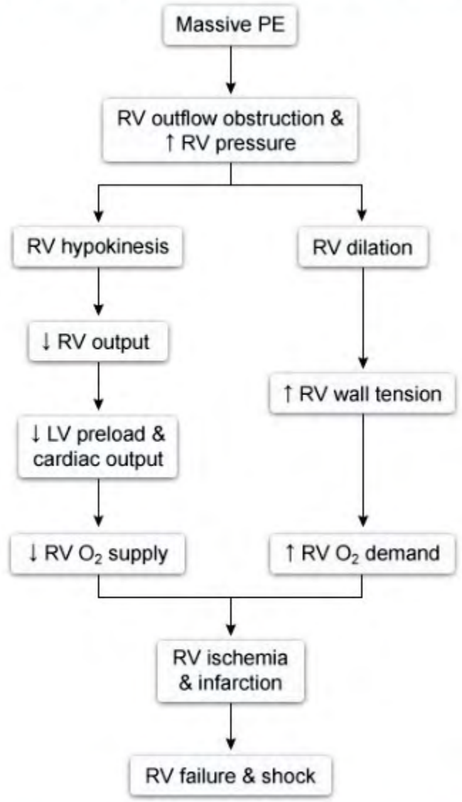
Pathophysiology of massive pulmonary embolism



LV = left ventricular; **PE** = pulmonary embolism; **RV** = right ventricular.

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This patient most likely has an **acute pulmonary embolism** (PE). The classic presentation is acute-onset chest pain and shortness of breath that may be accompanied by syncope or near-syncope. Patients usually have tachycardia, tachypnea, jugular venous distension, and clear lungs. Arterial blood gas typically show hypoxemia and acute respiratory alkalosis due to hyperventilation. Up to 30% of cases may present with no apparent risk factors (eg, hypercoagulability).

Massive PE can lead to hypotension and obstructive shock. There is a rapid increase in pulmonary arterial resistance that leads to an increase in pulmonary arterial and right ventricular (RV) pressure. The rapid pressure increase causes **RV cavity enlargement** due to increased RV wall tension and cardiac muscle stretching. RV myocardial oxygen demand increases and coronary artery perfusion decreases, leading to a supply/demand mismatch and RV ischemia. Consequent **RV dysfunction** then leads to an inability to pump blood through the pulmonary circulation, resulting in decreased left-sided preload and **decreased cardiac output**. Such RV failure caused by an increase in pulmonary vascular resistance is sometimes called **cor pulmonale**.

(Choice A) An enlarged left ventricular (LV) cavity usually results from eccentric hypertrophy in the setting of chronic volume overload, such as occurs in dilated cardiomyopathy or severe aortic or mitral valve regurgitation. In acute PE, the LV cavity is either normal or somewhat reduced in size due to reduced blood flow coming from the right side of the heart.

(Choice C) Left atrial enlargement is typical of severe mitral stenosis, which usually occurs in the setting of rheumatic heart disease. Undiagnosed mitral stenosis can sometimes present acutely with decompensated heart failure; however, a rumbling diastolic murmur and evidence of pulmonary edema (eg, crackles on lung auscultation) are expected.

(Choice D) LV wall thickening is expected with the concentric hypertrophy that occurs with prolonged systemic hypertension or severe aortic stenosis.

(Choice E) RV wall thickening is seen with the concentric hypertrophy that occurs in patients with chronic pulmonary hypertension. In acute PE, there is no time for compensatory wall thickening to occur in response to the increased pressure load; the rapid increase in pressure causes RV dilation that may be accompanied by RV failure.

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resistance that leads to an increase in pulmonary arterial and right ventricular (RV) pressure. The rapid pressure increase causes **RV cavity enlargement** due to increased RV wall tension and cardiac muscle stretching. RV myocardial oxygen demand increases and coronary artery perfusion decreases, leading to a supply/demand mismatch and RV ischemia. Consequent **RV dysfunction** then leads to an inability to pump blood through the pulmonary circulation, resulting in decreased left-sided preload and **decreased cardiac output**. Such RV failure caused by an increase in pulmonary vascular resistance is sometimes called **cor pulmonale**.

(Choice A) An enlarged left ventricular (LV) cavity usually results from eccentric hypertrophy in the setting of chronic volume overload, such as occurs in dilated cardiomyopathy or severe aortic or mitral valve regurgitation. In acute PE, the LV cavity is either normal or somewhat reduced in size due to reduced blood flow coming from the right side of the heart.

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(Choice D) LV wall thickening is expected with the concentric hypertrophy that occurs with prolonged systemic hypertension or severe aortic stenosis.

(Choice E) RV wall thickening is seen with the concentric hypertrophy that occurs in patients with chronic pulmonary hypertension. In acute PE, there is no time for compensatory wall thickening to occur in response to the increased pressure load; the rapid increase in pressure causes RV dilation that may be accompanied by RV failure.

Educational objective:
A large, acute pulmonary embolism causes a rapid increase in right ventricular (RV) pressure that leads to RV cavity enlargement and RV dysfunction. Thickening of the RV wall is not seen in acute pulmonary embolism, as there is no time for compensatory hypertrophy to occur in response to the increased pressure load.

References

- Pulmonary embolism, part I: Epidemiology, risk factors and stratification, pathophysiology, clinical presentation, diagnosis, and nonthrombotic pulmonary embolism

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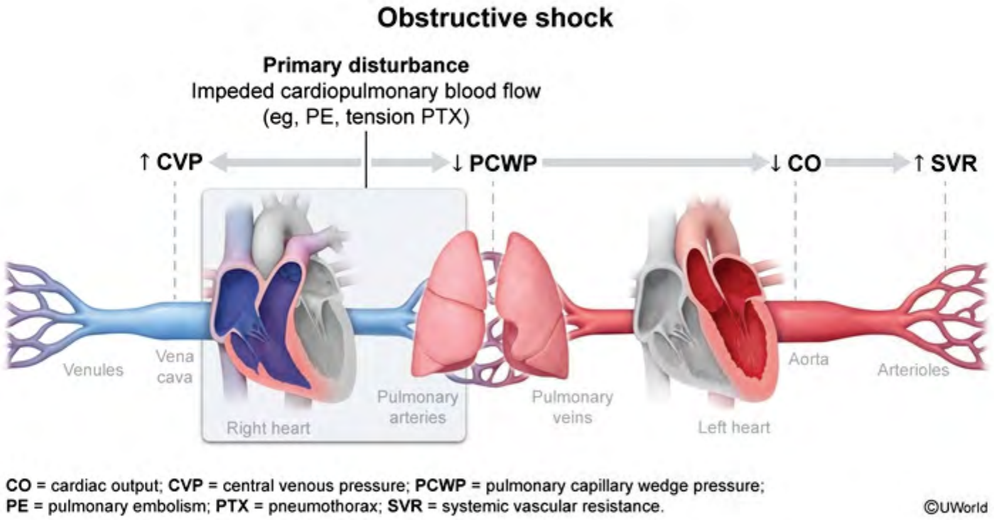

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(Choice E) RV wall thickening is seen with the concentric hypertrophy that occurs in patients with chronic

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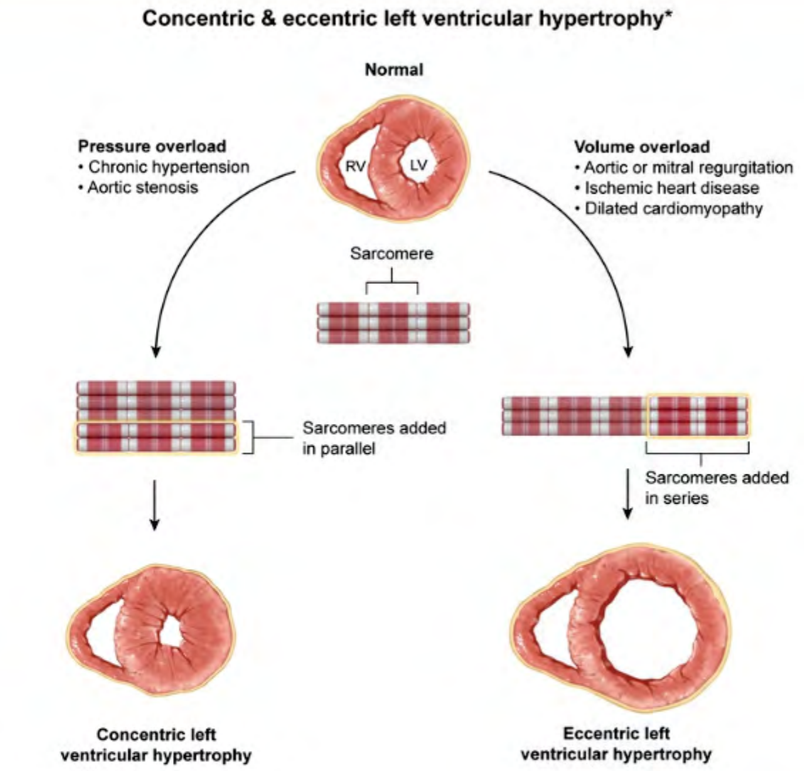

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(Choice D) LV wall thickening is expected with the concentric hypertrophy that occurs with prolonged systemic hypertension or severe aortic stenosis.

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A 45-year-old man comes to clinic due to frequent episodes of palpitations accompanied by dizziness, fatigue, and shortness of breath. Prolonged ECG monitoring identifies episodes of atrial fibrillation associated with a rapid ventricular response rate. A radiofrequency ablation procedure is planned. The access site is the right femoral vein. The ablation catheter is advanced to the left atrium where radiofrequency energy is used to eliminate an ectopic focus of abnormal electrical activity. During the procedure, the catheter most likely passes through which of the following structures?

- A. Aortic valve
- B. Interatrial septum
- C. Interventricular septum
- D. Pulmonic valve
- E. Tricuspid valve

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- A. Aortic valve (6%)
- ✓ B. Interatrial septum (62%)
- C. Interventricular septum (2%)
- D. Pulmonic valve (11%)
- E. Tricuspid valve (16%)

Correct

62% Answered correctly

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Explanation

Trans-septal left atrial catheterization

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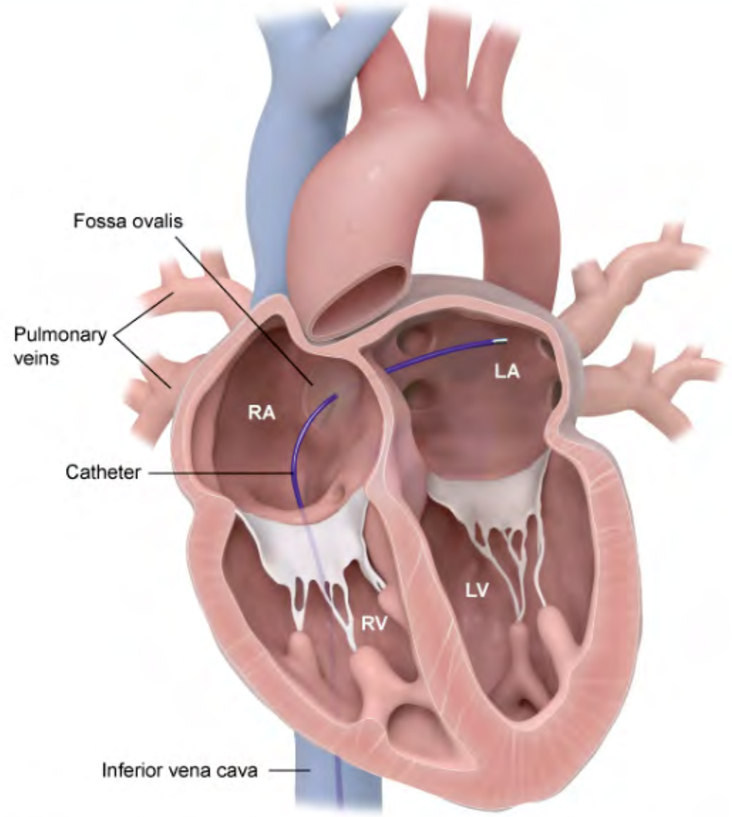
Trans-septal left atrial catheterization



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Fossa ovalis

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A **venous catheter** traveling from the femoral vein to the heart passes through the iliac vein and inferior vena cava to reach the **right atrium**. Once in the right atrium, structures within the right side of the heart and the pulmonary arteries are readily accessible. However, because the pulmonary capillaries are far too small to pass through, the left side of the heart must be accessed by **traversing the interatrial septum**. The interatrial septum is traversed at the site of the **foramen ovale**, which in adults is typically covered by a thin membrane of fibrous tissue that can be easily punctured.

Entry into the **left atrium** allows for direct measurement of left atrial pressure (rather than an estimate via pulmonary capillary wedge pressure) and for access to arrhythmogenic foci that may be located on the left atrial myocardium or the pulmonary veins. Following the procedure, the small atrial septal defect created by the catheter typically closes spontaneously.

(Choice A) Arterial catheterization of the left side of the heart typically starts in the femoral or radial artery and proceeds to the ascending aorta where pressure can be measured or dye can be placed into the coronary arteries to visualize atherosclerotic obstruction (ie, coronary angiography). In addition, the aortic valve may be crossed to measure left ventricular pressure. However, retrograde crossing of the structurally complex mitral valve is highly difficult, and the left atrium is not accessed via this route.

(Choice C) The interventricular septum is not traversed during heart catheterization because the myocardium is thick and difficult to puncture, and high left ventricular pressure would likely prevent spontaneous closure of the ventricular septal defect that is created.

(Choices D and E) The tricuspid valve is crossed during venous catherization to access the right ventricle, and the pulmonic valve is subsequently crossed to access the pulmonary arteries. However, because the catheter cannot pass through the pulmonary capillaries, the left atrium cannot be accessed via this route.

Educational objective:
To access the left side of the heart, cardiac venous catheters must cross the interatrial septum at the site of the foramen ovale. Entry into the left atrium allows for direct measurement of left atrial pressure and for access to arrhythmogenic foci on the left atrial myocardium or pulmonary veins.

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A **venous catheter** traveling from the femoral vein to the heart passes through the iliac vein and inferior vena cava

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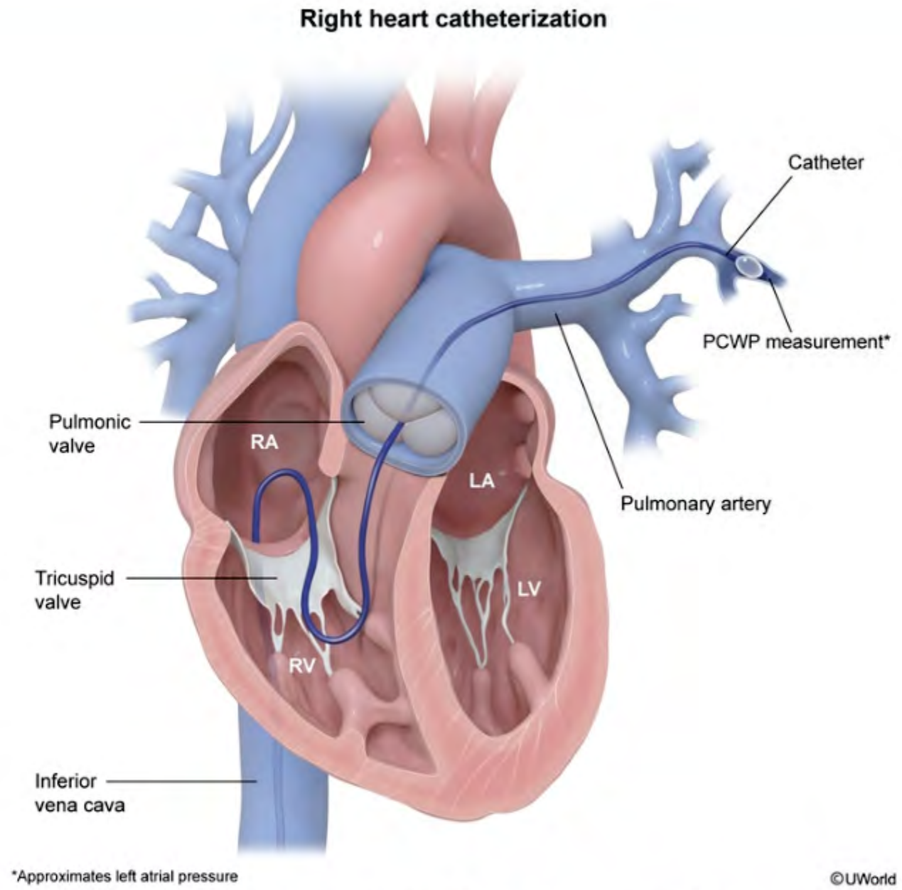

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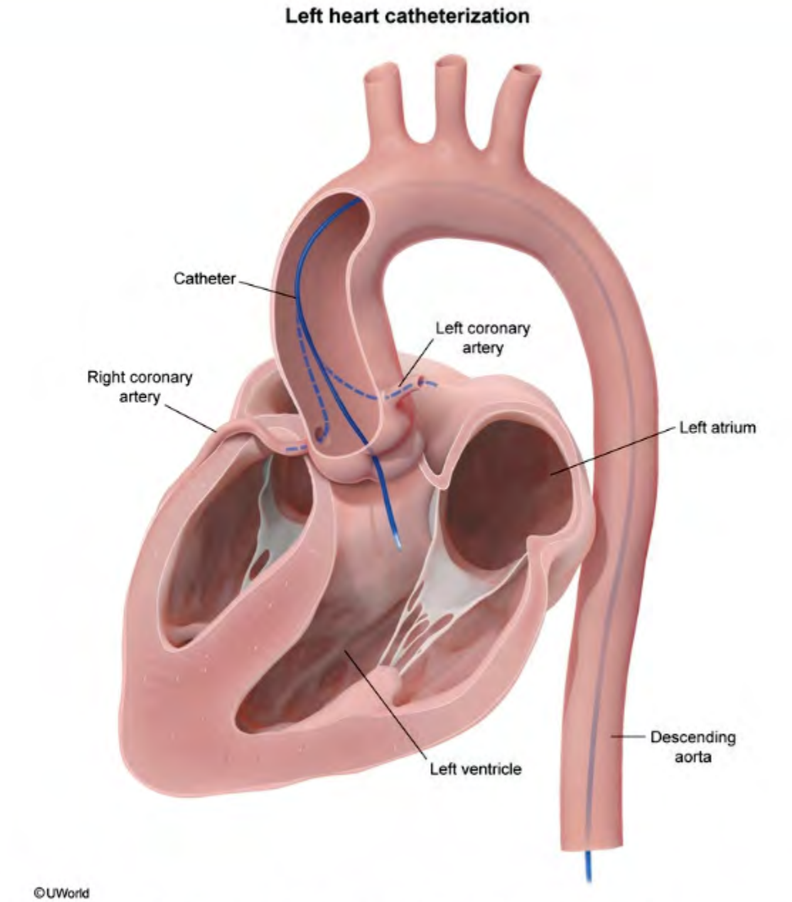

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A 14-year-old girl is brought to the office to establish medical care. Vital signs are within normal limits. Cardiac examination is significant for a murmur that is best heard at the left upper sternal border. Phonocardiogram obtained at this location reveals the findings shown below:

This patient's murmur is most likely due to which of the following mechanisms?

- A. Abnormal narrowing of the aortic valve
- B. Incomplete ventricular septum development
- C. Increased blood flow to the lungs
- D. Regurgitation of blood from the aorta

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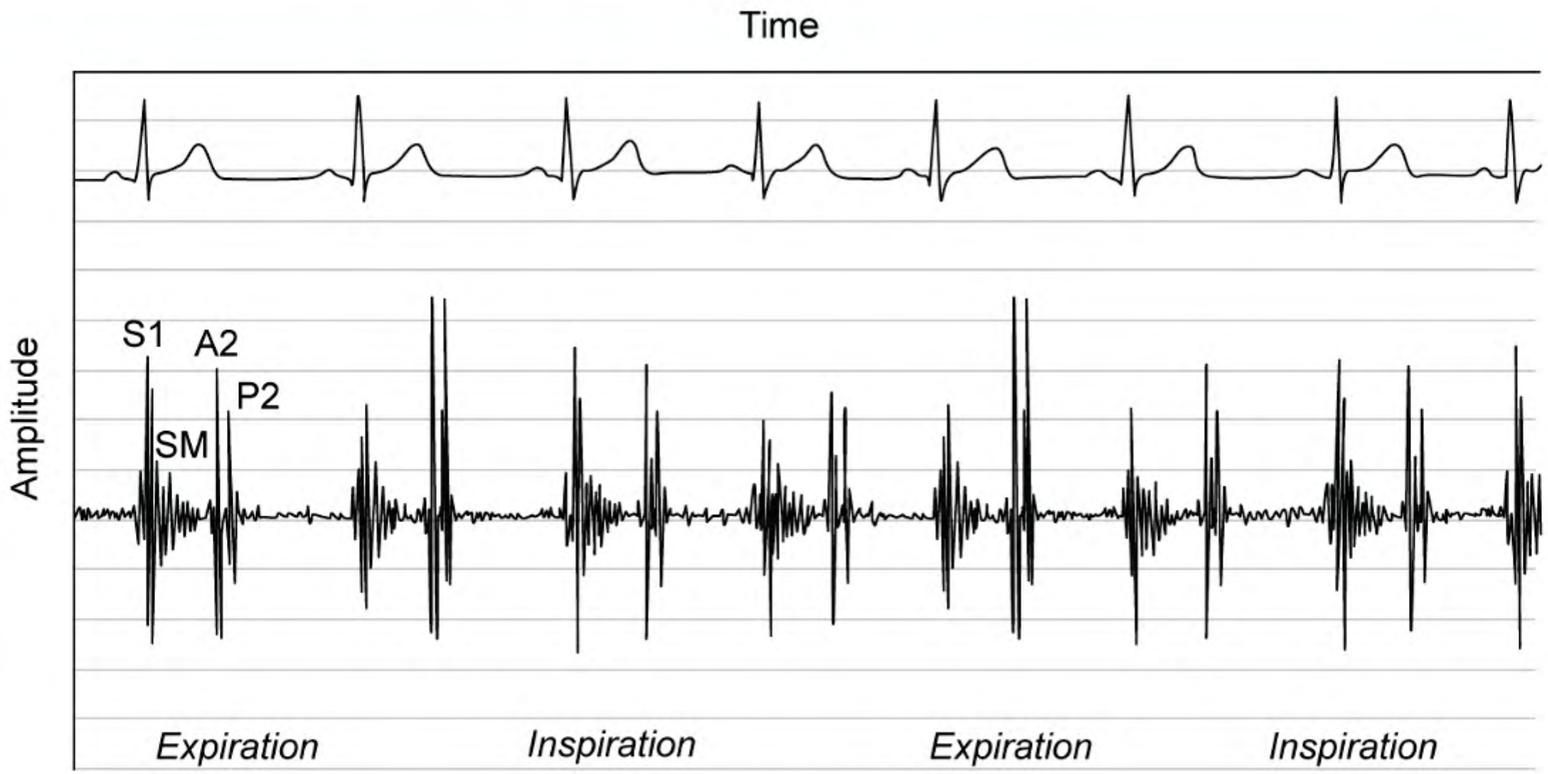

A2 = aortic component of 2nd heart sound; **P2** = pulmonic component of 2nd heart sound;
S1 = 1st heart sound; **SM** = systolic murmur.

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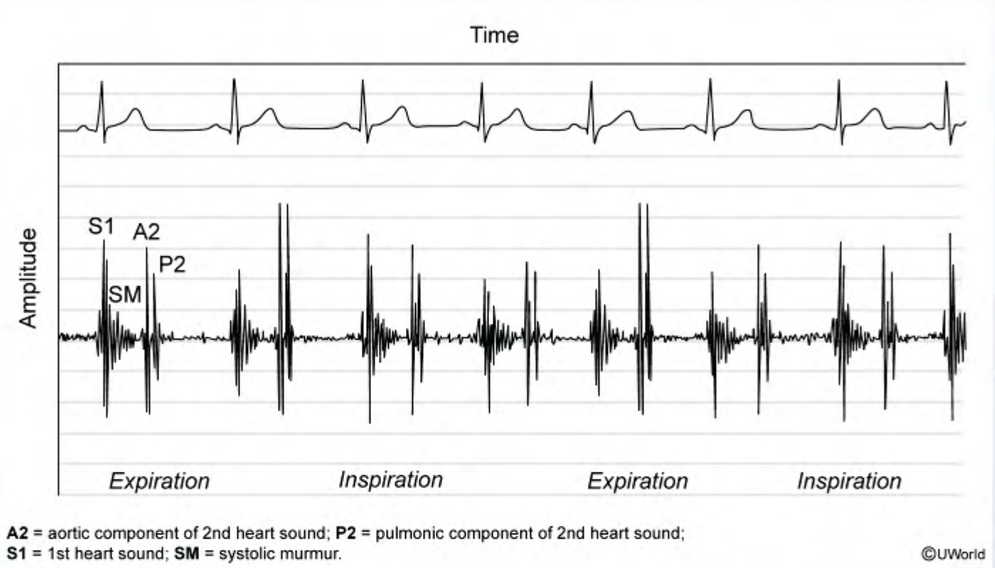
A 14-year-old girl is brought to the office to establish medical care. Vital signs are within normal limits. Cardiac examination is significant for a murmur that is best heard at the left upper sternal border. Phonocardiogram obtained at this location reveals the findings shown below:



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- A. Abnormal narrowing of the aortic valve
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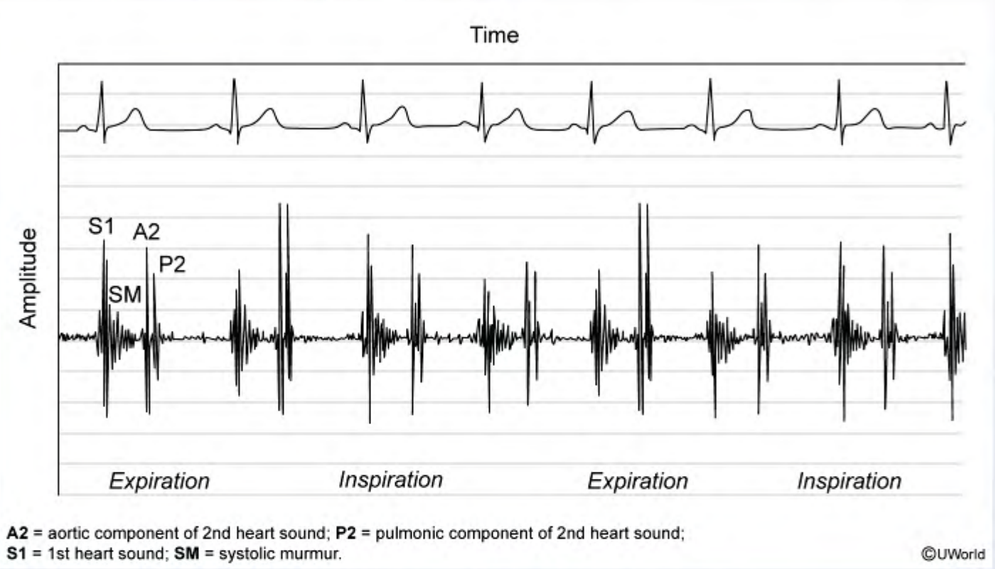
A 14-year-old girl is brought to the office to establish medical care. Vital signs are within normal limits. Cardiac examination is significant for a murmur that is best heard at the left upper sternal border. Phonocardiogram obtained at this location reveals the findings shown below:



A2 = aortic component of 2nd heart sound; **P2** = pulmonic component of 2nd heart sound;
S1 = 1st heart sound; **SM** = systolic murmur.

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This patient's murmur is most likely due to which of the following mechanisms?

- A. Abnormal narrowing of the aortic valve (25%)
- ✗ B. Incomplete ventricular septum development (26%)
- ✓ C. Increased blood flow to the lungs (37%)
- D. Regurgitation of blood from the aorta (10%)

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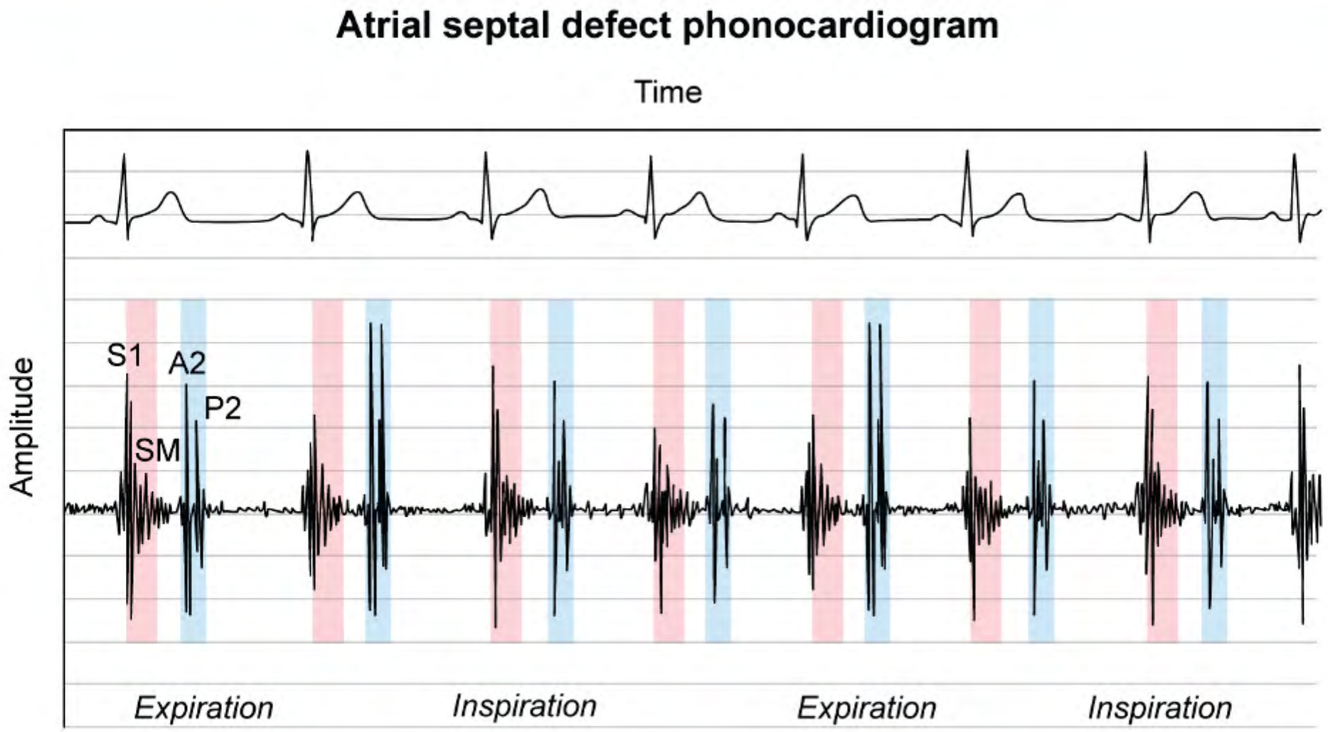

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the pulmonary arteries across the **pulmonic valve**. This creates the characteristic early-peaking **midsystolic**

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This patient's phonocardiogram shows an early-peaking systolic murmur as well as a widely split S2 that is fixed (ie, no variation between inspiration and expiration). These findings are characteristic of an **atrial septal defect** (ASD), a common congenital heart lesion resulting in an abnormal connection between the right and left atria.

Left atrial pressure is normally slightly higher than in the right atrium due to the greater compliance of the thin-walled right ventricle; as a result, ASDs cause **left-to-right shunting**. This blood flow has low velocity and turbulence and therefore does not create a murmur directly. Instead, the increased volume of blood delivered to the right heart (ie, normal venous return plus extra blood through the shunt) results in **increased blood flow** into the pulmonary arteries across the **pulmonic valve**. This creates the characteristic early-peaking **midsystolic ejection murmur** over the left upper sternal border (ie, pulmonic flow murmur), as seen in this patient. Increased blood flow through the tricuspid valve may also cause a diastolic rumble at the left lower sternal border (not present in this patient).

Increased blood flow across the pulmonic valve also accounts for the characteristic ASD finding of a **widely split, fixed S2**. Under normal conditions, splitting of S2 is heard only during inspiration when there is increased venous return to the right heart. This results in delayed pulmonic valve closure (P2) relative to aortic valve closure (A2) due to the increased time needed for right ventricular blood ejection. With ASD, right ventricular volume remains increased throughout the entire respiratory cycle, leading to a persistently delayed P2 with no apparent respiratory variation in splitting.

(Choice A) Aortic stenosis causes a crescendo-decrescendo systolic murmur over the right second intercostal space. A2 may be delayed until after P2 (paradoxical splitting) due to the increase in left ventricular ejection time.

(Choice B) Ventricular septal defects create a holosystolic murmur along the left lower sternal border due to a high-pressure, left-to-right shunt. Splitting of S2 may be widened but is usually not fixed. Fixed splitting is more typical of ASDs because atrial shunts reduce the normal variation in right ventricular volume during the respiratory cycle (flow through the ASD increases when venous return decreases, minimizing inspiratory/expiratory differences in right ventricular volume).

(Choice D) Aortic insufficiency is characterized by regurgitant blood flow through the aortic valve, resulting in a decrescendo diastolic murmur along the sternal border. Splitting of S2 is usually not affected.

Educational objective:

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Left atrial pressure is normally slightly higher than in the right atrium due to the greater compliance of the thin-walled right ventricle; as a result, ASDs cause **left-to-right shunting**. This blood flow has low velocity and turbulence and therefore does not create a murmur directly. Instead, the increased volume of blood delivered to the right heart (ie, normal venous return plus extra blood through the shunt) results in **increased blood flow** into the pulmonary arteries across the **pulmonic valve**. This creates the characteristic early-peaking **midsystolic ejection murmur** over the left upper sternal border (ie, pulmonic flow murmur), as seen in this patient. Increased blood flow through the tricuspid valve may also cause a diastolic rumble at the left lower sternal border (not present in this patient).

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(Choice D) Aortic insufficiency is characterized by regurgitant blood flow through the aortic valve, resulting in a decrescendo diastolic murmur along the sternal border. Splitting of S2 is usually not affected.

Educational objective:
An atrial septal defect leads to increased right-sided blood flow due to left-to-right shunting. Characteristic findings are an early-peaking midsystolic ejection murmur over the left upper sternal border (ie, pulmonic flow murmur) and wide and fixed splitting of S2.

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Early-peaking systolic ejection murmur
Fixed, widely split S2

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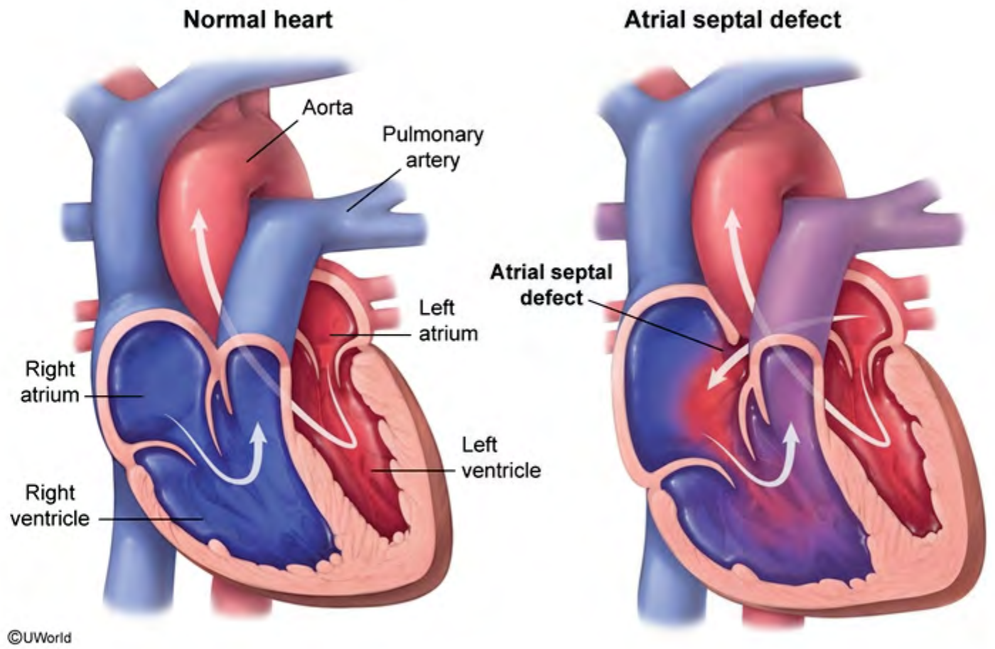

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Early-peaking systolic ejection murmur
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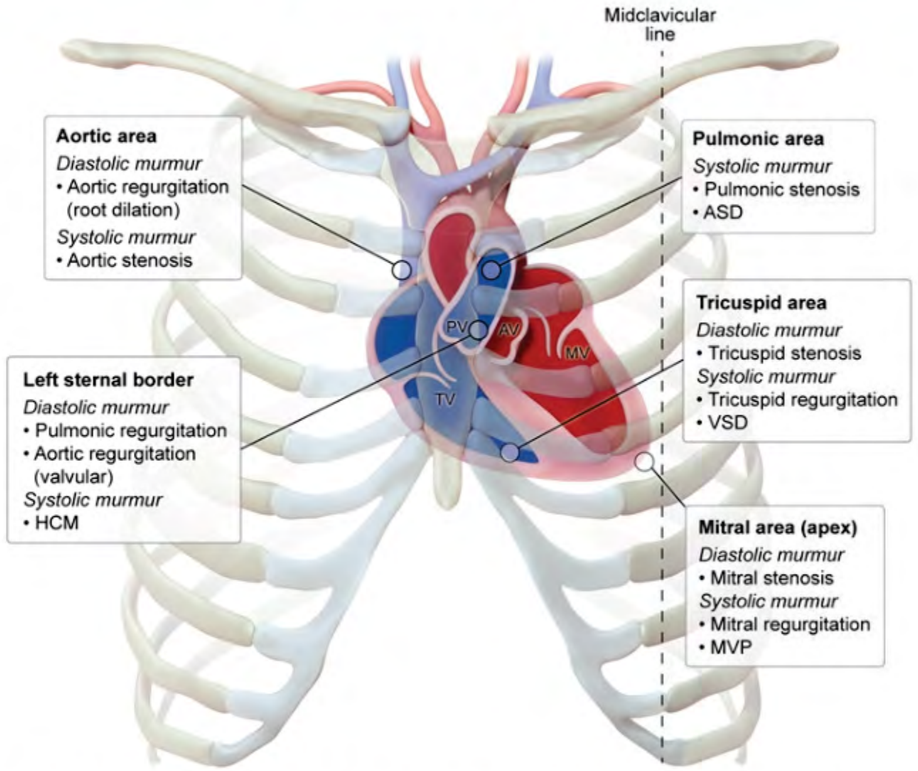
Cardiac auscultatory locations



ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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Early-peaking systolic ejection murmur Fixed, widely split S2

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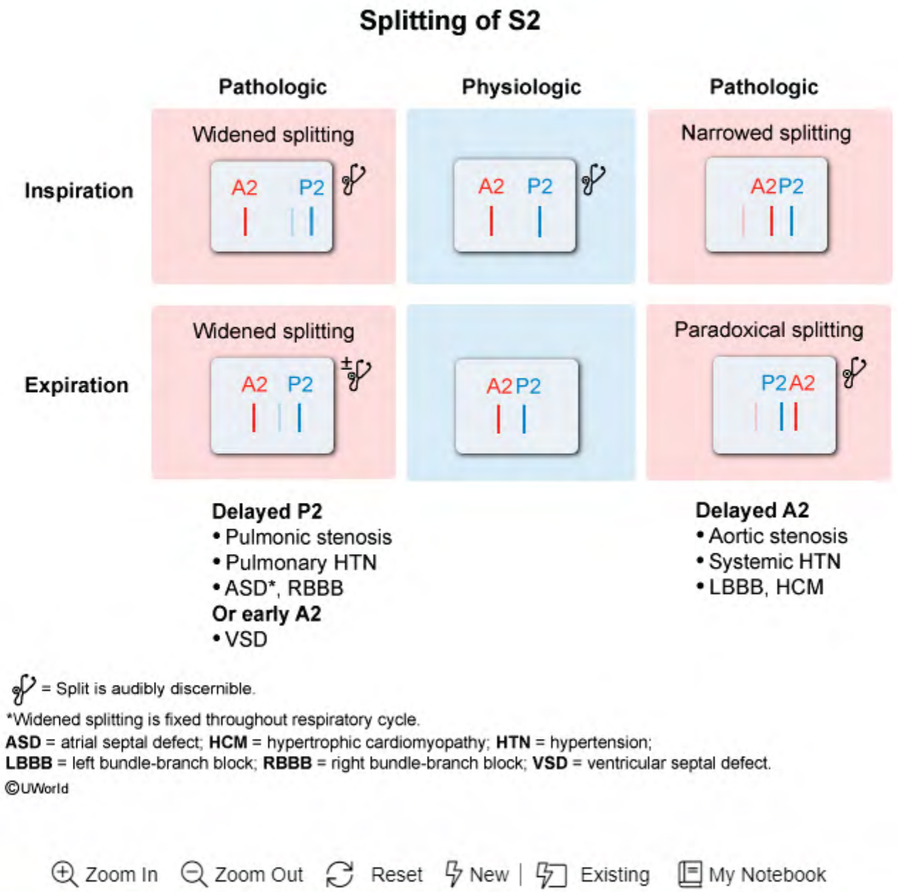

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Early-peaking systolic ejection murmur
Fixed, widely split S2

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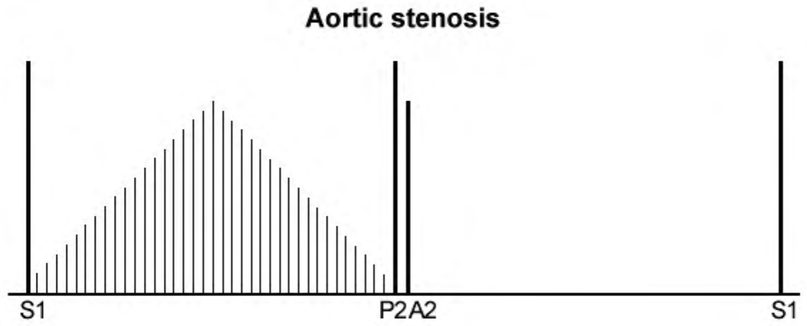

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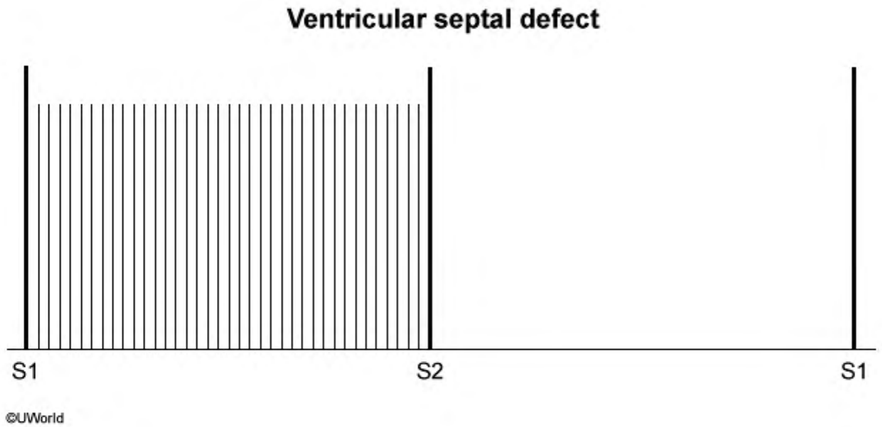

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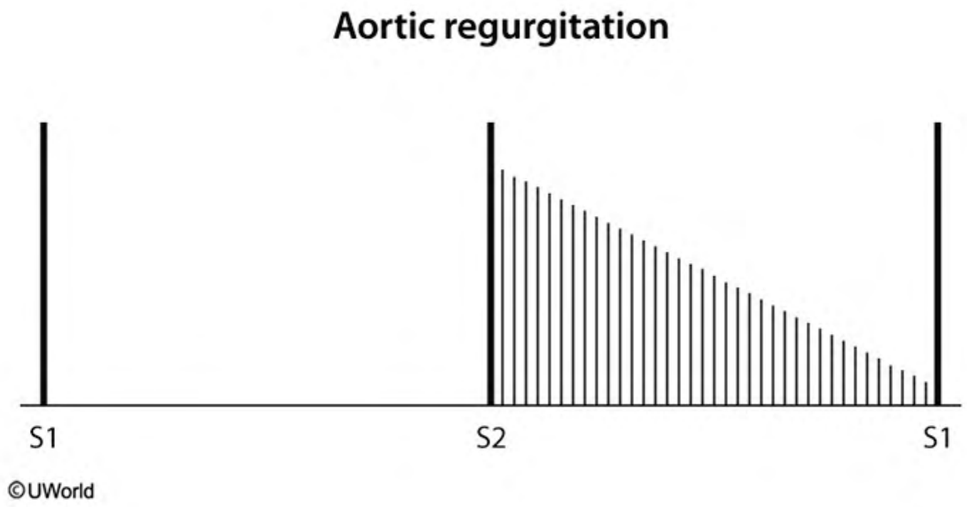

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A 65-year-old man comes to the emergency department with sudden onset of right-sided calf and foot pain. His past medical history is significant for hypertension, type II diabetes mellitus, atrial fibrillation and stable angina. Physical examination reveals paleness of the right leg and diminished right popliteal pulse. Immediate angiography is ordered that reveals an obstructive thrombus in the right common femoral artery. The thrombus extraction is followed by a rapid surge of serum creatine kinase level, which is best explained by:

- A. Cellular swelling
- B. Cell membrane damage
- C. Glutathione peroxidase production
- D. Mitochondrial vacuolization
- E. Nuclear shrinkage

Submit

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- A. Cellular swelling (6%)
- ✓ B. Cell membrane damage (70%)
- C. Glutathione peroxidase production (10%)
- D. Mitochondrial vacuolization (10%)
- E. Nuclear shrinkage (1%)

Correct | 70% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Ischemia is characterized by the reduction of blood flow, usually as a result of mechanical obstruction within the arterial system (eg, thrombus). If the flow of blood to the ischemic tissue is restored in a timely manner, those cells that were reversibly injured will typically recover. Sometimes, however, the cells within the damaged tissue will paradoxically die at an accelerated pace through apoptosis or necrosis after resumption of blood flow. This process is termed reperfusion injury, and is thought to occur secondary to one or more of the following mechanisms: 1) oxygen free radical generation by parenchymal cells, endothelial cells, and leukocytes; 2) severe, irreversible mitochondrial damage described as "mitochondrial permeability transition"; 3) inflammation, which attracts circulating neutrophils that cause additional injury; and 4) activation of the complement pathway, causing cell injury and further inflammation. When the cells within heart, brain, or skeletal muscle are injured, the enzyme creatine kinase leaks across the damaged cell membrane and into circulation (as seen in this patient)

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Ischemia is characterized by the reduction of blood flow, usually as a result of mechanical obstruction within the arterial system (eg, thrombus). If the flow of blood to the ischemic tissue is restored in a timely manner, those cells that were reversibly injured will typically recover. Sometimes, however, the cells within the damaged tissue will paradoxically die at an accelerated pace through apoptosis or necrosis after resumption of blood flow. This process is termed reperfusion injury, and is thought to occur secondary to one or more of the following mechanisms: 1) oxygen free radical generation by parenchymal cells, endothelial cells, and leukocytes; 2) severe, irreversible mitochondrial damage described as "mitochondrial permeability transition"; 3) inflammation, which attracts circulating neutrophils that cause additional injury; and 4) activation of the complement pathway, causing cell injury and further inflammation. When the cells within heart, brain, or skeletal muscle are injured, the enzyme creatine kinase leaks across the damaged cell membrane and into circulation (as seen in this patient).

(Choice A) Cellular swelling arises secondary to changes in ion concentration and the influx of water. This state is considered a hallmark of reversible injury, and is not directly associated with the leakage of intracellular proteins such as creatine kinase.

(Choice C) Glutathione peroxidase actually reduces cellular injury by catalyzing free radical breakdown. The presence of this enzyme is not responsible for the release of creatine kinase.

(Choice D) Mitochondrial vacuolization reduces the cellular capacity for ATP generation and is associated with irreversible injury. Creatine kinase release is not directly associated with this mitochondrial change, however.

(Choice E) Nuclear shrinkage (pyknosis), fragmentation, and dissolution characterize irreversible injury of the cell. Creatine kinase release is not directly associated with such nuclear changes, however.

Educational Objective:
Reperfusion injury is thought to occur secondary to oxygen free radical generation, mitochondrial damage, and inflammation.

Pathophysiology	Cardiovascular System	Peripheral vascular disease
Subject	System	Topic

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A 44-year-old woman comes to the office due to fatigue, nausea, and weight loss for the last 3-4 months. She also has noticed her freckles becoming darker and reports frequent dizziness upon standing. Temperature is 36.8 C (98.2 F), blood pressure is 120/80 mm Hg when sitting and 95/65 mm Hg when standing, and pulse is 77/min. Hyperpigmentation of the vermillion border of the lips is noted. The remainder of the physical examination is unremarkable. Comprehensive diagnostic workup reveals a diagnosis of idiopathic primary adrenal insufficiency. The patient is started on hydrocortisone and fludrocortisone. Fludrocortisone is most likely to cause which of the following effects in this patient?

	Vascular adrenergic sensitivity	Circulating blood volume	Urine sodium
○ A.	Increased	Decreased	Increased
○ B.	Increased	Unchanged	Decreased
○ C.	Increased	Increased	Decreased
○ D.	Unchanged	Increased	Decreased
○ E.	Decreased	Decreased	Increased
○ F.	Decreased	Increased	Unchanged

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	Vascular adrenergic sensitivity	Circulating blood volume	Urine sodium	
✗ A.	Increased	Decreased	Increased	(4%)
B.	Increased	Unchanged	Decreased	(11%)
✓ C.	Increased	Increased	Decreased	(48%)
D.	Unchanged	Increased	Decreased	(29%)
E.	Decreased	Decreased	Increased	(2%)
F.	Decreased	Increased	Unchanged	(3%)

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Explanation

This patient's fatigue, nausea, hyperpigmentation, and orthostatic hypotension are due to **primary adrenal insufficiency** (PAI). PAI results from reduced levels of both mineralocorticoid (mainly aldosterone) and

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Explanation

This patient's fatigue, nausea, hyperpigmentation, and orthostatic hypotension are due to **primary adrenal insufficiency** (PAI). PAI results from reduced levels of both mineralocorticoid (mainly aldosterone) and glucocorticoid (mainly cortisol), typically due to autoimmune destruction of the bilateral adrenal cortex. Orthostasis in PAI is due primarily to hypovolemia from reduced mineralocorticoid production.

Reduced mineralocorticoid activity in the renal collecting tubule principal cells leads to markedly decreased sodium absorption (ie, renal salt wasting) and increased potassium absorption. The decreased sodium reabsorption leads to decreased fluid retention, overall decreased circulating blood volume, and hypovolemia-induced orthostasis. **Fludrocortisone**, a synthetic adrenal steroid with high mineralocorticoid activity, is used to treat mineralocorticoid deficiency. It works by 2 primary mechanisms:

- Increased sodium reabsorption in the distal convoluted tubule and collecting ducts, causing increased fluid retention and circulating blood volume. This leads to **decreased urine sodium**.
- **Increase vascular sensitivity** to multiple endogenous vasoconstrictors (eg, angiotensin II, norepinephrine), causing a potentiation of their pressor effects **(Choices D, E, and F)**.

The **increased circulating plasma volume** and increased sensitivity to vasoconstricting mediators leads to an increase in blood pressure and improvement in orthostatic hypotension **(Choices A and B)**.

Educational objective:
Fludrocortisone improves hypovolemia and orthostatic hypotension in primary adrenal insufficiency by decreasing urine sodium excretion (thereby increasing circulating blood volume) and increasing the sensitivity of the vasculature to endogenous vasoconstrictors (eg, norepinephrine).

References

- Adrenal insufficiency – recognition and management.

Pharmacology	Cardiovascular System	Syncope
Subject	System	Topic

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Within 2-5 min of standing from a supine position	• ≥20 mm Hg ↓ in systolic blood pressure OR • ≥10 mm Hg ↓ in diastolic blood pressure

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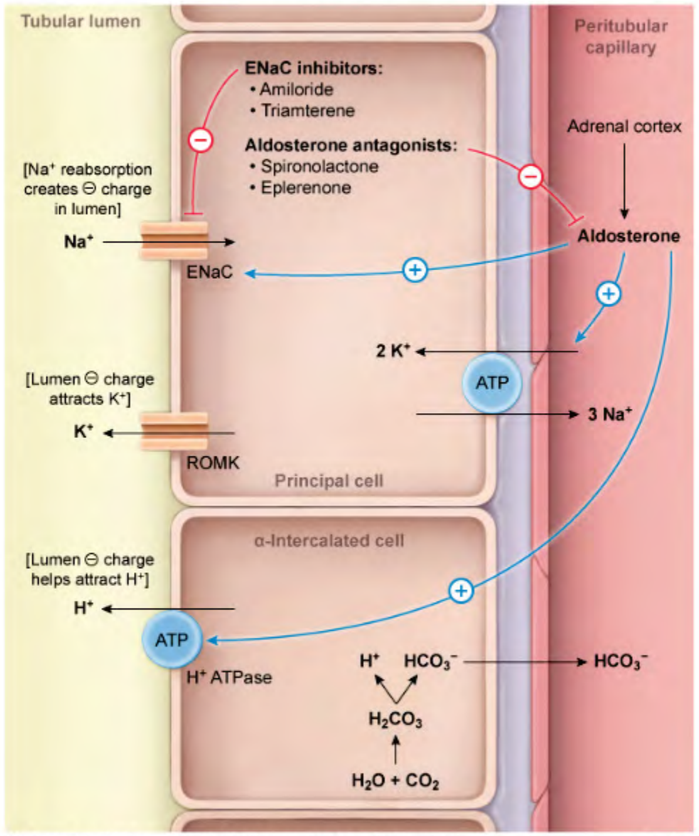
Action of aldosterone in the collecting duct of the nephron



ENaC = epithelial sodium channel; **ROMK** = renal outer medullary potassium channel. ©UWorld

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Pharmacology (Subject) | Cardiovascular System (System) | Syncope (Topic)

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A 32-year-old woman comes to the outpatient clinic due to shortness of breath for the past 6 months. The patient says she can walk only 3-4 blocks on level ground before feeling out of breath. She also has an occasional dry cough at night. The patient denies any chest pain, palpitations, syncope, weight loss, or lower extremity swelling. She immigrated from Guatemala 8 years ago. Medical history is unremarkable. On physical examination, the patient appears comfortable. Blood pressure is 128/75 mm Hg and pulse is 78/min and regular. Peripheral pulses are full and symmetric. The lungs are clear on auscultation. S1 is loud. With the patient positioned on her left side and breath held at the end of expiration, a diastolic murmur is heard at mid- and end diastole. Otherwise, examination is unremarkable. Which of the following are the most likely left ventricular hemodynamic findings at rest in this patient?

	Diastolic pressure	Afterload	Contractility
○ A.	Increased	Increased	Decreased
○ B.	Increased	Increased	Normal
○ C.	Normal	Normal	Decreased
○ D.	Normal	Normal	Normal

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		Diastolic pressure	Afterload	Contractility	
✗	A.	Increased	Increased	Decreased	(19%)
	B.	Increased	Increased	Normal	(31%)
	C.	Normal	Normal	Decreased	(13%)
✓	D.	Normal	Normal	Normal	(35%)

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Correct answer
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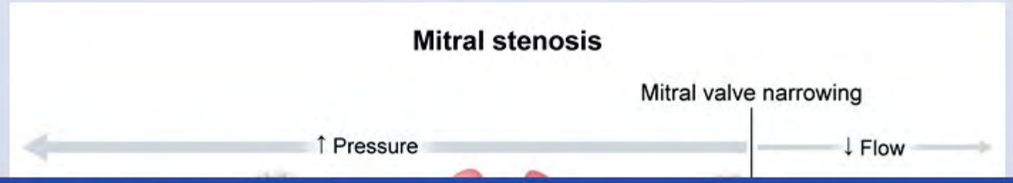
Explanation



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Correct answer
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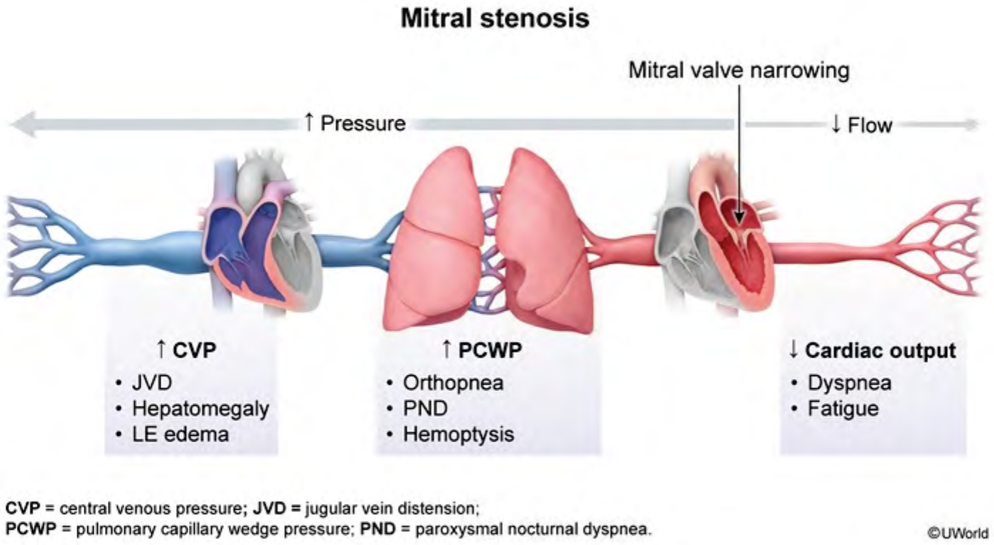

CVP = central venous pressure; **JVD** = jugular vein distension;
PCWP = pulmonary capillary wedge pressure; **PND** = paroxysmal nocturnal dyspnea.

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This patient's presentation with exertional dyspnea in the **absence of volume overload** (ie, dry cough, no pulmonary crackles, no peripheral edema) suggests relatively **early, mild-to-moderate** disease. A **loud S1**

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CVP = central venous pressure; **JVD** = jugular vein distension;
PCWP = pulmonary capillary wedge pressure; **PND** = paroxysmal nocturnal dyspnea.

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This patient's **mid-to-late diastolic murmur** is consistent with **mitral stenosis** (MS). MS usually occurs due to underlying rheumatic heart disease, which in the United States is most commonly seen in patients who emigrated from Latin America, Africa, or Asia.

MS can lead to heart failure due to restriction of left ventricular (LV) diastolic filling. Patients usually experience **exertional dyspnea** as the first symptom, which results from an inability to increase cardiac output to meet exertional demands. As MS worsens, elevated left atrial pressure can lead to orthopnea, paroxysmal nocturnal dyspnea, productive cough/hemoptysis, and eventual right-sided heart failure with resulting jugular venous distension and lower extremity edema.

This patient's presentation with exertional dyspnea in the **absence of volume overload** (ie, dry cough, no pulmonary crackles, no peripheral edema) suggests relatively **early, mild-to-moderate** disease. A **loud S1** (caused by the stiff leaflets snapping shut) is further suggestive of more mild disease, as the intensity of S1 diminishes with disease progression due to heavy calcification that limits valve motion. Although severe MS can cause reductions in **LV diastolic pressure** and **afterload** (due to reduced ventricular filling), these parameters typically remain normal until later in the disease course (especially at rest). **LV contractility** is also **normal** at rest in early disease; however, with severe MS the reduction in cardiac output stimulates increased sympathetic activity to increase LV contractility.

(Choices A and C) Decreased LV contractility occurs in heart failure due to LV systolic dysfunction (eg, dilated cardiomyopathy, ischemic heart disease). LV diastolic pressure and afterload are initially near normal but increase over time due to worsening systolic function and progressive ventricular dilation.

(Choice B) Increased LV diastolic pressure in the setting of normal contractility occurs in heart failure due to LV diastolic dysfunction. Depending on etiology, afterload may be increased (eg, early stages of hypertensive heart disease) or normal (eg, cardiac amyloidosis).

Educational objective:
Mitral stenosis usually occurs due to underlying rheumatic heart disease and initially presents with exertional dyspnea. Orthopnea, paroxysmal nocturnal dyspnea, productive cough, and hemoptysis can develop as the disease becomes more advanced. At rest, left ventricular diastolic pressure, afterload, and contractility remain

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Educational objective:
Mitral stenosis usually occurs due to underlying rheumatic heart disease and initially presents with exertional dyspnea. Orthopnea, paroxysmal nocturnal dyspnea, productive cough, and hemoptysis can develop as the disease becomes more advanced. At rest, left ventricular diastolic pressure, afterload, and contractility remain normal until the mitral stenosis is severe.

References

- Mitral stenosis

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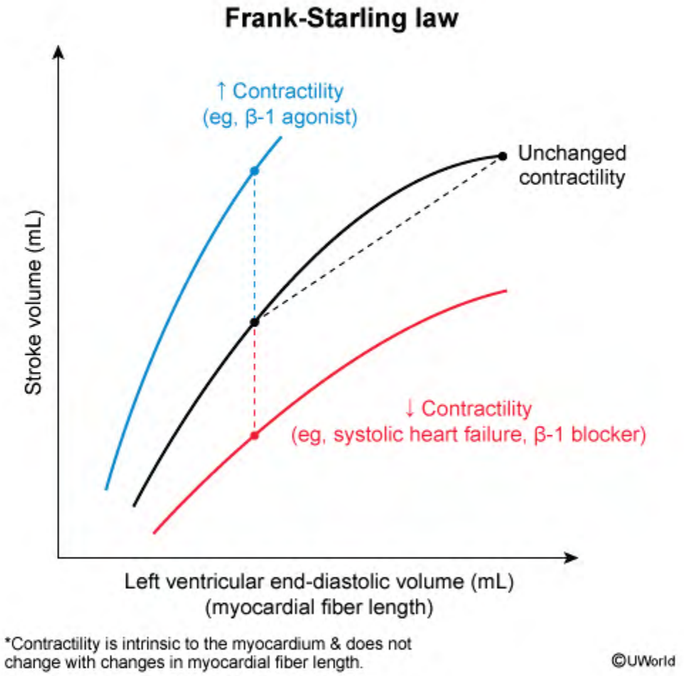

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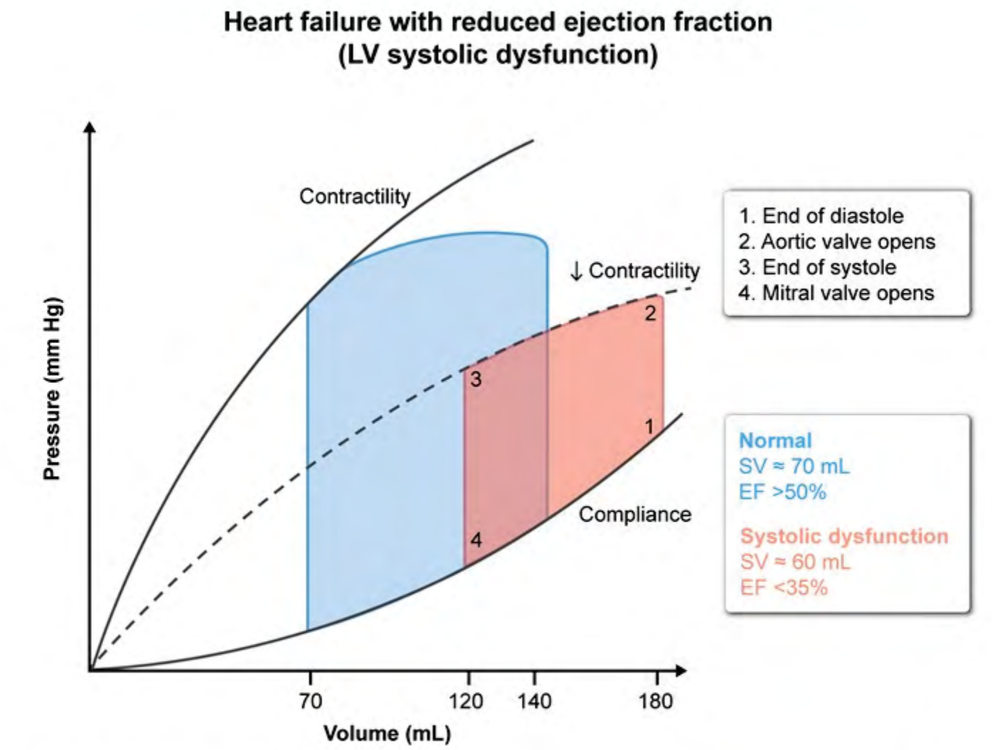

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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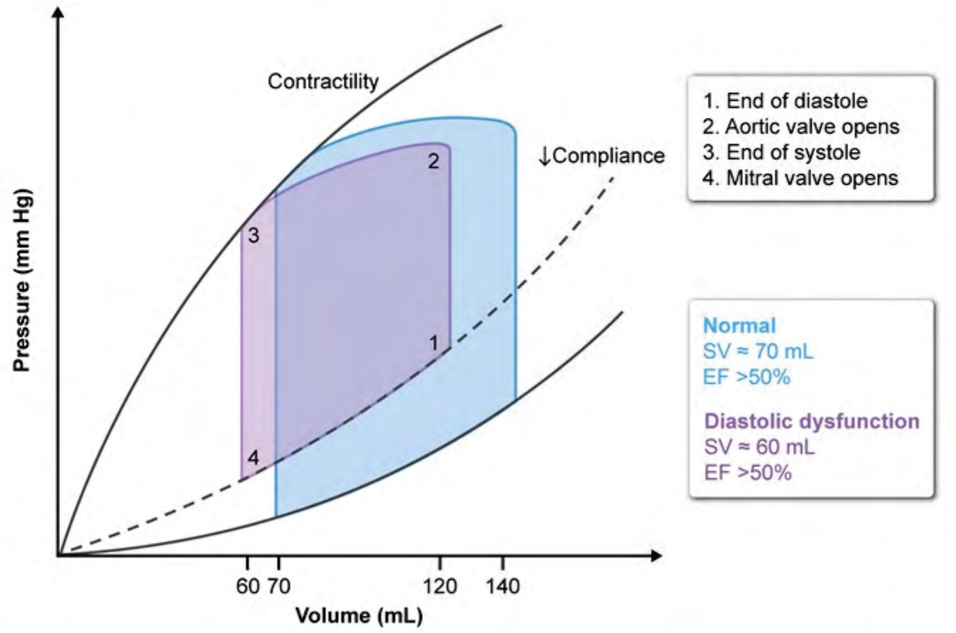

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A 47-year-old man comes to the office for follow-up of his elevated blood pressure. The patient's medical history is significant for stable angina pectoris, for which he takes sublingual nitroglycerin as needed. He has no orthopnea, paroxysmal nocturnal dyspnea, or lower extremity swelling. His blood pressure is 154/107 mm Hg and pulse is 86/min. Physical examination shows no abnormalities. The patient is started on a low dose of atenolol. Which of the following cellular changes will most likely occur as a direct effect of this medication?

	Cardiomyocyte [cAMP]	Juxtaglomerular cell [cAMP]	Vascular smooth muscle [cAMP]
○ A.	Decreased	Decreased	Decreased
○ B.	Decreased	Decreased	No significant change
○ C.	Decreased	No significant change	Decreased
○ D.	Decreased	No significant change	No significant change
○ E.	No significant change	No significant change	No significant change

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A.	Decreased	Decreased	Decreased	(13%)
✓ B.	Decreased	Decreased	No significant change	(54%)
C.	Decreased	No significant change	Decreased	(13%)
D.	Decreased	No significant change	No significant change	(17%)
E.	No significant change	No significant change	No significant change	(1%)

Correct

54% Answered correctly

03 secs Time Spent

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Explanation

G protein–coupled receptors & their signal transducers		
Adrenergic	α_1	G_q
	α_2	G_i
	β_1	G_s
	β_2	G_s

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Explanation

G protein–coupled receptors & their signal transducers		
Adrenergic	α_1	G_q
	α_2	G_i
	β_1	G_s
	β_2	G_s
Cholinergic	M_1	G_q
	M_2	G_i
	M_3	G_q
Dopaminergic	D_1	G_s
	D_2	G_i

Beta blockers are the preferred treatment for patients with chronic stable angina and hypertension. They relieve anginal symptoms by inhibiting sympathetic stimulation of the heart, reducing both heart rate and contractility. Because beta blockers reduce heart rate and blood pressure during rest and exercise, the onset of angina is avoided or delayed.

At low doses, **atenolol** is a **selective β1 adrenergic antagonist**. β_1 receptors are found in **cardiac tissue** and on **renal juxtaglomerular cells**, but not in vascular smooth muscle. The β_1 receptor is a G protein-coupled receptor (GPCR) associated with **Gs**, which increases intracellular cAMP levels. Blockade of the β_1 receptor leads to **decreased cAMP levels** in cardiac and renal tissue without significantly affecting cAMP levels in vascular smooth muscle **(Choices C and D)**.

GPCRs comprise a large family of 7 transmembrane domain receptors that activate intracellular second messenger systems. There are 2 principal second messenger systems involving GPCRs:

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	D_2	G_i

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GPCRs comprise a large family of 7 transmembrane domain receptors that activate intracellular second messenger systems. There are 2 principal second messenger systems involving GPCRs:

1. cAMP signaling pathway: The activated G_s α subunit activates the enzyme adenylyl cyclase, which catalyzes the conversion of ATP into cAMP. Elevated cAMP leads to the activation of protein kinase A. Conversely, the G_i α subunit inhibits adenylyl cyclase, thereby reducing cAMP levels.
2. Phosphatidylinositol signaling pathway: The activated G_q α subunit activates phospholipase C, which degrades membrane lipids into diacylglycerol (DAG) and inositol triphosphate (IP3). Protein kinase C is activated by DAG, and calcium is released from the sarcoplasmic reticulum under the influence of IP3.

(Choice A) Propranolol is a nonselective beta blocker that blocks both β1 and β2 adrenergic receptors. β2 receptors are found in vascular and bronchial smooth muscle. Blockade of β1and β2 receptors would therefore result in decreased cAMP levels in cardiac myocytes, renal juxtaglomerular cells, and vascular smooth muscle.

(Choice E) A medication such as diltiazem, a non-dihydropyridine calcium channel blocker, would not directly affect cAMP levels.

Educational objective:

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Educational objective:
β_1 adrenergic receptors are found in cardiac tissue and on renal juxtaglomerular cells, but not in vascular smooth muscle. Selective blockade of the β_1 receptor (eg, with atenolol) leads to decreased cAMP levels in cardiac and renal tissue without significantly affecting cAMP levels in vascular smooth muscle.

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	Dopaminergic	D_1	G_s

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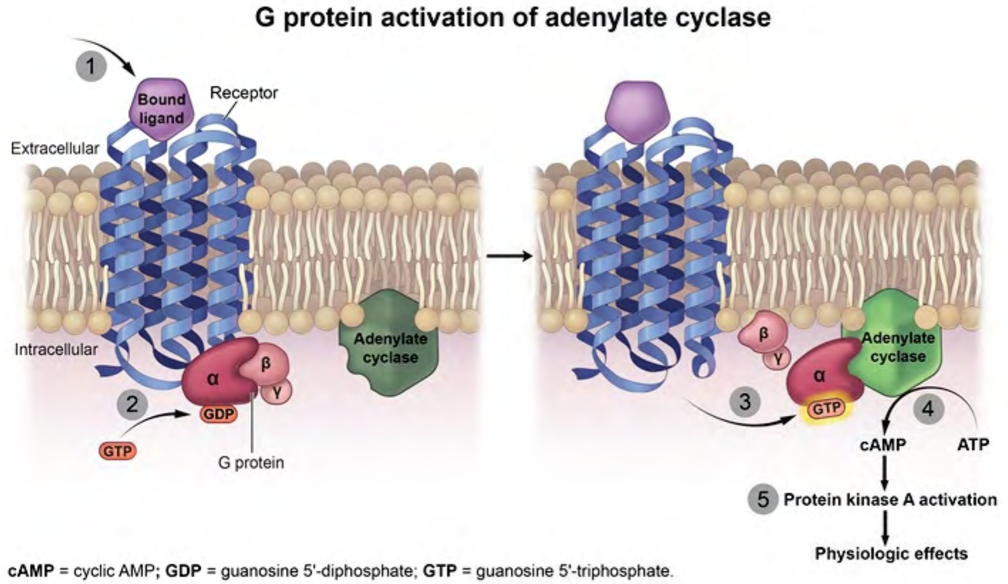

cAMP = cyclic AMP; **GDP** = guanosine 5'-diphosphate; **GTP** = guanosine 5'-triphosphate.

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Educational objective:

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A 54-year-old woman comes to the office due to exertional dyspnea and fatigue. The patient gets short of breath while walking to a store 2 blocks away from her home. She also has trouble breathing at night and sleeps with 2 or 3 pillows to prop her up. She has had occasional palpitations but no chest pain. The patient takes no medications on a regular basis, and does not use tobacco, alcohol, or illicit drugs. After initial evaluation, she is scheduled for a left heart catheterization, the findings of which are shown below.

Which of the following is the most likely diagnosis?

- A. Aortic regurgitation
- B. Aortic stenosis
- C. Mitral regurgitation
- D. Mitral stenosis
- E. Tricuspid regurgitation

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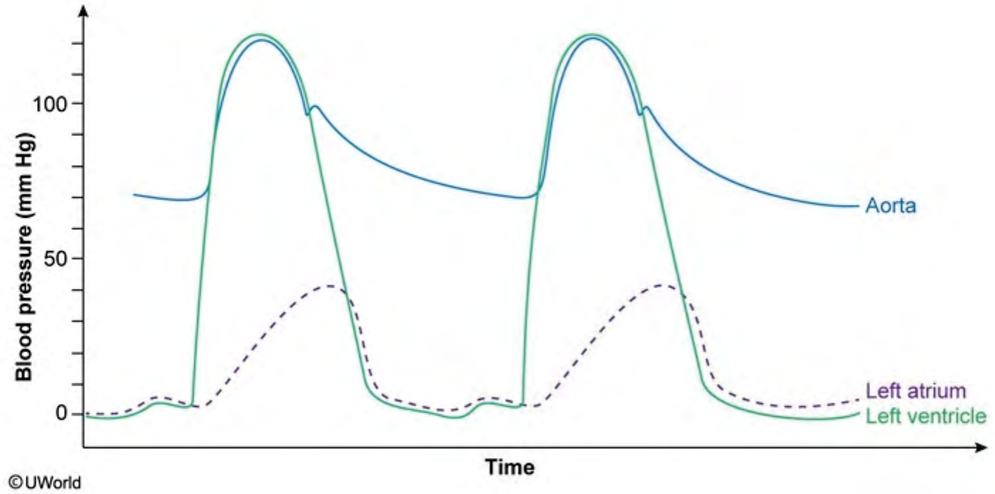

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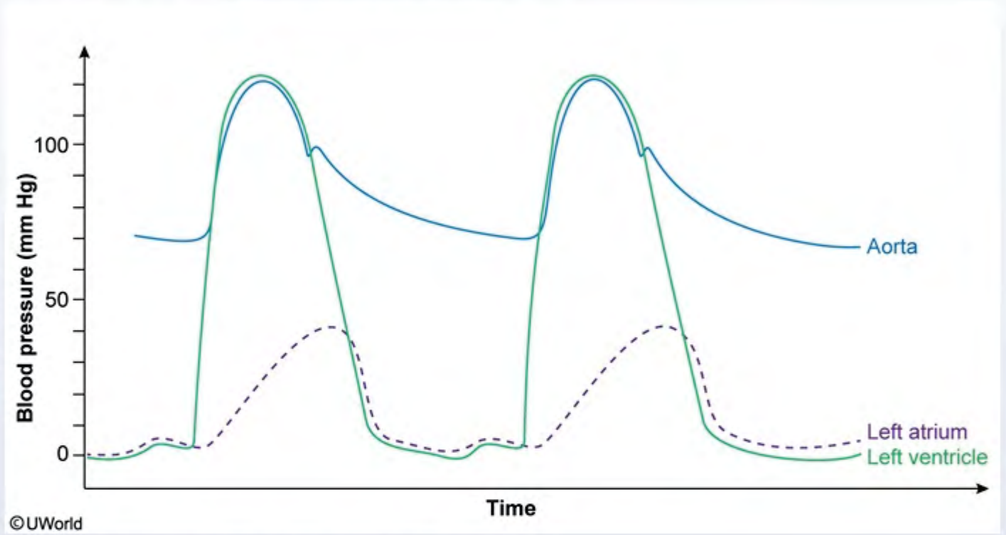

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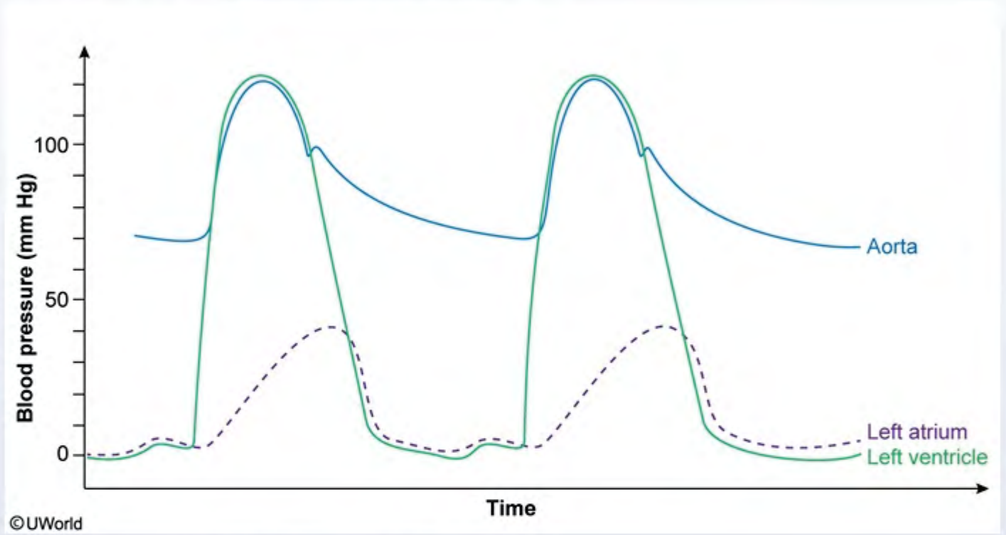

Which of the following is the most likely diagnosis?

- A. Aortic regurgitation (6%)
- ✗ B. Aortic stenosis (5%)
- ✓ C. Mitral regurgitation (66%)
- D. Mitral stenosis (19%)
- E. Tricuspid regurgitation (1%)

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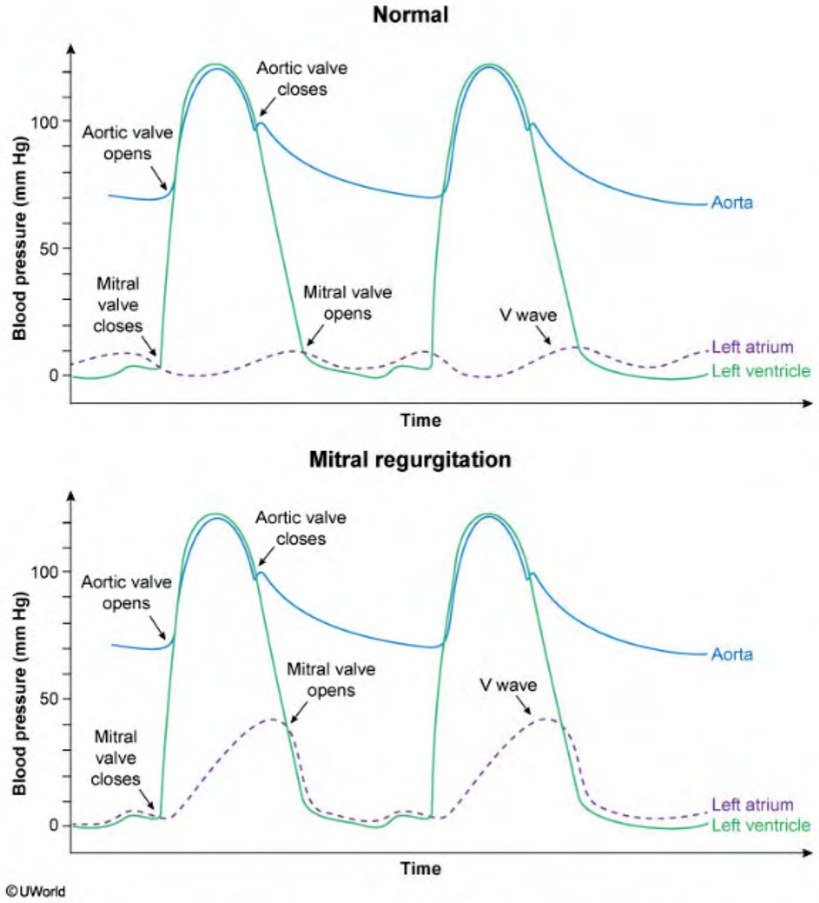

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This patient's left heart catheterization shows pressure tracings consistent with **mitral regurgitation** (MR). In the cardiac cycle, passive filling of the left atrium with blood from the pulmonary veins (atrial diastole) occurs essentially in sync with left ventricular (LV) systole. Left atrial pressure gradually increases to approximately 10 mm Hg in the normal heart with a characteristic "V wave" created at the end of passive filling. However, in the setting of MR, an incompetent mitral valve allows backflow of blood from the left ventricle during ventricular systole, leading to markedly **elevated left atrial pressure** during this time. This creates an **early and large V wave**, the characteristic pressure tracing finding in patients with MR.

The elevated left atrial pressure in MR leads to atrial dilation and often precipitates atrial fibrillation, symptomatically manifesting with palpitations. Over time, decompensated heart failure, evidenced by dyspnea and orthopnea in this patient, can also occur.

(Choice A) In aortic regurgitation, backflow of blood from the aorta into the left ventricle during diastole results in rapid loss of aortic pressure and elevated LV end-diastolic volume with a compensatory increase in LV stroke volume. Pressure tracings show loss of the aortic dicrotic notch (normally seen around aortic valve closure), rapid decline of aortic pressure during diastole, and elevated peak LV and aortic systolic pressures. There is also early closure of the mitral valve due to elevated LV diastolic pressure.

(Choice B) Aortic stenosis causes obstruction of LV outflow resulting in discordance of LV and aortic systolic pressure. Pressure tracings characteristically show peak LV systolic pressure significantly higher than aortic systolic pressure.

(Choice D) Mitral stenosis causes elevated left atrial pressures throughout the cardiac cycle with peak atrial pressure occurring at the time of atrial kick, just before the mitral valve closes at the end of ventricular diastole.

(Choice E) Tricuspid regurgitation causes an early and large V wave on right atrial pressure tracing, similar to that seen on left atrial pressure tracing in the setting of MR. Left-sided pressure tracings (left atrial, left ventricular, aortic) are minimally affected in tricuspid regurgitation.

Educational objective:
Left ventricular systole corresponds to the time of passive filling of the left atrium (atrial diastole). Mitral valve regurgitation leads to markedly elevated left atrial pressure during this period, creating the characteristic early and large V wave on left atrial pressure tracing.

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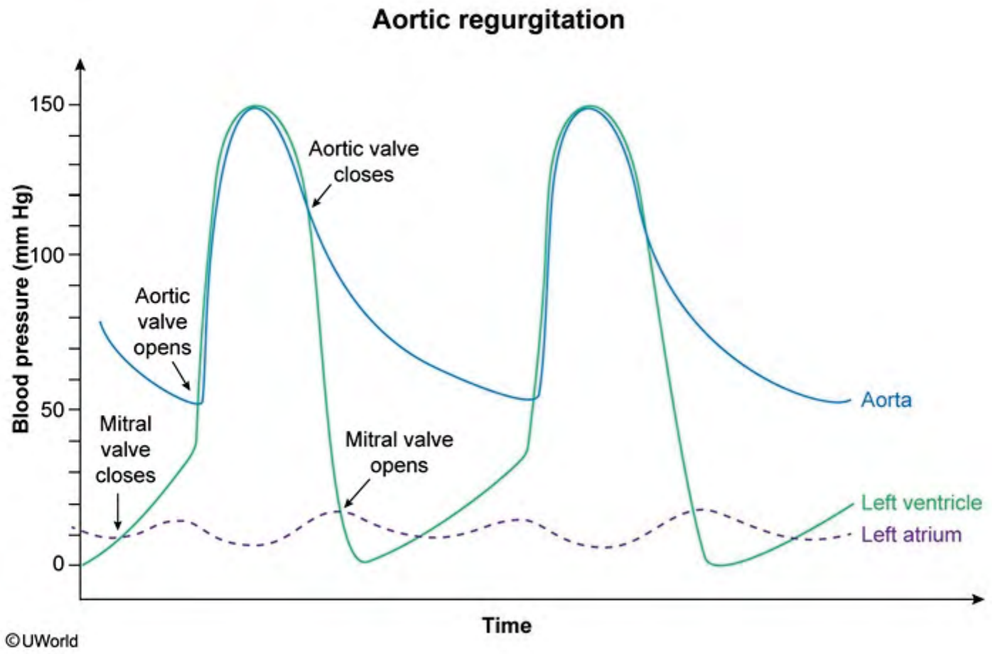

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regurgitation leads to markedly elevated left atrial pressure during this period, creating the characteristic early and large V wave on left atrial pressure tracing.

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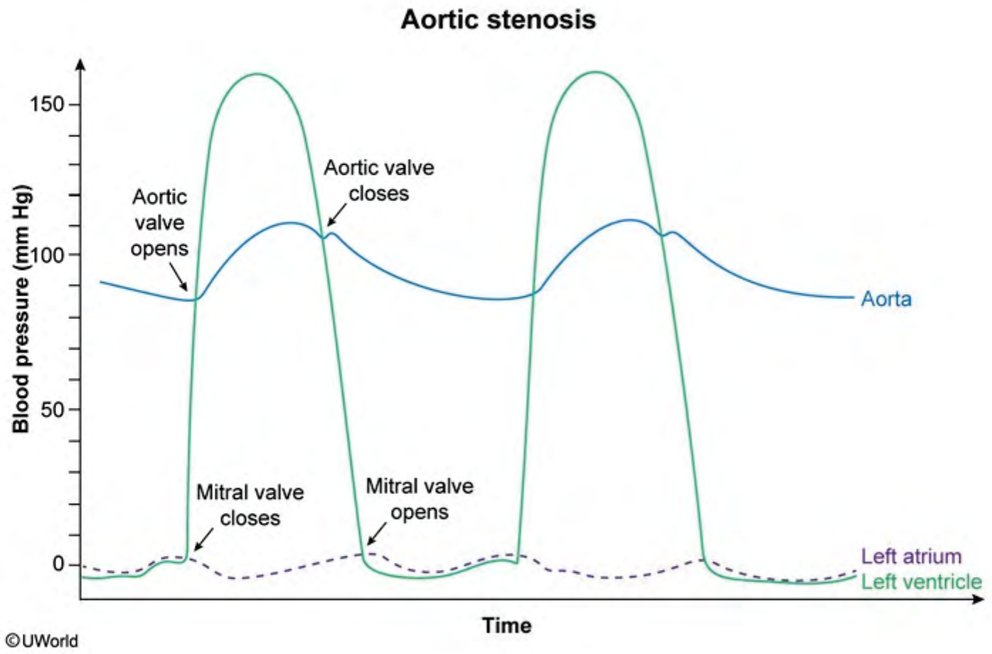

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This patient's left heart catheterization shows pressure tracings consistent with **mitral regurgitation** (MR). In the cardiac cycle, passive filling of the left atrium with blood from the pulmonary veins (atrial diastole) occurs essen
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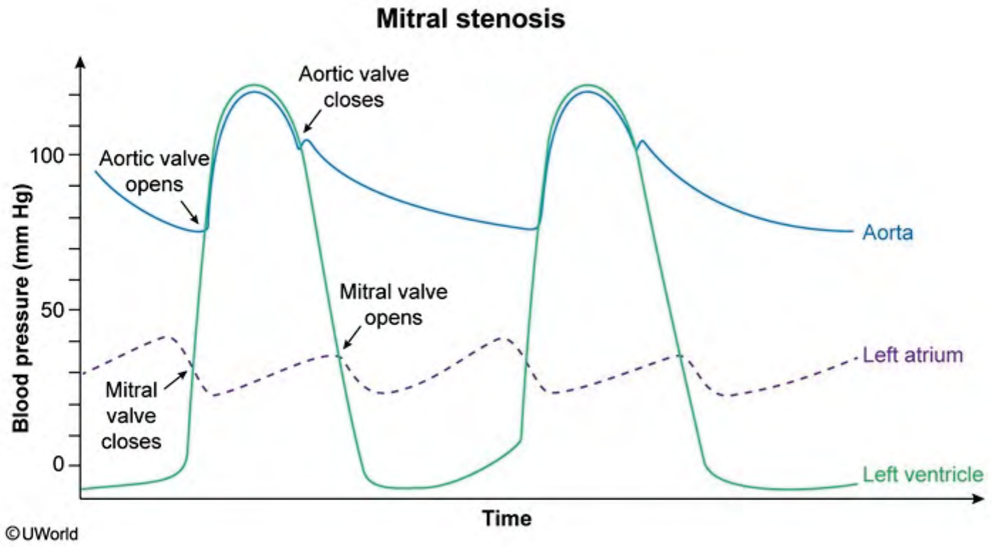

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A 25-year-old woman comes to the office with a 3-month history of fatigue, myalgias, arthralgias, and a 5-kg (11-lb) weight loss. She has also recently experienced increased leg pain with physical activity, which resolves with rest. The patient has no other medical conditions and takes no medications. Temperature is 37.6 C (99.7 F), and blood pressure is 160/90 mm Hg in the right arm and 120/80 mm Hg in the left. A bruit is heard over the left subclavian artery. Left radial pulse and dorsalis pedis pulses in both legs are weak. The remainder of the examination shows no abnormalities. Laboratory results reveal a hemoglobin level of 9.8 g/dL and an erythrocyte sedimentation rate of 110 mm/hr. Which of the following pathologic mechanisms most likely underlies this patient's current condition?

- A. Exaggerated vascular smooth muscle contraction
- B. Granulomatous inflammation of large arteries
- C. Lipid-rich deposits in large- and medium-sized arteries
- D. Necrotizing inflammation of medium-sized arteries
- E. Segmental inflammation of medium-sized arteries and veins

Submit

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- ✗ A. Exaggerated vascular smooth muscle contraction (5%)
- ✓ B. Granulomatous inflammation of large arteries (54%)
- C. Lipid-rich deposits in large- and medium-sized arteries (8%)
- D. Necrotizing inflammation of medium-sized arteries (12%)
- E. Segmental inflammation of medium-sized arteries and veins (18%)

Incorrect
Correct answer
B

54%
Answered correctly

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Time Spent

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Explanation

Takayasu arteritis	
Risk factors	• **Female** • Asian • Age <40
Clinical/laboratory	• **Constitutional**: fever, weight loss, fatigue • **Arterio-occlusive**: claudication, BP discrepancies, bruits, pulse deficits

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Takayasu arteritis	
Risk factors	• **Female** • Asian • Age <40
Clinical/laboratory findings	• **Constitutional**: fever, weight loss, fatigue • **Arterio-occlusive**: claudication, BP discrepancies, bruits, pulse deficits • Visual & neurologic deficits • Arthralgias & myalgias • Elevated inflammatory markers (eg, CRP, ESR)
Histopathology	• Granulomatous inflammation of arterial media • Transmural fibrous thickening, narrowing of lumen • Predominantly affects aorta & its branches
BP = blood pressure; **CRP** = C-reactive protein; **ESR** = erythrocyte sedimentation rate.	

This patient with lower extremity claudication (ie, exertional pain due to limited blood flow reserve) and constitutional symptoms has typical features of **Takayasu arteritis** (TA). TA is a chronic, large-artery vasculitis that primarily involves the **aorta and its branches**. TA predominantly affects women of reproductive age and is more common in Asians.

Arterial narrowing in TA can manifest as audible bruits, blood pressure discrepancies, pulse deficits, and distal ulcerations. Characteristic histopathologic findings include mononuclear infiltrates and **granulomatous inflammation** of the vascular media, leading to arterial wall thickening and occlusion. The pathologic abnormalities in TA closely resemble giant cell (temporal) arteritis; however, TA primarily affects **younger individuals**, whereas giant cell arteritis occurs almost exclusively in patients age ≥50.

(Choice A) Exaggerated vascular smooth muscle contraction (eg, in response to cold or vibration) causes Raynaud phenomenon. Symptoms are typically acute and episodic and manifest as a triphasic color change

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common in Asians.

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(Choice A) Exaggerated vascular smooth muscle contraction (eg, in response to cold or vibration) causes Raynaud phenomenon. Symptoms are typically acute and episodic and manifest as a triphasic color change (pallor, cyanosis, erythema) in the distal extremities. This patient's progressive symptoms and proximal arterial findings are more consistent with TA.

(Choice C) Atherosclerosis is characterized by lipid-rich deposits in large- and medium-sized arteries. It is common in older patients with cardiovascular risk factors (eg, smoking, hypertension, hyperlipidemia) but is rare in individuals as young as this patient. In addition, in contrast to this patient who has both upper and lower extremity abnormalities, upper extremity atherosclerosis is usually a late finding.

(Choice D) Necrotizing inflammation of medium-sized arteries is seen in polyarteritis nodosa, which is characterized by multisystem involvement (eg, renal insufficiency, skin lesions, neuropathy, bowel ischemia, myositis). This patient's bruits, blood pressure discrepancies, and pulse deficits indicate a large artery disorder.

(Choice E) Thromboangiitis obliterans (Buerger disease) is a segmental, thrombosing vasculitis of medium- and small-sized arteries, principally the tibial and radial arteries. Manifestations are most noticeable in the distal extremities (eg, ulcerations), and the condition is almost always seen in association with heavy cigarette smoking.

Educational objective:
Takayasu arteritis is a chronic, large-artery vasculitis that primarily involves the aorta and its branches. It presents with constitutional (eg, fever, weight loss) and arterio-occlusive (eg, claudication, blood pressure discrepancies, pulse deficits) findings in patients age <40. Histopathology shows granulomatous inflammation of the vascular media.

Pathology	Cardiovascular System	Takayasu arteritis
Subject	System	Topic

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common in Asians.

Arterial narrowing in TA can manifest as audible bruits, blood pressure discrepancies, pulse deficits, and distal ulcera
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Pathology | Cardiovascular System | Takayasu arteritis

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Raynaud phenomenon

*Due to vasoconstriction

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common in Asians.

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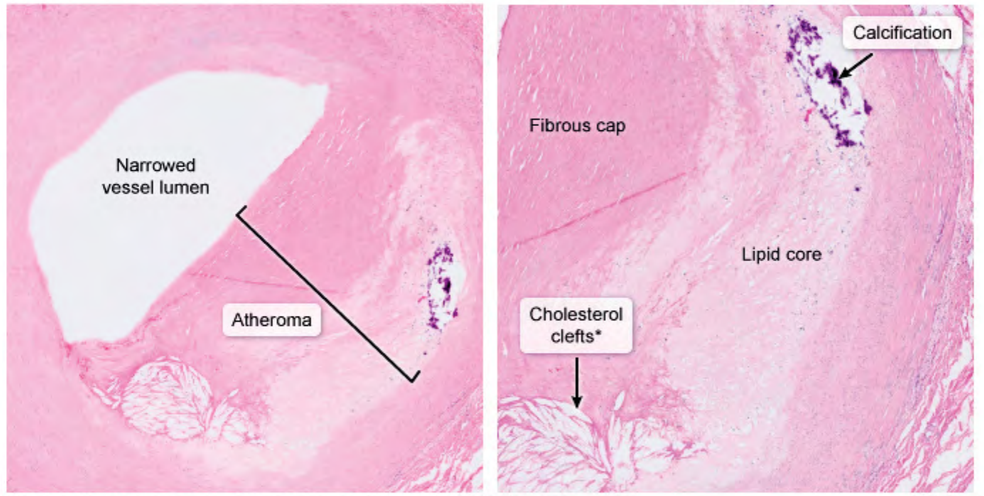
Atherosclerosis



*Cholesterol dissolves during tissue processing, leaving empty needle-shaped clefts.

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Pathology (Subject) | Cardiovascular System (System) | Takayasu arteritis (Topic)

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A 58-year-old man with a history of hypertension and hyperlipidemia comes to the emergency department due to substernal chest pain that began 6 hours ago and has gradually worsened. Blood pressure is 104/72 mm Hg and pulse is 88/min. ECG shows normal sinus rhythm with ST-segment depression and T-wave inversion, and troponin levels are elevated. The patient is diagnosed with non–ST-segment elevation myocardial infarction and admitted to the hospital for medical management, with coronary angiography scheduled the following morning. That evening, telemetry monitoring shows a new Mobitz type 1 second-degree atrioventricular block. Which of the following arteries was most likely obstructed during this patient's myocardial infarction?

- A. Left anterior descending
- B. Left circumflex
- C. Left main coronary
- D. Ramus intermedius
- E. Right coronary

Submit

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- ✗ A. Left anterior descending (17%)
- B. Left circumflex (5%)
- C. Left main coronary (2%)
- D. Ramus intermedius (1%)
- ✓ E. Right coronary (72%)

Incorrect
Correct answer
E

72%
Answered correctly

03 secs
Time Spent

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Version

Explanation

Typical blood supply to the cardiac conduction system

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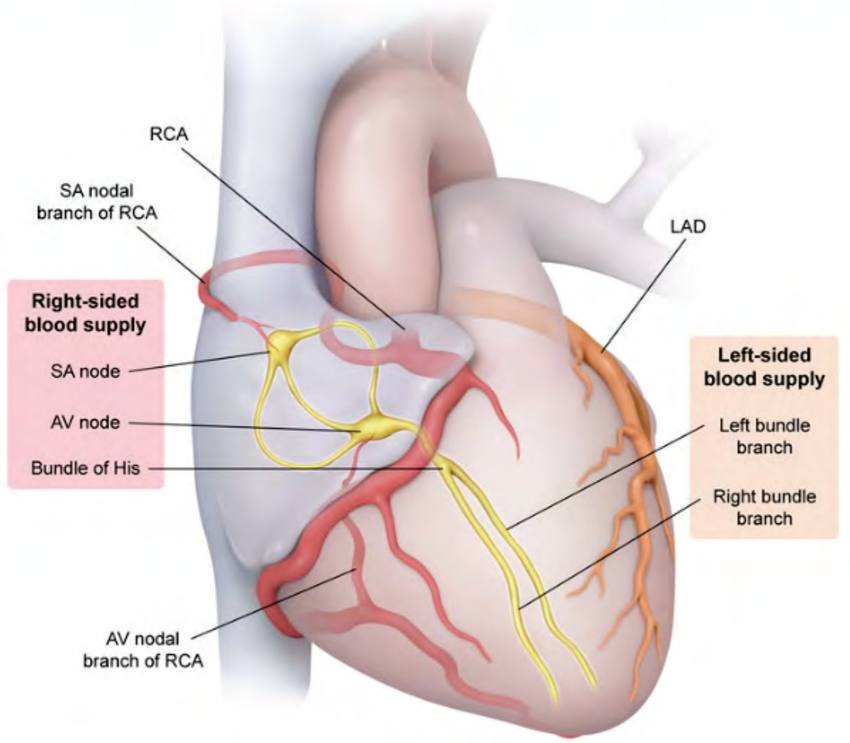
✓ E. Right coronary (72%)

Exhibit Display

Typical blood supply to the cardiac conduction system



AV = atrioventricular; **LAD** = left anterior descending artery; **RCA** = right coronary artery; **SA** = sinoatrial.

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AV nodal branch of RCA

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AV = atrioventricular; LAD = left anterior descending artery; RCA = right coronary artery; SA = sinoatrial. ©UWorld

Low-degree atrioventricular (AV) block, including first-degree and Mobitz type I second-degree, usually involves conduction disruption **within the AV node**. This patient's development of Mobitz type I second-degree AV block in the setting of acute **myocardial infarction** (MI) suggests obstruction of the **right coronary artery** (RCA). In approximately 90% of patients, the **RCA supplies blood to the AV node** while coursing around the right side of the heart to perfuse the right ventricle and inferior portion of the left ventricle. Because the RCA also usually supplies blood to the sinoatrial node, sinus bradycardia is also commonly seen with MI involving the RCA.

In contrast, the left anterior descending (LAD) artery **(Choice A)**, which courses down the anterior left ventricle, typically supplies blood to the left and right bundle branches and sometimes the bundle of His. MI involving the LAD can lead to high-degree AV block (ie, Mobitz type II second-degree, infranodal third-degree) involving conduction disruption below the AV node. Because the LAD does not supply blood to the AV node, low-degree AV block is not typically seen.

(Choices B and C) The left main coronary artery bifurcates into the LAD artery and the left circumflex artery, which courses around the left side of the heart to supply the lateral left ventricle. The left main coronary artery does not directly supply blood to the conduction system. The left circumflex artery frequently supplies blood to the sinoatrial node after coursing around to the back of the heart, but only supplies the AV node in about 10% of patients (ie, those with left-dominant coronary circulation).

(Choice D) Present in approximately 20% of the population, a ramus intermedius is a third branch off of the left main coronary artery. It originates at the bifurcation of the LAD and left circumflex arteries and courses between them to supply the anterolateral left ventricular wall. It does not supply blood to the conduction system.

Educational objective:
Low-grade atrioventricular block (ie, first-degree, Mobitz type I second-degree) involves conduction disruption within the AV node. The occurrence of low-grade AV block in the setting of myocardial infarction suggests obstruction of the right coronary artery because it supplies blood to the AV node in approximately 90% of patients.

Anatomy	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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AV = atrioventricular; LAD = left anterior descending artery; RCA = right coronary artery; SA = sinoatrial. ©UWorld

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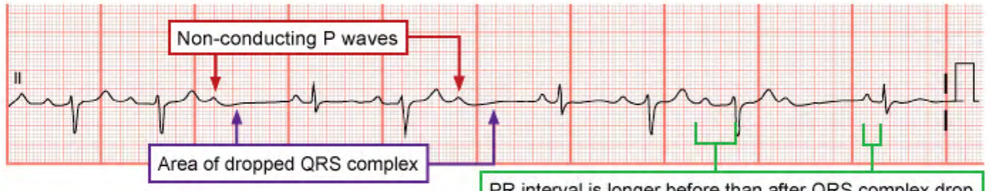
Mobitz Type 1 Second degree AV block

Progressive PR interval lengthening leading to a drop of a QRS complex after a non-conducting P wave



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Anatomy
Subject
Cardiovascular System
System
Myocardial Infarction
Topic

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A 69-year-old man comes to the clinic due to a squeezing pressure in his midline chest and neck that occurs when walking. He has a history of hypertension and type 2 diabetes mellitus. The patient is an ex-smoker with a 20-pack-year history. Coronary angiography shows extensive atherosclerosis and near-total occlusion of the left anterior descending artery. Further testing shows normal resting left ventricular ejection fraction with no regional wall motion abnormalities. The absence of myocardial necrosis and scarring despite vessel occlusion in this patient can be best explained by which of the following features of the occluding plaque?

- A. Active peripheral inflammation
- B. Lipid-rich core
- C. Low calcium content
- D. Ostial location
- E. Slow growth rate
- F. Thin fibrous cap

Submit

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- A. Active peripheral inflammation (2%)
- B. Lipid-rich core (5%)
- ✗ C. Low calcium content (4%)
- D. Ostial location (6%)
- ✓ E. Slow growth rate (61%)
- F. Thin fibrous cap (18%)

Incorrect
Correct answer
E

61%
Answered correctly

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Time Spent

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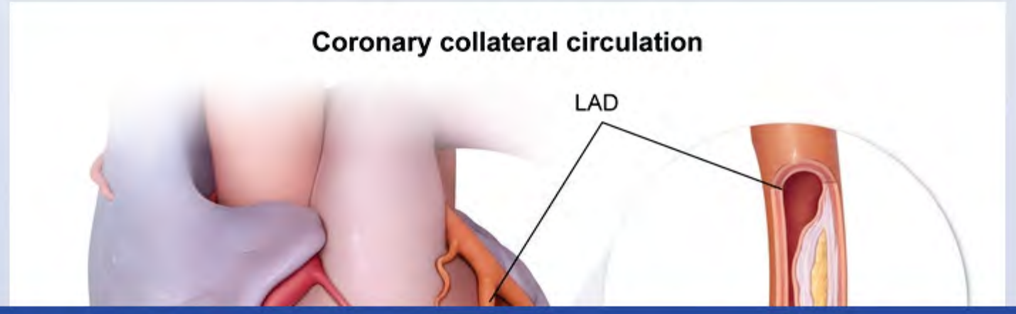
Explanation



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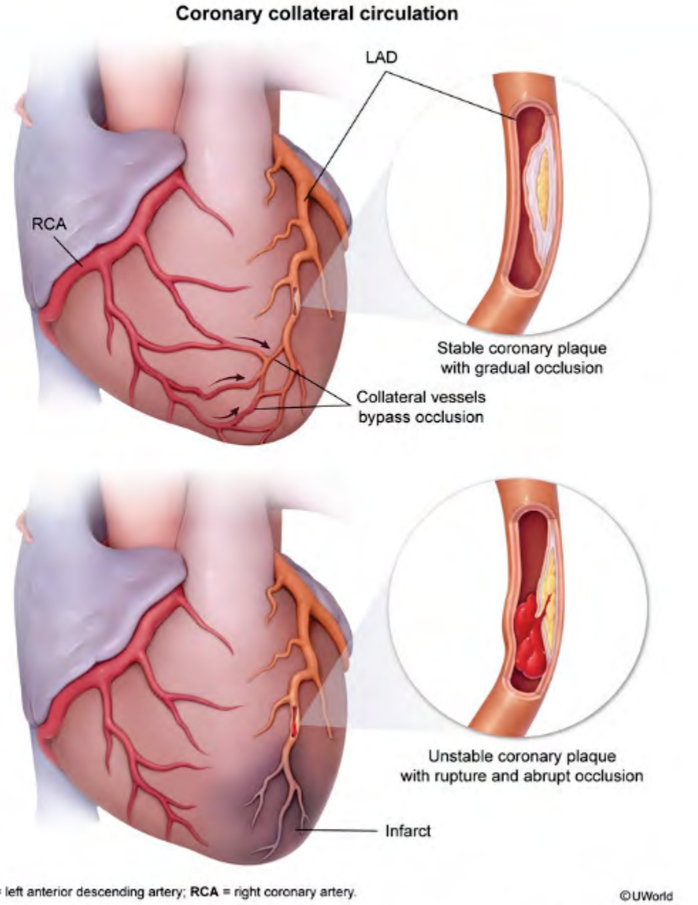

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LAD = left anterior descending artery; **RCA** = right coronary artery.

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In response to progressive reductions in blood flow caused by atherosclerotic coronary artery disease, **ischemic myocardium** may release growth factors (eg, vascular endothelial growth factor) to stimulate the formation and maturation of **collateral vessels**. In this patient, the **slow development** of a **stable atherosclerotic plaque** in the left anterior descending artery (LAD) likely allowed collateral vessels from the right coronary artery to supply the ischemic myocardium located distal to the LAD occlusion. Because there is low hydrostatic pressure distal to the LAD occlusion, a pressure gradient is created that facilitates blood flow through the small collateral vessels.

An unstable atherosclerotic plaque is less likely to allow the development of viable collateral vessels, as it is more likely to rupture before it becomes large enough to cause significant chronic ischemia; rupture leads to overlying thrombus and **abrupt arterial occlusion** with acute myocardial ischemia/**infarction** and cellular necrosis. The rapid development of ischemia allows insufficient time for collateral vessels to develop and mature.

(Choices A, B, and F) Atherosclerotic plaques with active inflammation (containing activated macrophages that secrete metalloproteinases and degrade collagen), high core concentration of lipids, and/or a thin fibrous cap are unstable and more likely to rupture. All of these properties are less likely in an atherosclerotic lesion that has grown to create near-total occlusion without rupturing.

(Choice C) A patient's total coronary artery calcium content correlates with the total atherosclerotic plaque burden, and calcium scoring by cardiac CT scan is sometimes used to estimate the severity of coronary artery disease. However, low calcium content (or immature calcification) within an individual plaque may decrease stability of that plaque and increase the likelihood of rupture.

(Choice D) Ostial atherosclerotic plaques (those located at a branch point of 2 arteries) are more likely to occlude blood flow to multiple areas of myocardium, thereby limiting the availability of nearby well-perfused arteries from which viable collateral vessels can develop.

Educational objective:
Gradually developing myocardial ischemia encourages the formation and maturation of collateral vessels and is most likely to occur in the setting of a slow-growing, stable atherosclerotic plaque. An unstable atherosclerotic plaque (eg, that with active inflammation, a lipid-rich core, and/or a thin fibrous cap) is more likely to rupture,

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Educational objective:
Gradually developing myocardial ischemia encourages the formation and maturation of collateral vessels and is most likely to occur in the setting of a slow-growing, stable atherosclerotic plaque. An unstable atherosclerotic plaque (eg, that with active inflammation, a lipid-rich core, and/or a thin fibrous cap) is more likely to rupture, resulting in the abrupt onset of ischemia/infarction that precludes the development of viable collateral vessels.

References

- Importance of angina for development of collateral circulation.

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A 52-year-old man with hypertension begins taking a new antihypertensive agent that is expected to enhance natriuresis while also causing a sustained decrease in serum angiotensin II concentration and aldosterone production. The effects of this medication are most similar to which of the following drug classes?

- A. Alpha-1 adrenergic blocker
- B. Angiotensin II receptor blocker
- C. Direct renin inhibitor
- D. Loop diuretic
- E. Mineralocorticoid receptor antagonist

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A 52-year-old man with hypertension begins taking a new antihypertensive agent that is expected to enhance natriuresis while also causing a sustained decrease in serum angiotensin II concentration and aldosterone production. The effects of this medication are most similar to which of the following drug classes?

- A. Alpha-1 adrenergic blocker (2%)
- ✗ B. Angiotensin II receptor blocker (23%)
- ✓ C. Direct renin inhibitor (58%)
- D. Loop diuretic (6%)
- E. Mineralocorticoid receptor antagonist (9%)

Incorrect
Correct answer
C

58%
Answered correctly

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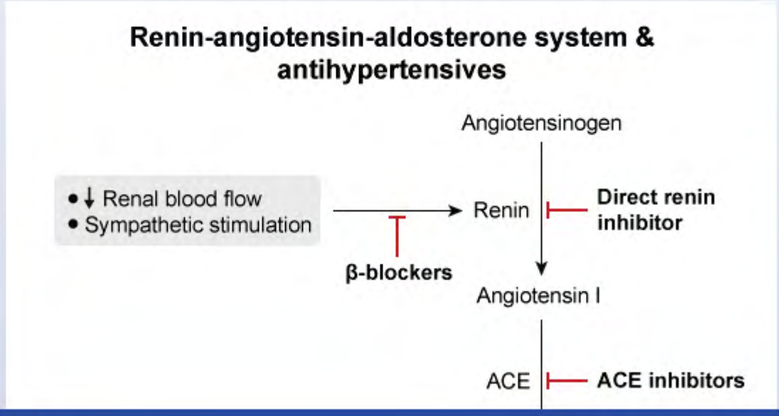
Explanation



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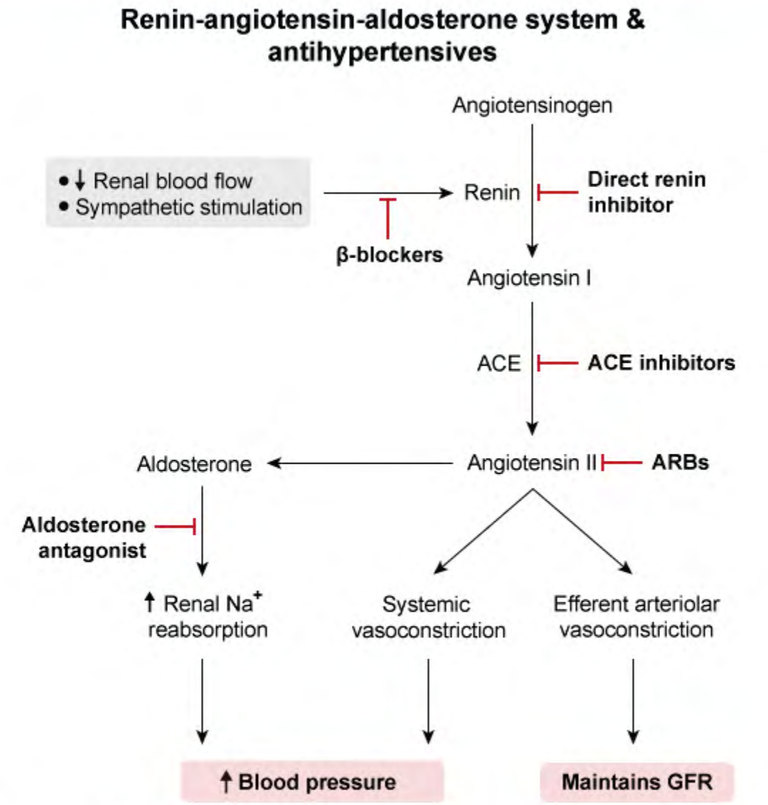

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Renin-angiotensin-aldosterone system &

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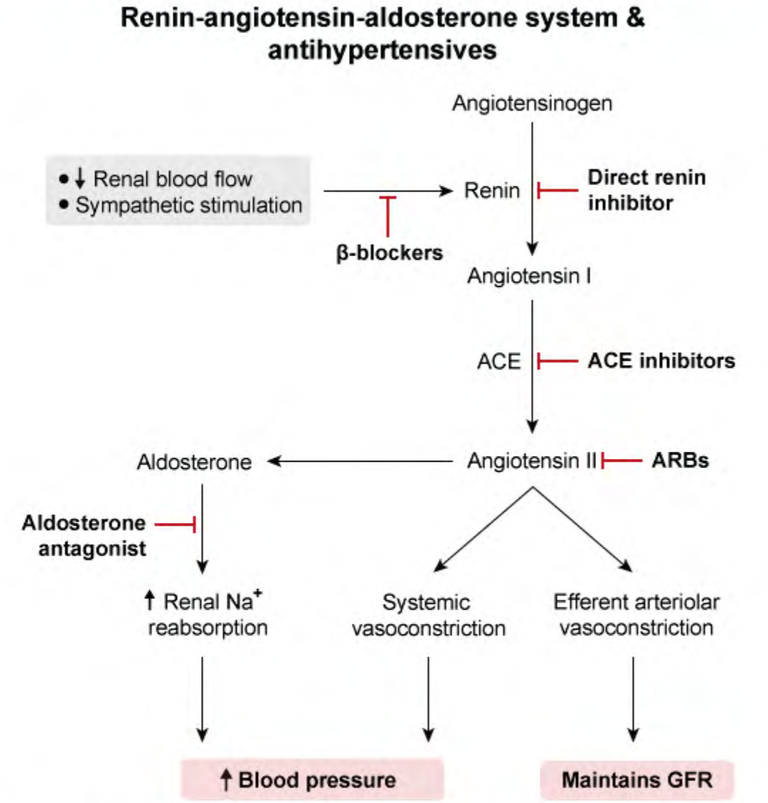

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GFR = glomerular filtration rate.
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Renin-angiotensin-aldosterone system (RAAS) overactivity can have a significant role in the development of **hypertension**. Renin is produced in the juxtaglomerular cells of the kidney in response to hypoperfusion. Renin cleaves angiotensinogen into angiotensin I, which is converted to angiotensin II by angiotensin-converting enzyme (ACE) in the lungs. Angiotensin II is a potent vasoconstrictor that also promotes aldosterone production in the adrenal cortex. Aldosterone acts on the collecting ducts to increase renal sodium and water reabsorption, which increases intravascular blood volume and blood pressure.

Any medication that **inhibits RAAS** activity will **enhance natriuresis** via a reduction in aldosterone activity. The only listed medication that enhances natriuresis while also decreasing angiotensin II level and aldosterone production is a **direct renin inhibitor** (eg, aliskiren). ACE inhibitors (eg, lisinopril) prevent conversion of angiotensin I to angiotensin II and also enhance natriuresis while **decreasing angiotensin II** levels and aldosterone production.

(Choice A) Alpha-1 adrenergic blockers (eg, terazosin, doxazosin) are vasodilators that are occasionally used to treat hypertension. These drugs decrease blood pressure via direct vasodilation and do not decrease angiotensin II or aldosterone concentrations or induce natriuresis.

(Choice B) Angiotensin II receptor blockers (eg, losartan) prevent angiotensin II from acting on angiotensin receptors. These drugs reduce aldosterone production and induce natriuresis, but they increase angiotensin II levels (the RAAS is blocked after angiotensin I is converted to angiotensin II).

(Choice D) Loop diuretics (eg, furosemide) induce natriuresis but do not inhibit RAAS activity. The decreased blood volume caused by loop diuretics actually stimulates renin release, which then increases angiotensin II and aldosterone concentrations.

(Choice E) Mineralocorticoid receptor antagonists (eg, spironolactone) block aldosterone receptors to induce natriuresis. However, angiotensin II and aldosterone levels are increased.

Educational objective:

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increases intravascular blood volume and blood pressure.

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(Choice E) Mineralocorticoid receptor antagonists (eg, spironolactone) block aldosterone receptors to induce natriuresis. However, angiotensin II and aldosterone levels are increased.

Educational objective:
The renin-angiotensin-aldosterone system (RAAS) is integrally involved in the pathophysiology and treatment of hypertension. RAAS activity can be reduced by several classes of antihypertensives, all of which will induce natriuresis by decreasing the effects of aldosterone. Direct renin inhibitors and ACE inhibitors accomplish this by lowering circulating levels of angiotensin II.

Pharmacology	Cardiovascular System	Primary hypertension
Subject	System	Topic

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A 36-year-old man comes to the emergency department after briefly losing consciousness while watching television half an hour ago. The patient had no preceding chest pain or shortness of breath, but he has been having recurrent palpitations over the past several days. One month ago, he noticed a non-itchy rash on his thigh that he treated with an over-the-counter steroid cream. Medical history is otherwise unremarkable. There is no family history of heart disease or sudden cardiac death. The patient recently began working as a forest ranger in Pennsylvania. He does not use tobacco, alcohol, or illicit drugs. Blood pressure is 130/80 mm Hg and pulse is 46/min. The lungs are clear on auscultation and heart sounds are normal. The remainder of the examination shows no abnormalities. ECG reveals complete atrioventricular block. Echocardiography shows normal ventricular function with no major valvular disease. Which of the following is the most likely underlying cause of this patient's current condition?

- A. Disseminated spirochetal infection
- B. Granulomatous myocardial inflammation
- C. Missense mutation of a sarcomere protein
- D. Myocyte potassium channel mutation
- E. Viral infection–induced cardiac injury

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- ✓ A. Disseminated spirochetal infection (80%)
- ✗ B. Granulomatous myocardial inflammation (5%)
- C. Missense mutation of a sarcomere protein (1%)
- D. Myocyte potassium channel mutation (3%)
- E. Viral infection–induced cardiac injury (10%)

Incorrect
Correct answer
A

80%
Answered correctly

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Time Spent

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Explanation

This patient most likely has **early disseminated Lyme disease**. Lyme disease is caused by the bacterial spirochete *Borrelia burgdorferi*, which is transmitted by the *Ixodes* tick that is endemic to the northeastern and parts of the midwestern United States. Most cases initially present with a non-itchy, bull's-eye–shaped rash, known

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This patient most likely has **early disseminated Lyme disease**. Lyme disease is caused by the bacterial spirochete *Borrelia burgdorferi*, which is transmitted by the *Ixodes* tick that is endemic to the northeastern and parts of the midwestern United States. Most cases initially present with a non-itchy, bull's-eye-shaped rash, known as erythema migrans, at the site of infection. Left untreated for several weeks or more, a small percentage of those infected will develop cardiac involvement **(Lyme carditis)**.

Lyme carditis most commonly manifests with varying degrees of **atrioventricular (AV) conduction block**. Many patients remain asymptomatic; however, patients who develop complete AV block are likely to have symptoms of dyspnea, **lightheadedness**, or **syncope**. Treatment for symptomatic AV block due to Lyme carditis typically requires hospitalization with temporary pacing and the administration of intravenous antibiotics (ceftriaxone is the drug of choice).

(Choice B) Granulomatous myocardial inflammation occurs in cardiac sarcoidosis, and AV conduction block is the most common disease manifestation. However, this patient's recent rash and residence in an endemic area make Lyme carditis more likely.

(Choice C) A missense mutation affecting the cardiac sarcomere protein beta-myosin heavy chain is a common cause of hypertrophic cardiomyopathy. Myocardial disarray creates a propensity for ventricular arrhythmias, but AV conduction block is not typical.

(Choice D) Congenital long QT syndrome results from a mutation affecting cardiomyocyte potassium channels. It can cause syncope or sudden cardiac death, typically via the triggering of torsades de pointes (polymorphic ventricular tachycardia). AV conduction block would be an unusual manifestation.

(Choice E) Viral infection of the heart typically manifests as myocarditis. The most common symptomatic presentation is decompensated heart failure due to dilated cardiomyopathy. AV conduction block is not typical.

Educational objective:
Early disseminated Lyme disease can have cardiac involvement (Lyme carditis) that most commonly manifests with varying degrees of atrioventricular (AV) conduction block. Patients may be asymptomatic, but those with

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Educational objective:
Early disseminated Lyme disease can have cardiac involvement (Lyme carditis) that most commonly manifests with varying degrees of atrioventricular (AV) conduction block. Patients may be asymptomatic, but those with complete AV conduction block are likely to have dyspnea, lightheadedness, or syncope.

Pathology	Cardiovascular System	Lyme disease
Subject	System	Topic

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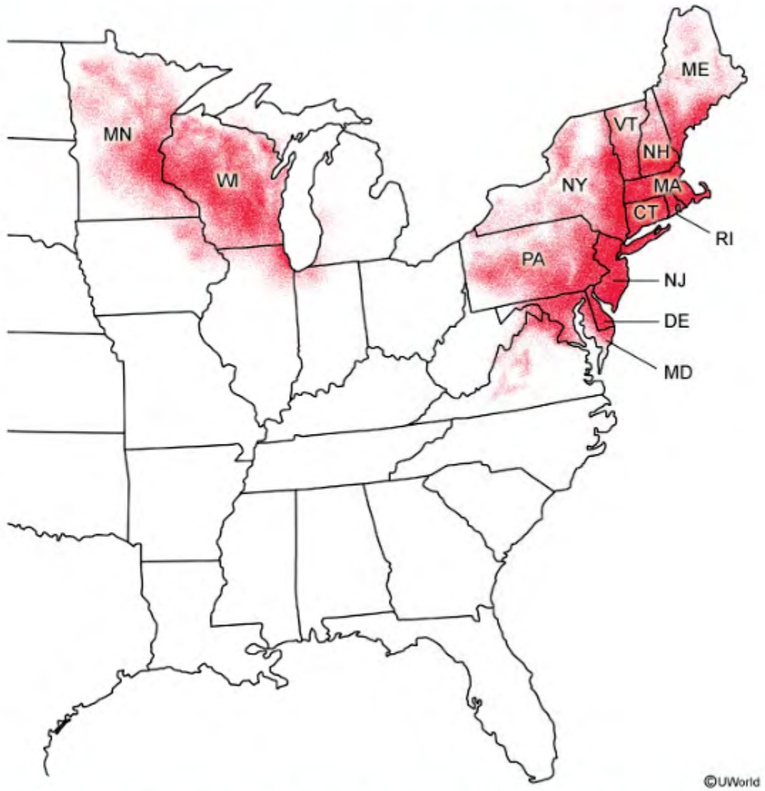
Lyme disease–endemic areas in the United States



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Erythema migrans

*Expands from central tick bite (due to Lyme disease).

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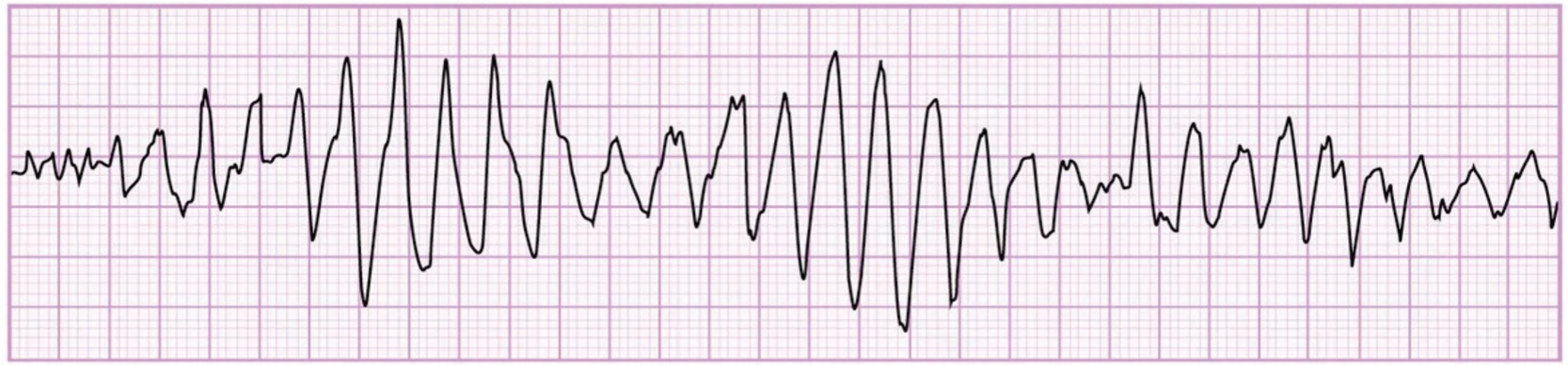
Torsade de pointes



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Pathology (Subject) | Cardiovascular System (System) | Lyme disease (Topic)

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A 32-year-old woman is recruited for a research study. She is evaluated in the hospital as part of the study protocol. Her medical history includes an uncomplicated appendectomy, and medications include oral contraceptive pills and a multivitamin. During the evaluation, increases in heart rate and cardiac output are observed, and an arterial blood gas study taken at the same time shows normal partial pressures of arterial oxygen (PaO_2) and carbon dioxide ($PaCO_2$). These changes are most likely part of an integrated response to which of the following?

- A. Carotid sinus massage
- B. Exercise
- C. High altitude exposure
- D. Panic attack
- E. Pulmonary artery occlusion

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- A. Carotid sinus massage (9%)
- ✓ B. Exercise (76%)
- C. High altitude exposure (2%)
- D. Panic attack (9%)
- E. Pulmonary artery occlusion (1%)

Correct | 76% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

This patient with increased heart rate, cardiac output, and unchanged partial pressures of arterial oxygen (PaO_2) and carbon dioxide ($PaCO_2$) is likely undergoing an **exercise** test. **Heart rate** and **cardiac output increase** to meet increased tissue oxygen demands, and the respiratory rate increases to eliminate the additional carbon dioxide produced. Changes in the partial pressures of oxygen and carbon dioxide during exercise occur predominantly in the venous blood as muscle tissue extracts a higher portion of oxygen and produces greater amounts of carbon dioxide. The **PaO_2** and **$PaCO_2$** remain relatively close to **resting values** at most levels of

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This patient with increased heart rate, cardiac output, and unchanged partial pressures of arterial oxygen (PaO_2) and carbon dioxide ($PaCO_2$) is likely undergoing an **exercise** test. **Heart rate** and **cardiac output increase** to meet increased tissue oxygen demands, and the respiratory rate increases to eliminate the additional carbon dioxide produced. Changes in the partial pressures of oxygen and carbon dioxide during exercise occur predominantly in the venous blood as muscle tissue extracts a higher portion of oxygen and produces greater amounts of carbon dioxide. The **PaO_2** and **$PaCO_2$** remain relatively close to **resting values** at most levels of exercise due to a number of adaptations in the lungs and pulmonary circulation, such as hyperventilation and improved ventilation-perfusion (V/Q) matching.

(Choice A) Carotid sinus massage causes reflex parasympathetic stimulation of the sinoatrial node, atrial myocytes, and atrioventricular node, resulting in a transient decrease in heart rate and cardiac output.

(Choice C) High altitude simulation is associated with lower ambient oxygen pressures; consequently, heart rate and cardiac output increase in order to improve oxygen delivery to tissues. The lower oxygen pressures cause hypoxemia and result in hypoxic stimulation of respiration, causing hyperventilation and decreased $PaCO_2$.

(Choice D) A panic attack can increase heart rate and cardiac output via sympathetic stimulation of the heart; however, there is usually associated hyperventilation without a significant increase in muscle activity and therefore a fall in the $PaCO_2$.

(Choice E) Pulmonary artery occlusion as seen in pulmonary embolism (PE) results in increased pulmonary vascular resistance, right ventricular strain, and frequently decreased left ventricular stroke volume. Reflex tachycardia can result to maintain (not increase) cardiac output. Additionally, PE often causes hypoxemia (low PaO_2) due to acute V/Q mismatching in the affected lung.

Educational objective:
The cardiorespiratory response to exercise includes increased heart rate, cardiac output, and respiratory rate in order to balance the increased total tissue oxygen consumption and carbon dioxide production. These coordinated adaptations result in relatively constant arterial blood gas values whereas venous oxygen is decreased and venous carbon dioxide is increased.

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References

- Integrative biology of exercise.
- Organ-specific physiological responses to acute physical exercise and long-term training in humans

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A 42-year-old man comes to the emergency department due to acute-onset shortness of breath. He has no medical conditions and does not use alcohol, tobacco, or illicit drugs. The patient works as a box handler at a warehouse and has excellent exercise tolerance. Family history is insignificant. Blood pressure is 98/60 mm Hg and pulse is 112/min and regular. A short systolic murmur is heard at the apex. Bilateral crackles are present on lung auscultation. Bedside echocardiography reveals a flail posterior mitral leaflet due to chordal rupture and severe mitral regurgitation. Which of the following best reflects this patient's left ventricular hemodynamics compared to the normal heart?

- A. Afterload decrease, preload decrease, ejection fraction decrease
- B. Afterload decrease, preload increase, ejection fraction increase
- C. Afterload increase, preload decrease, ejection fraction decrease
- D. Afterload increase, preload decrease, ejection fraction increase
- E. Afterload increase, preload increase, ejection fraction increase

Submit

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A 42-year-old man comes to the emergency department due to acute-onset shortness of breath. He has no medical conditions and does not use alcohol, tobacco, or illicit drugs. The patient works as a box handler at a warehouse and has excellent exercise tolerance. Family history is insignificant. Blood pressure is 98/60 mm Hg and pulse is 112/min and regular. A short systolic murmur is heard at the apex. Bilateral crackles are present on lung auscultation. Bedside echocardiography reveals a flail posterior mitral leaflet due to chordal rupture and severe mitral regurgitation. Which of the following best reflects this patient's left ventricular hemodynamics compared to the normal heart?

- A. Afterload decrease, preload decrease, ejection fraction decrease (22%)
- ✓ B. Afterload decrease, preload increase, ejection fraction increase (44%)
- C. Afterload increase, preload decrease, ejection fraction decrease (19%)
- ✗ D. Afterload increase, preload decrease, ejection fraction increase (2%)
- E. Afterload increase, preload increase, ejection fraction increase (11%)

Incorrect
Correct answer
B

44%
Answered correctly

02 mins, 52 secs
Time Spent

2023
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Explanation

Hemodynamic changes in mitral regurgitation

	Acute MR	Compensated chronic MR	Decompensated chronic MR
Preload	↑↑	↑	↑
Afterload	↓	No change	↑
Contractile function	No change	No change	↓

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Hemodynamic changes in mitral regurgitation			
	Acute MR	**Compensated chronic MR**	**Decompensated chronic MR**
Preload	↑↑	↑	↑
Afterload	↓	No change	↑
Contractile function	No change	No change	↓
Ejection fraction	↑↑	↑	↓
Forward stroke volume	↓	No change	↓
MR = mitral regurgitation.			

This patient has **acute mitral regurgitation** (MR) due to a spontaneous chordae tendineae rupture. In acute MR, the left atrium (LA) is of normal size and compliance and is suddenly exposed to a large volume of regurgitant blood from the left ventricle (LV). This leads to very high LA pressures and subsequent **pulmonary edema**.

In addition, because the LV must accommodate the regurgitant blood volume along with normal pulmonary venous return, there is **increased LV end-diastolic volume** (preload). Meanwhile, the incompetent mitral valve forms a low-resistance regurgitant pathway for blood to flow into the LA during ventricular systole, **decreasing LV afterload**. The decreased afterload leads to **increased LV ejection fraction** (via increased stroke volume); however, much of the stroke volume is lost to regurgitation into the LA, resulting in overall decreased forward stroke volume and reduced cardiac output (manifesting as **hypotension** and cardiogenic shock).

In chronic MR, compensatory LA enlargement allows the LA to receive the regurgitant volume at lower filling pressures, preventing pulmonary edema from developing. Chronic volume overload also causes the LV to undergo substantial enlargement due to eccentric hypertrophy. Early on, the larger ventricular volume helps maintain forward stroke volume even in the setting of substantial regurgitant flow. However, with prolonged hemodynamic overload, progressive eccentric remodeling of the LV becomes maladaptive, resulting in an overwhelming increase

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In chronic MR, compensatory LA enlargement allows the LA to receive the regurgitant volume at lower filling pressures, preventing pulmonary edema from developing. Chronic volume overload also causes the LV to undergo substantial enlargement due to eccentric hypertrophy. Early on, the larger ventricular volume helps maintain forward stroke volume even in the setting of substantial regurgitant flow. However, with prolonged hemodynamic overload, progressive eccentric remodeling of the LV becomes maladaptive, resulting in an overwhelming increase in wall stress with eventual contractile dysfunction. The impaired contractility culminates in decompensation of chronic MR with reduced forward stroke volume (decreased cardiac output) and increased left-sided filling pressures (pulmonary edema).

Educational objective:
Regurgitant flow into the left atrium in acute mitral regurgitation leads to increased left atrial pressure and increased left ventricular end-diastolic volume (preload). The low-resistance regurgitant pathway also decreases left ventricular afterload with a resulting increase in ejection fraction but overall decrease in forward stroke volume. Increased left atrial pressure and decreased cardiac output result in pulmonary edema and severe hypotension, respectively.

References

- Left ventricular response to mitral regurgitation: implications for management.

Pathophysiology Cardiovascular System Mitral regurgitation

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Ejection fraction	↓	↓	↓
Forward stroke volume	↓	No change	↓

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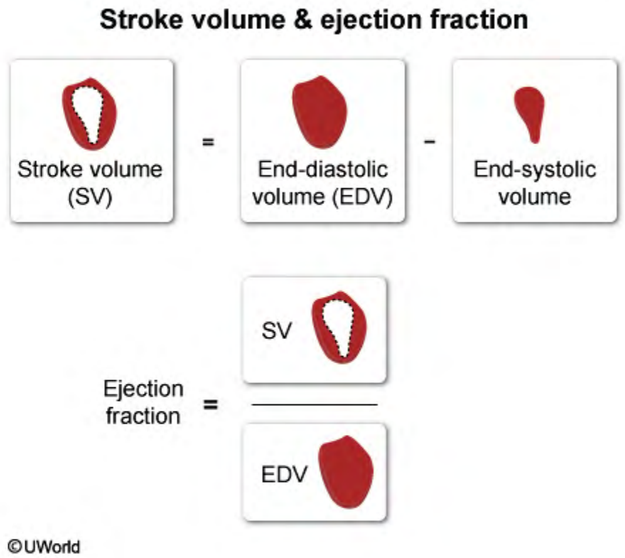

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Ejection fraction	↓		↓
Forward stroke volume	↓	No change	↓

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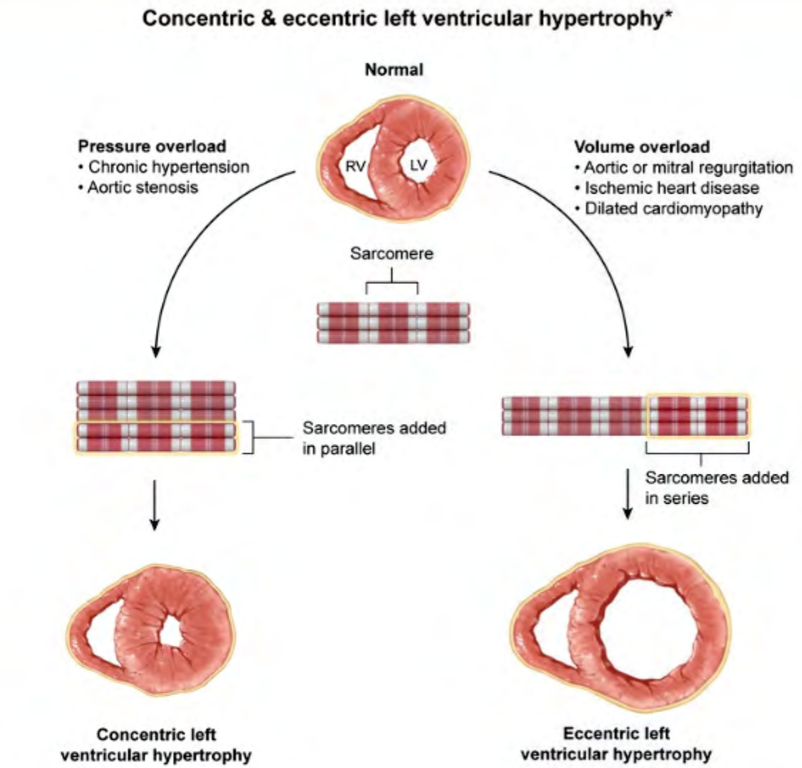

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Increased left atrial pressure and decreased cardiac output result in pulmonary edema and severe hypotension, respectively.

References

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A 79-year-old man is brought to the hospital due to confusion for the past 2 days. The patient has had nausea, decreased appetite, and poor oral intake for several weeks. He has also mentioned that everything around him has "a yellow tint." He has no abdominal pain. Medical history includes long-standing heart failure with reduced ejection fraction and atrial fibrillation. Medications include metoprolol, digoxin, lisinopril, and apixaban. The patient is afebrile. Blood pressure is 130/80 mm Hg and pulse is 58/min. The abdomen is soft and nontender; there is no rigidity or rebound and no masses are present. Age-related changes in which of the following factors most likely accounts for this patient's symptoms?

- A. Intestinal absorption
- B. Liver enzyme activity
- C. Muscle mass
- D. Plasma protein binding
- E. Renal clearance

Submit

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- ✗ A. Intestinal absorption (2%)
- B. Liver enzyme activity (24%)
- C. Muscle mass (3%)
- D. Plasma protein binding (2%)
- ✓ E. Renal clearance (66%)

Incorrect
Correct answer
E

66%
Answered correctly

04 secs
Time Spent

2023
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Explanation

Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
	• Fatigue

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Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
Neurologic	• Fatigue • Confusion • Weakness • **Color vision alterations**

Digoxin is a cardiac glycoside that can be used to treat atrial fibrillation and systolic heart failure. This patient is experiencing symptoms classically associated with **digoxin toxicity**, which include visual changes (particularly **yellow tinting** of objects) and gastrointestinal disturbances. Digoxin is **renally cleared** and has a narrow therapeutic window, so toxicity frequently results from alterations in kidney function. As patients age, renal function decreases progressively. This decrease in renal function is often not accompanied by a concomitant rise in serum creatinine, as creatinine is derived from muscle tissue and lean body mass also decreases with age. Therefore, elderly patients (even those with a normal serum creatinine) should receive reduced doses of renally cleared medications to prevent toxicity.

(Choice A) Intestinal absorption affects the dose of digoxin that reaches the bloodstream. Some preparations of digoxin may have a higher bioavailability than others, and some patients may carry gut bacteria that degrade digoxin before it has the ability to cross into the bloodstream.

(Choice B) Liver enzyme activity plays a small role in the elimination of digoxin, as this agent is primarily excreted unchanged by the kidney.

(Choice C) This patient's lean muscle mass is important to consider when dosing digoxin based on weight, as this serves as the primary site where digoxin is stored in the body. Lean body mass does tend to decrease in an age-related manner, but age-related renal insufficiency is more directly related to toxicity of digoxin in the elderly.

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- Weakness
- **Color vision alterations**

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(Choice D) Plasma protein binding of digoxin is not as significant as renal clearance when considering toxicity. Typically less than one-third of the digoxin that gains access to the bloodstream will be protein-bound; the remainder of the dose circulates unbound.

Educational objective:
Digoxin is a cardiac glycoside that is predominantly cleared by the kidneys. Elderly patients typically exhibit age-related renal insufficiency, even in the presence of normal creatinine levels. The dose of digoxin must be reduced in these patients to prevent toxicity.

References

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A 34-year-old man comes to the emergency department with fatigue and lightheadedness. The patient had an upper respiratory infection last week, and since then his energy level has been low with shortness of breath on mild exertion. Otherwise, his medical history is insignificant. He is a lifetime nonsmoker. The patient's temperature is 37 C (98.6 F), blood pressure is 80/60 mm Hg, and pulse is 120/min and regular. His pulse becomes undetectable to palpation during inspiration. The lungs are clear to auscultation, but the jugular veins are distended. Which of the following is the most likely diagnosis?

- A. Acute myocardial infarction
- B. Acute viral myocarditis
- C. Cardiac tamponade
- D. Constrictive pericarditis
- E. Septic shock
- F. Tension pneumothorax

Submit

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A 34-year-old man comes to the emergency department with fatigue and lightheadedness. The patient had an upper respiratory infection last week, and since then his energy level has been low with shortness of breath on mild exertion. Otherwise, his medical history is insignificant. He is a lifetime nonsmoker. The patient's temperature is 37 C (98.6 F), blood pressure is 80/60 mm Hg, and pulse is 120/min and regular. His pulse becomes undetectable to palpation during inspiration. The lungs are clear to auscultation, but the jugular veins are distended. Which of the following is the most likely diagnosis?

- ✗ A. Acute myocardial infarction (0%)
- B. Acute viral myocarditis (13%)
- ✓ C. Cardiac tamponade (48%)
- D. Constrictive pericarditis (33%)
- E. Septic shock (3%)
- F. Tension pneumothorax (1%)

Incorrect
Correct answer
C

48%
Answered correctly

03 secs
Time Spent

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Version

Explanation

Clinical features of cardiac tamponade	
Etiology	• Malignancy/radiation therapy • Infection (eg, viral, tuberculosis, HIV) • Drugs (eg, hydralazine, isoniazid) • Connective tissue diseases (eg, systemic lupus erythematous, rheumatoid arthritis)
	• Beck triad: jugular venous distension, hypotension, diminished heart sounds

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Clinical features of cardiac tamponade	
Etiology	• Malignancy/radiation therapy • Infection (eg, viral, tuberculosis, HIV) • Drugs (eg, hydralazine, isoniazid) • Connective tissue diseases (eg, systemic lupus erythematous, rheumatoid arthritis)
Clinical signs	• Beck triad: jugular venous distension, hypotension, diminished heart sounds • Pulsus paradoxus (abnormally large inspiratory decrease in systolic blood pressure >10 mm Hg)
Diagnosis	• ECG: low-voltage QRS complexes, electrical alternans • Chest x-ray: enlarged water bottle–shaped heart, clear lungs

This presentation of **hypotension**, tachycardia, **jugular venous distension** with clear lungs, and **pulsus paradoxus** (manifesting as loss of palpable pulse during inspiration) is consistent with **cardiac tamponade**. This is most likely due to the patient's recent viral illness causing viral pericarditis with significant pericardial fluid accumulation. Normally there is a <10 mm Hg decrease in systolic blood pressure during inspiration. Pulsus paradoxus refers to an abnormal exaggerated decrease in systolic blood pressure >10 mm Hg on inspiration, a common finding in patients with large pericardial effusions causing tamponade.

Inspiration causes an increase in systemic venous return, resulting in increased right heart volumes. Under normal conditions, this results in expansion of the right ventricle into the pericardial space with little impact on the left side of the heart. However, in conditions that impair expansion into the pericardial space (eg, cardiac tamponade), the increased right ventricular volume that occurs with inspiration leads to bowing of the interventricular septum toward the left ventricle. This leads to a decrease in left ventricular (LV) end-diastolic volume and forward stroke volume, with a resultant decrease in systolic pressure during inspiration.

(Choice A) Acute myocardial infarction typically presents with sudden onset of substernal discomfort, diaphoresis, and/or dyspnea, and can progress to cardiogenic shock (hypotension, tachycardia, jugular venous distension, and pulmonary edema). Patients can have weak or undetectable peripheral pulses; however, pulsus paradoxus is

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Increased right ventricular volume that occurs with inspiration leads to bowing of the interventricular septum toward the left ventricle. This leads to a decrease in left ventricular (LV) end-diastolic volume and forward stroke volume, with a resultant decrease in systolic pressure during inspiration.

(Choice A) Acute myocardial infarction typically presents with sudden onset of substernal discomfort, diaphoresis, and/or dyspnea, and can progress to cardiogenic shock (hypotension, tachycardia, jugular venous distension, and pulmonary edema). Patients can have weak or undetectable peripheral pulses; however, pulsus paradoxus is typically not seen.

(Choice B) Acute viral myocarditis can lead to severe LV systolic dysfunction and present with acute congestive heart failure and cardiogenic shock. Patients with severe LV failure can have pulsus alternans - a variation in pulse amplitude with alternate beats. Pulsus paradoxus is typically not seen in such patients.

(Choice D) Pulsus paradoxus can also occur in patients with asthma, chronic obstructive pulmonary disease, and (less frequently) constrictive pericarditis. Constrictive pericarditis is caused by scarring and loss of elasticity of the normal pericardial sac; it takes several weeks to months to develop and only rarely occurs after recurrent episodes of acute pericarditis.

(Choice E) The initial stage of septic shock is a hyperdynamic circulatory state with lowered systemic vascular resistance and increased cardiac output (warm shock). Weak pulses and pulsus paradoxus would not be expected in such patients.

(Choice F) In tension pneumothorax, breath sounds are typically absent on the affected side with hyperresonance to percussion. Other features of tension pneumothorax include tachypnea, tachycardia, hypotension, distended neck veins, and tracheal deviation to the contralateral side.

Educational objective:
Cardiac tamponade typically presents with hypotension with pulsus paradoxus, elevated jugular venous pressure, and muffled heart sounds (Beck's triad). Pulsus paradoxus refers to an abnormal exaggerated decrease in systolic blood pressure >10 mm Hg on inspiration, and is a common finding in patients with pericardial effusion with cardiac tamponade.

Pathophysiology	Cardiovascular System	Cardiac tamponade
Subject	System	Topic

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signs	mm Hg)
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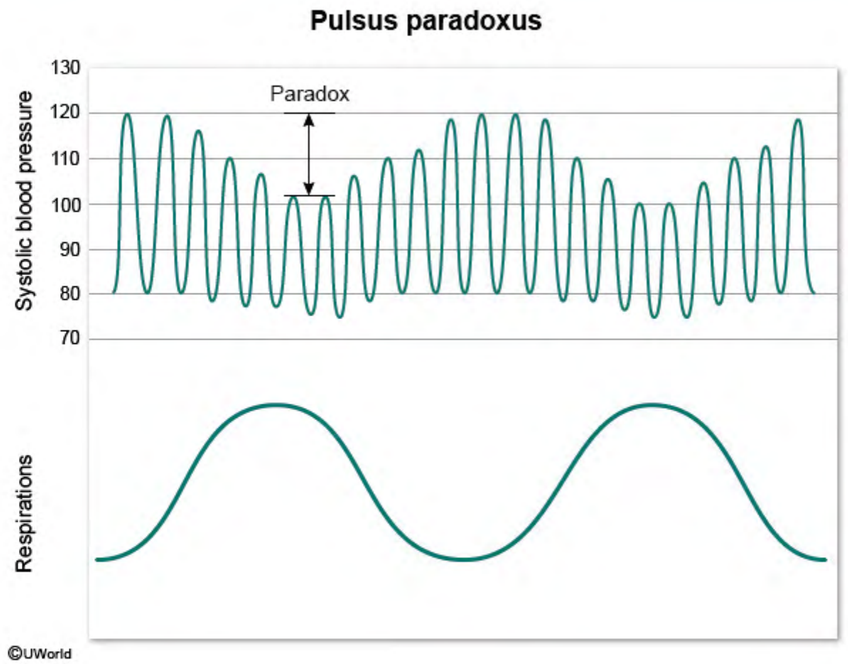

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(Choice E) The initial stage of septic shock is a hyperdynamic circulatory state with lowered systemic vascular

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A 62-year-old man comes to the office due to chest pain over the last 6 months. He describes pressure-like pain in the substernal area when walking fast or climbing stairs that gradually subsides when he stops. The patient does not smoke cigarettes or use illicit drugs. He has no family history of heart disease or sudden death. Physical examination shows a mid-systolic murmur at the upper sternum and slow-rising carotid pulses. After initial evaluation, cardiac catheterization is performed and reveals 30% stenosis of the mid-left anterior descending artery. A 50 mm Hg pressure gradient is seen when a catheter is passed across the aortic valve. Which of the following is the most important contributor to this patient's presenting symptoms?

- A. Decrease in coronary blood supply due to lipid-rich plaque
- B. Decrease in coronary blood supply due to vasospasm
- C. Decrease in left ventricular compliance
- D. Increase in left ventricular stroke volume
- E. Increase in left ventricular wall stress

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- ✗ A. Decrease in coronary blood supply due to lipid-rich plaque (23%)
- B. Decrease in coronary blood supply due to vasospasm (6%)
- C. Decrease in left ventricular compliance (17%)
- D. Increase in left ventricular stroke volume (5%)
- ✓ E. Increase in left ventricular wall stress (47%)

Incorrect
Correct answer
E

47%
Answered correctly

03 secs
Time Spent

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Version

Explanation

This patient's slow-rising carotid pulses, mid-systolic murmur, and large pressure gradient across the aortic valve are consistent with **severe aortic stenosis** (AS). Patients can remain asymptomatic for long periods but eventually develop classic symptoms of severe AS; dyspnea is typically the first manifestation, followed by **angina**, presyncope or **syncope**, and overt **heart failure**.

The symptoms of severe AS usually occur with exertion, as the capacity to increase stroke volume and cardiac output is limited due to the fixed left ventricular outflow obstruction **(Choice D)**. This outflow obstruction also leads to higher chamber pressures and **increased wall stress** during systole and diastole. The rise in systolic wall

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This patient's slow-rising carotid pulses, mid-systolic murmur, and large pressure gradient across the aortic valve are consistent with **severe aortic stenosis** (AS). Patients can remain asymptomatic for long periods but eventually develop classic symptoms of severe AS; dyspnea is typically the first manifestation, followed by **angina**, presyncope or **syncope**, and overt **heart failure**.

The symptoms of severe AS usually occur with exertion, as the capacity to increase stroke volume and cardiac output is limited due to the fixed left ventricular outflow obstruction **(Choice D)**. This outflow obstruction also leads to higher chamber pressures and **increased wall stress** during systole and diastole. The rise in systolic wall stress **increases myocardial oxygen demand** while the increased diastolic wall stress reduces the coronary perfusion pressure gradient, leading to decreased myocardial perfusion. Increased left ventricular mass due to concentric hypertrophy further increases myocardial oxygen demand and contributes to anginal symptoms.

(Choice A) Reduced coronary blood supply due to flow-limiting atherosclerotic plaque can cause angina in patients with obstructive coronary artery disease. Stenosis >70% is typically necessary for anginal symptoms to develop; this patient's 30% stenosis is not clinically significant. Angina often occurs in severe AS in the absence of coronary artery disease.

(Choice B) Decreased coronary blood supply due to vasospasm is seen in patients with vasospastic (Prinzmetal) angina. Coronary vasospasm typically affects relatively young patients (age <50) and is not related to the angina that occurs with severe AS.

(Choice C) Decreased left ventricular compliance occurs over time in AS due to the prolonged high-pressure load created by the stenotic valve. This can result in heart failure (eg, fatigue, dyspnea) due to diastolic dysfunction, but would be less likely to induce angina symptoms.

Educational objective:
Angina often occurs in aortic stenosis even in the absence of obstructive coronary artery disease. It results from increased myocardial oxygen demand due to an increase in left ventricular mass (ie, concentric hypertrophy) and ventricular wall stress.

References

- Angina pectoris in patients with aortic stenosis and normal coronary arteries. Mechanisms and

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- Angina pectoris in patients with aortic stenosis and normal coronary arteries. Mechanisms and

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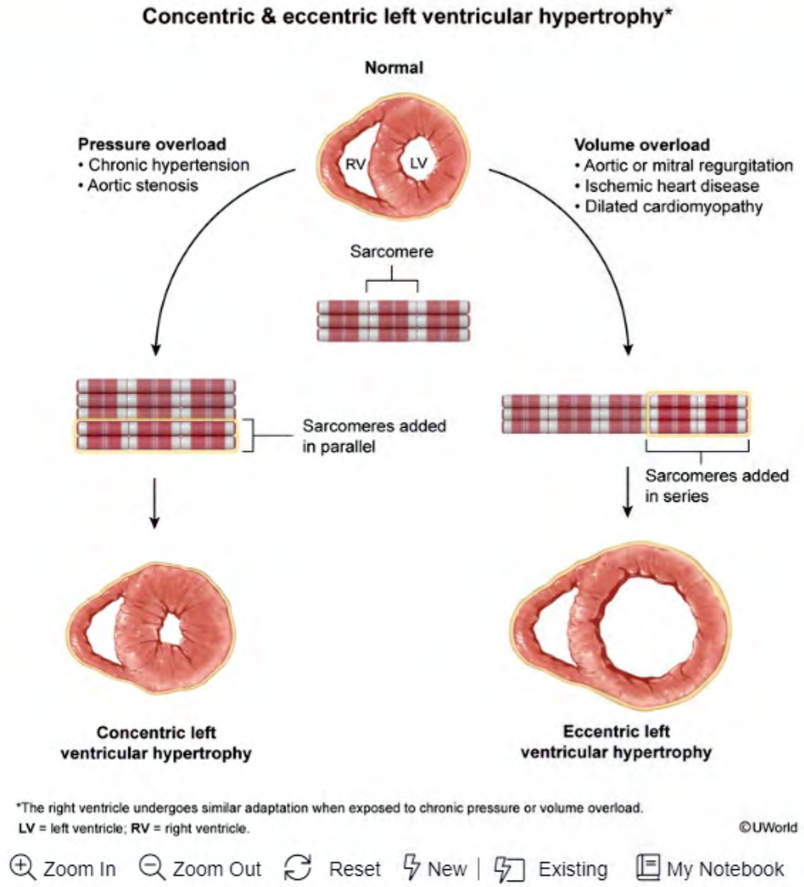

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A 64-year-old man becomes unconscious after experiencing severe retrosternal chest pain. He has a history of hypertension and dyslipidemia but is nonadherent with his prescribed medication regimen. He also has a long history of heavy smoking. Paramedics are called, and the man is pronounced dead on arrival to the emergency department. An autopsy reveals diffuse atherosclerotic vascular disease involving the coronary arteries. One of the atheromatous plaques in the left main artery has a ruptured fibrous cap with an overlying occluding thrombus. Which of the following cellular components is most directly involved in formation of the fibrous portion of this patient's plaque?

- A. Activated platelets
- B. Endothelial cells
- C. Foamy macrophages
- D. Infiltrating T lymphocytes
- E. Smooth muscle cells

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- ✗ A. Activated platelets (18%)
- B. Endothelial cells (14%)
- C. Foamy macrophages (31%)
- D. Infiltrating T lymphocytes (2%)
- ✓ E. Smooth muscle cells (32%)

Incorrect
Correct answer
E

32%
Answered correctly

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Time Spent

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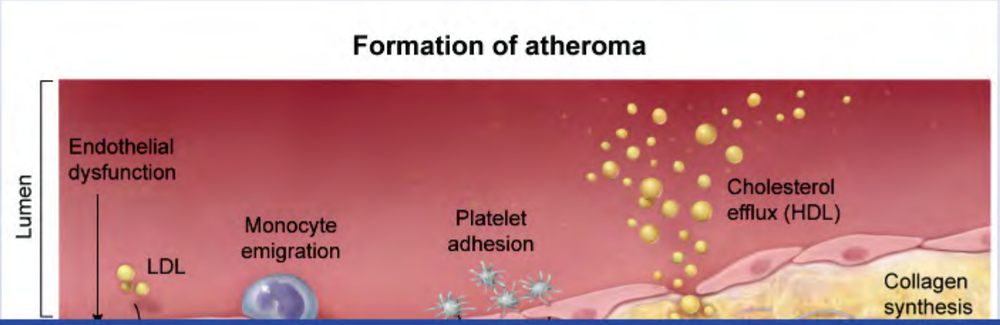
Explanation



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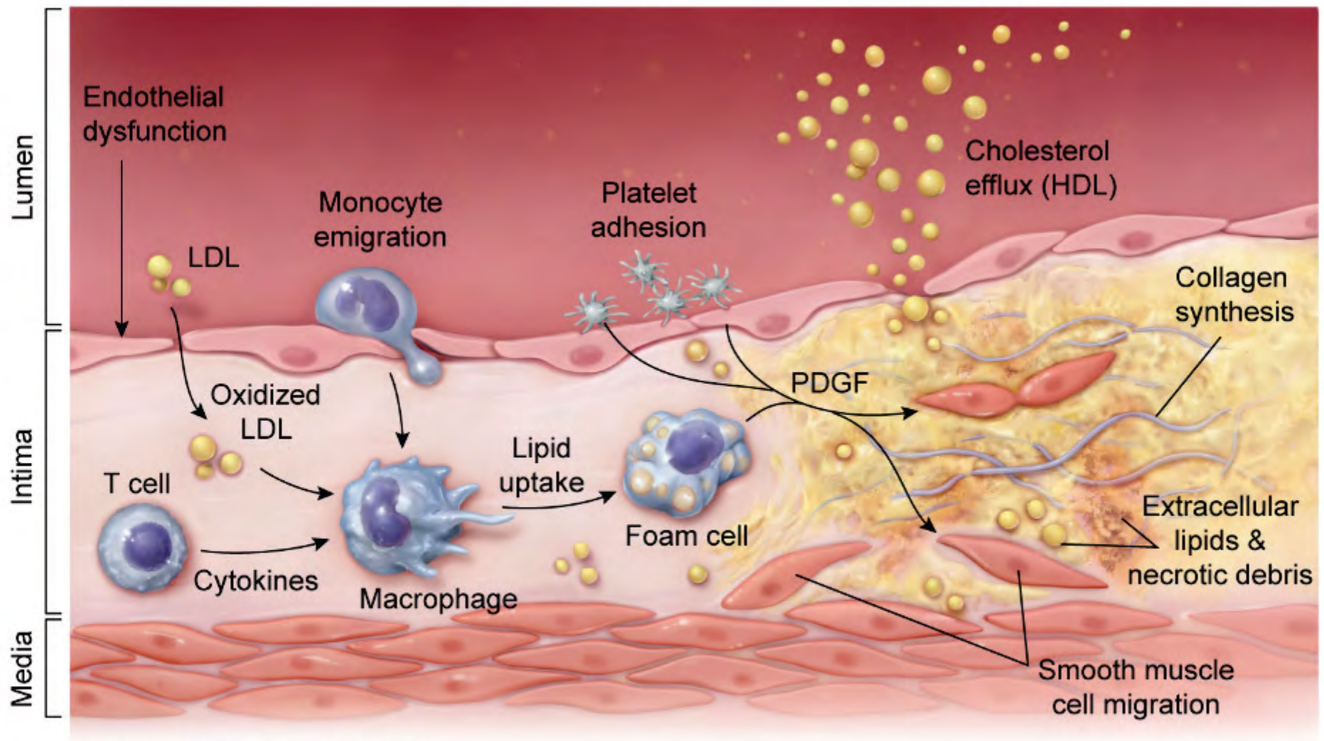
Formation of atheroma



PDGF = platelet-derived growth factor.

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PDGF = platelet-derived growth factor.

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Atherosclerosis is a chronic inflammatory and fibroproliferative disease triggered by **endothelial cell injury** from chronic hemodynamic stress (eg, hypertension), hypercholesterolemia, diabetes mellitus, and/or cigarette smoking. The resulting endothelial cell dysfunction allows adherence and migration of monocytes and T lymphocytes into the intima **(Choices B and D)**. Increased vascular permeability also allows LDL cholesterol into the intima, where it becomes oxidized and is phagocytosed by the accumulating macrophages to produce lipid-laden foam cells **(Choice C)**.

Over time, the infiltrating leukocytes and dysfunctional endothelium (along with adherent activated platelets **[Choice A]**) release cytokines and growth factors (eg, platelet-derived growth factor, fibroblast growth factor, endothelin-1) that promote **migration and proliferation** of medial **smooth muscle cells** (SMCs) into the intima.

This chronic inflammatory state drives further deposition of oxidized LDL cholesterol and proliferation of SMCs with increased production of extracellular matrix proteins (eg, **collagen**, proteoglycans), eventually forming the **fibrous cap** typical of a mature atheroma (ie, fibrofatty plaque). As the atheroma grows larger, the likelihood increases that the fibrous cap will be disrupted (atherosclerotic plaque rupture), followed by rapid luminal thrombosis that has catastrophic clinical consequences (eg, acute coronary syndrome, sudden cardiac death).

Educational objective:
Medial smooth muscle cells are responsible for synthesizing the extracellular matrix proteins (eg, collagen, proteoglycans) that form the fibrous cap of a mature atheroma. Progressive atheroma enlargement increases the likelihood of plaque rupture with resulting luminal thrombosis.

References

- The role of vascular smooth muscle cells on the pathogenesis of atherosclerosis.
- Pathogenesis of atherosclerosis.

Pathology	Cardiovascular System	Coronary artery disease
Subject	System	Topic

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A 4-month-old boy is brought to the cardiology clinic by his parents for follow-up of tetralogy of Fallot. The diagnosis was made during routine antenatal sonography, and the pregnancy and delivery were otherwise uncomplicated. The patient has been seen frequently in the clinic and has had no cyanosis, respiratory distress, or difficulty feeding. The parents become concerned when their son's surgical plan is discussed because he does not have the clinical signs demonstrated by other children with tetralogy of Fallot. Which of the following is the greatest determinant of symptom severity in this condition?

- A. Degree of aortic valve insufficiency
- B. Degree of right ventricular outflow tract obstruction
- C. Degree of tricuspid valve insufficiency
- D. Presence of associated pulmonary parenchymal malformations
- E. Size of ventricular septal defect

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- E. Size of ventricular septal defect

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Correct answer
B

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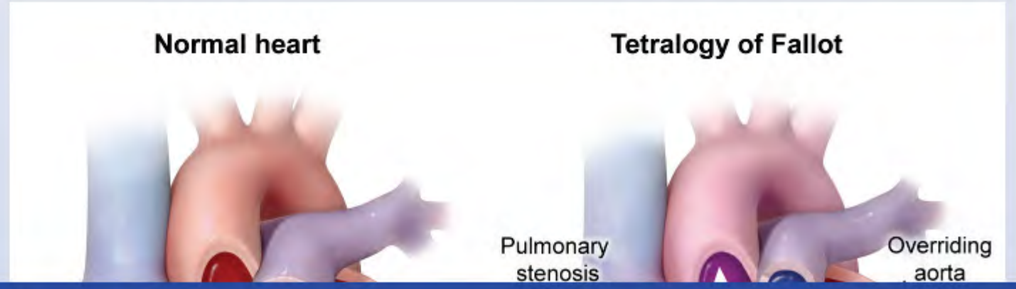
Explanation



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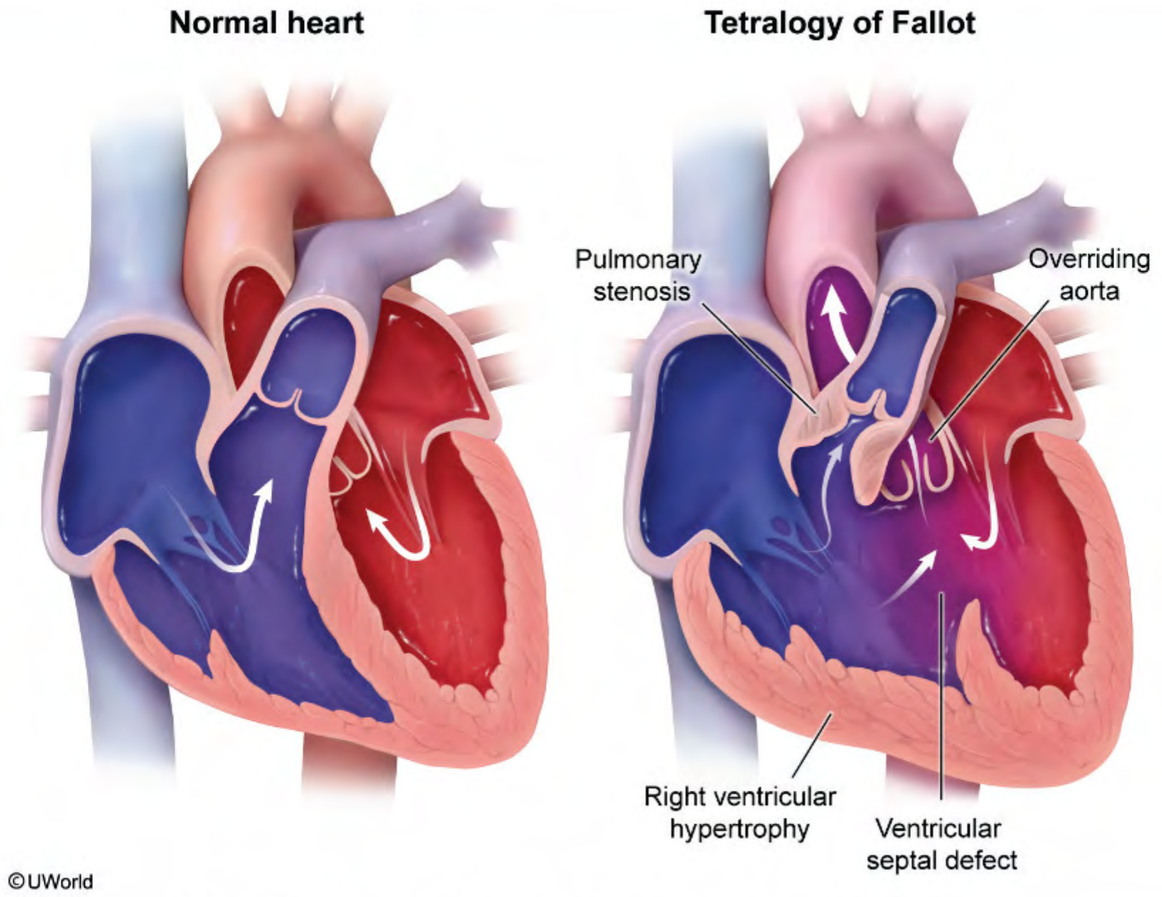

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Tetralogy of Fallot (ToF) is a congenital heart defect characterized by right ventricular outflow tract (RVOT) obstruction, a ventricular septal defect (VSD), an overriding aorta, and right ventricular hypertrophy. Because the VSD is invariably large and unrestrictive, right and left ventricular pressures are generally equal, and the magnitude and direction of shunting through the VSD are determined by the relative resistances of the ventricular outflow tracts **(Choice E)**. Therefore, the **greatest determinant** of symptom severity is the **degree of RVOT obstruction**:

- Severe RVOT obstruction: Insufficient pulmonary arterial flow with obligatory right-to-left ventricular shunting leads to hypoxemia and profound cyanosis requiring medical/surgical intervention in the immediate neonatal period.
- Moderate RVOT obstruction: Moderate obstruction typically permits adequate pulmonary arterial flow and oxygenation. These patients are acyanotic at baseline due to left-to-right or neutral shunting through the VSD. However, some degree of the RVOT obstruction is dynamic, and transient increases in RVOT obstruction (eg, infundibular muscle spasm while feeding or crying) may force the reversal of flow (ie, right-to-left) and cause episodic cyanosis.
- Mild RVOT obstruction: Patients with mild RVOT, which is likely the case in this boy, may have no cyanotic episodes and develop heart failure later in childhood due to pulmonary overcirculation from the high volume of left-to-right shunting via the large VSD.

(Choices A and C) Although the pulmonic valve may be stenotic in patients with ToF (contributing to RVOT obstruction), the aortic and tricuspid valves are typically normal and have no stenosis or insufficiency.

(Choice D) The majority of patients with ToF do not have pulmonary parenchymal or other extracardiac malformations.

Educational objective:

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(Choices A and C) Although the pulmonic valve may be stenotic in patients with ToF (contributing to RVOT obstruction), the aortic and tricuspid valves are typically normal and have no stenosis or insufficiency.

(Choice D) The majority of patients with ToF do not have pulmonary parenchymal or other extracardiac malformations.

Educational objective:
Tetralogy of Fallot is a congenital heart defect characterized by right ventricular outflow tract (RVOT) obstruction, a large ventricular septal defect (VSD), an overriding aorta, and right ventricular hypertrophy. The degree of RVOT obstruction is the greatest determinant of symptom severity because it regulates pulmonary arterial blood flow and the magnitude and direction of shunting through the VSD.

References

- Tetralogy of Fallot.

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leads to hypoxemia and profound cyanosis requiring medical/surgical intervention in the immediate neonatal period.

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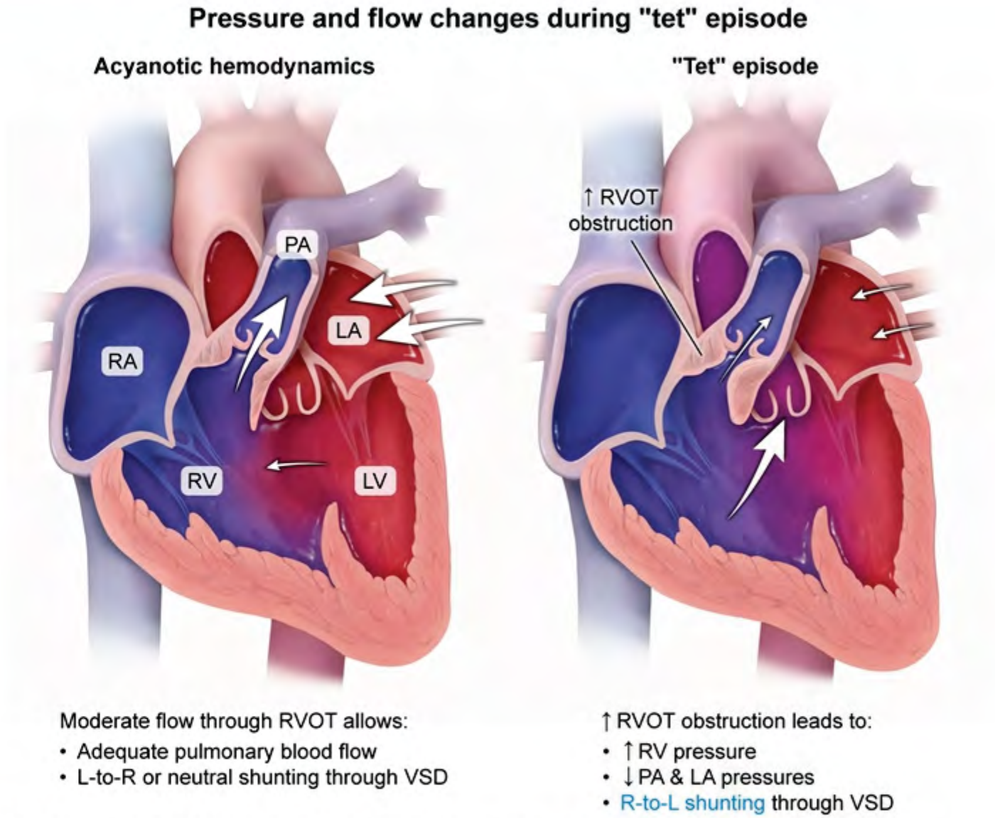

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A 23-year-old man comes to the office due to chest discomfort that usually occurs during exercise, such as jogging or climbing stairs. The symptoms go away 5 to 10 minutes after he stops. The patient has not had syncope but mentions some shortness of breath that accompanies the chest pain. Family history includes an uncle who died suddenly at age 35. Blood pressure is 122/70 mm Hg and pulse is 70/min and regular. The apical impulse is strong and sustained. While supine, the patient has a soft crescendo-decrescendo systolic murmur at the apex and left sternal border; it becomes quite pronounced when he stands. Which of the following medications should be avoided while treating this patient's condition?

- A. Amiodarone
- B. Disopyramide
- C. Isosorbide dinitrate
- D. Metoprolol
- E. Verapamil

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- A. Amiodarone (12%)
- ✗ B. Disopyramide (7%)
- ✓ C. Isosorbide dinitrate (54%)
- D. Metoprolol (14%)
- E. Verapamil (11%)

Incorrect
Correct answer
C

54%
Answered correctly

03 secs
Time Spent

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Explanation

Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	Left ventricular blood volume	Murmur intensity
Valsalva			

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Effect of maneuvers on hypertrophic cardiomyopathy			
	Physiologic effect	**Left ventricular blood volume**	**Murmur intensity**
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

This patient with exertional chest pain, family history of sudden cardiac death, and a crescendo-decrescendo systolic murmur that intensifies on standing most likely has **hypertrophic cardiomyopathy** (HCM), an autosomal dominant inherited condition involving mutations in cardiac sarcomere proteins. The clinical manifestations of HCM result mostly from enlargement of the intraventricular septum that leads to **left ventricular outflow tract (LVOT) obstruction**. Patients may be asymptomatic or can experience exertional dyspnea, fatigue, anginal pain, lightheadedness, or syncope; sudden cardiac arrest (or death) can occur due to reduced cardiac output and ventricular arrhythmia.

The degree of LVOT obstruction in HCM varies with changes in left ventricular (LV) blood volume. Low LV blood

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Passive leg raise	↑ Preload		

This patient with exertional chest pain, family history of sudden cardiac death, and a crescendo-decrescendo systolic murmur that intensifies on standing most likely has **hypertrophic cardiomyopathy** (HCM), an autosomal dominant inherited condition involving mutations in cardiac sarcomere proteins. The clinical manifestations of HCM result mostly from enlargement of the intraventricular septum that leads to **left ventricular outflow tract (LVOT) obstruction**. Patients may be asymptomatic or can experience exertional dyspnea, fatigue, anginal pain, lightheadedness, or syncope; sudden cardiac arrest (or death) can occur due to reduced cardiac output and ventricular arrhythmia.

The degree of LVOT obstruction in HCM varies with changes in left ventricular (LV) blood volume. Low LV blood volume collapses the LVOT and worsens the obstruction; therefore, **medications that decrease LV blood volume** should be **avoided**. These medications include the following:

- **Preload reducers:** Venous dilators (eg, **nitrates**) and diuretics (eg, furosemide) reduce venous return to the heart and decrease LV end-diastolic volume.
- **Afterload reducers:** Arterial dilators (eg, hydralazine) facilitate increased stroke volume, which decreases LV end-systolic volume.
- **Balanced vasodilators:** Combined venous and arterial dilators (eg, dihydropyridine calcium channel blockers, ACE inhibitors) can worsen LVOT obstruction via reductions in preload and afterload.

In contrast, negative chronotropic agents (which reduce heart rate, increasing diastolic filling time) and negative inotropic agents (which reduce contractility, decreasing the systolic ejection fraction) help improve LVOT obstruction by increasing LV volumes. Beta blockers (eg, metoprolol) and nondihydropyridine calcium channel blockers (eg, verapamil) have negative chronotropic and inotropic effects and are used for treating HCM **(Choices D and E)**. Disopyramide, a class I antiarrhythmic (ie, sodium channel blocker) that has negative inotropic effects, is also effective via the same mechanism **(Choice B)**.

(Choice A) Amiodarone is a class III antiarrhythmic (potassium channel blocker) that may be used in patients with

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obstruction. Patients may be asymptomatic or can experience exertional dyspnea, fatigue, anginal pain, lightheadedness, or syncope; sudden cardiac arrest (or death) can occur due to reduced cardiac output and ventricular arrhythmia.

The degree of LVOT obstruction in HCM varies with changes in left ventricular (LV) blood volume. Low LV blood volume collapses the LVOT and worsens the obstruction; therefore, **medications that decrease LV blood volume** should be **avoided**. These medications include the following:

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(Choice A) Amiodarone is a class III antiarrhythmic (potassium channel blocker) that may be used in patients with HCM who develop arrhythmia complications (eg, atrial fibrillation, ventricular tachycardia).

Educational objective:
Left ventricular outflow tract obstruction in hypertrophic cardiomyopathy worsens with decreased left ventricular blood volume. Therefore, medications that decrease preload (eg, nitrates), afterload (eg, hydralazine), or both (eg, dihydropyridine calcium channel blockers, ACE inhibitors) should be avoided.

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A 62-year-old, obese woman is brought to the hospital due to acute-onset chest pain and shortness of breath. Medical history includes hyperlipidemia, diet-controlled type 2 diabetes mellitus, and peripheral artery disease. Two years ago, she had an ischemic stroke but had no residual neurologic deficits. The patient is a current smoker with a 30-pack-year history. After initial evaluation, she is taken to the cardiac catheterization laboratory where she has a cardiac arrest due to ventricular arrhythmia and cannot be resuscitated. Autopsy reveals thrombotic occlusion of the left anterior descending artery. In addition, it shows 95% atherosclerotic narrowing of the proximal right renal artery but no significant stenosis of the left renal artery. Compared with the right kidney, this patient's left kidney is more likely to demonstrate which of the following findings?

- A. Arteriolar wall thickening
- B. Atrophic tubules
- C. Cortical thinning
- D. Interstitial fibrosis
- E. Small crowded glomeruli

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A 62-year-old, obese woman is brought to the hospital due to acute-onset chest pain and shortness of breath. Medical history includes hyperlipidemia, diet-controlled type 2 diabetes mellitus, and peripheral artery disease. Two years ago, she had an ischemic stroke but had no residual neurologic deficits. The patient is a current smoker with a 30-pack-year history. After initial evaluation, she is taken to the cardiac catheterization laboratory where she has a cardiac arrest due to ventricular arrhythmia and cannot be resuscitated. Autopsy reveals thrombotic occlusion of the left anterior descending artery. In addition, it shows 95% atherosclerotic narrowing of the proximal right renal artery but no significant stenosis of the left renal artery. Compared with the right kidney, this patient's left kidney is more likely to demonstrate which of the following findings?

- ✓ A. Arteriolar wall thickening (53%)
- B. Atrophic tubules (7%)
- C. Cortical thinning (9%)
- D. Interstitial fibrosis (4%)
- E. Small crowded glomeruli (24%)

Correct

53% Answered correctly

03 secs Time Spent

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Explanation

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A 62-year-old, obese woman is brought to the hospital due to acute-onset chest pain and shortness of breath.
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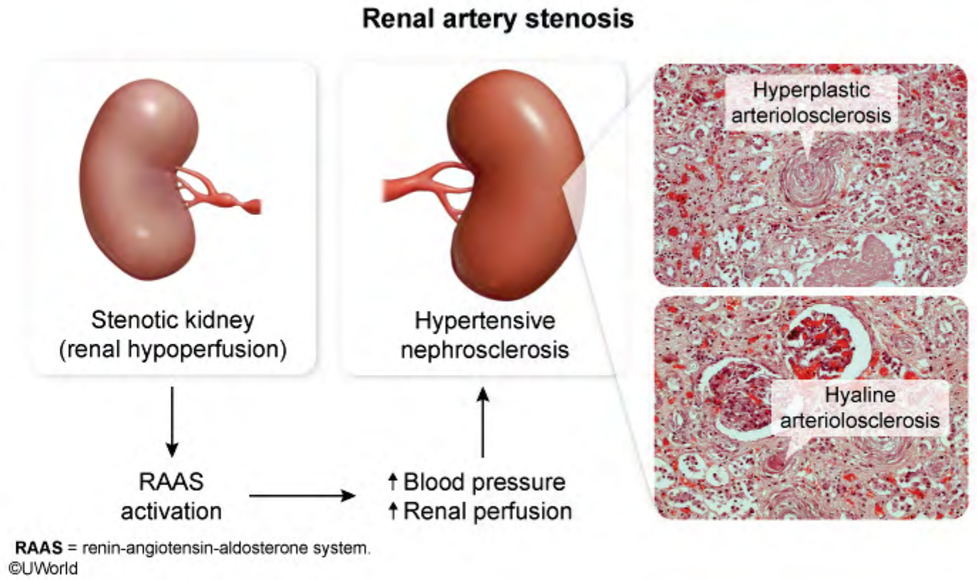

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This patient's autopsy findings are consistent with severe, **unilateral renal artery stenosis** (RAS), a condition that occurs most commonly in older individuals due to atherosclerosis. Hypoperfusion of the stenotic (right) kidney stimulates **renin release** by juxtaglomerular apparatus cells. Renin converts angiotensinogen to angiotensin I, which is then converted by angiotensin-converting enzyme to angiotensin II, a potent vasoconstrictor that causes **systemic hypertension**.

Unilateral RAS causes morphologic changes that markedly differ in the stenotic (right) kidney and the nonstenotic (left) kidney. Narrowing of the renal artery in the **stenotic (right) kidney** leads to hypoperfusion and **renal parenchymal ischemia**, which manifests as diffuse cortical thinning, tubular atrophy, interstitial fibrosis, and small crowded glomeruli. Juxtaglomerular apparatus enlargement can also occur due to chronic stimulation and increased renin release.

In contrast, the contralateral, **nonstenotic (left) kidney** is exposed to **high blood pressure** and therefore demonstrates typical signs of hypertensive nephrosclerosis, which is characterized by **arteriolar wall thickening** due to **hyaline** arteriolosclerosis (hyalinization of the arterioles due to extravasation of plasma proteins) and **hyperplastic arteriolosclerosis** (concentric smooth muscle cell proliferation in response to pressure ["onion-skinning"]).

(Choices B, C, D, and E) With unilateral RAS, the nonstenotic (left) kidney develops significant arteriolosclerosis due to exposure to high blood pressure. This can lead to narrowing of the arteriolar lumen with subsequent ischemic changes. However, these ischemic findings would be much more prominent in the stenotic (right) kidney.

Educational objective:
In unilateral renal artery stenosis, the narrowed renal artery causes hypoperfusion of the affected kidney with subsequent ischemic damage (eg, tubular atrophy, interstitial ischemia, glomerular crowding). In contrast, the contralateral kidney is exposed to high blood pressure and typically shows changes of hypertensive nephrosclerosis (eg, arteriolar wall thickening due to hyaline or hyperplastic arteriolosclerosis).

Pathology	Cardiovascular System	Renal artery stenosis
Subject	System	Topic

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Pathology	Cardiovascular System	Renal artery stenosis
Subject	System	Topic

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A 54-year-old man comes to the office due to episodic burning substernal chest pain. His pain increases with activity and improves with rest. The patient has a history of hypertension and hyperlipidemia. He has smoked a pack of cigarettes daily for the past 30 years. His blood pressure is 140/85 mm Hg and pulse is 76/min. Cardiac auscultation reveals a S4 heart sound. Lung and abdominal examinations are unremarkable. An ECG at rest shows left ventricular hypertrophy. A myocardial perfusion scan reveals inducible ischemia of the inferior surface of the heart. Which of the following coronary arteries is most likely occluded in this patient?

- A. Left anterior descending coronary artery
- B. Left circumflex coronary artery
- C. Left main coronary artery
- D. Right coronary artery
- E. Right marginal branch

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- ✗ A. Left anterior descending coronary artery (25%)
- B. Left circumflex coronary artery (8%)
- C. Left main coronary artery (1%)
- ✓ D. Right coronary artery (52%)
- E. Right marginal branch (11%)

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Correct answer
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52%
Answered correctly

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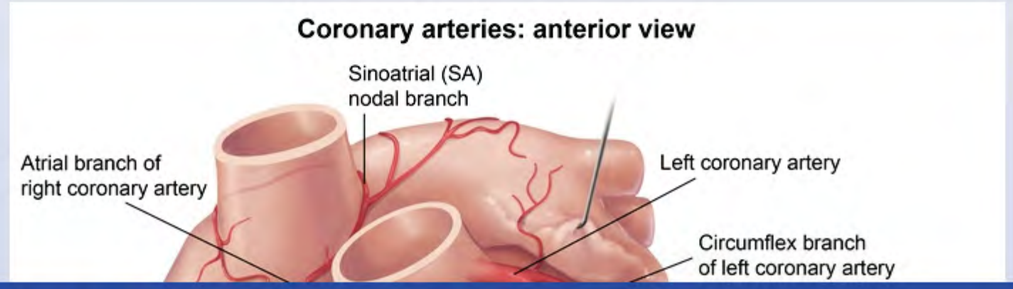
Explanation



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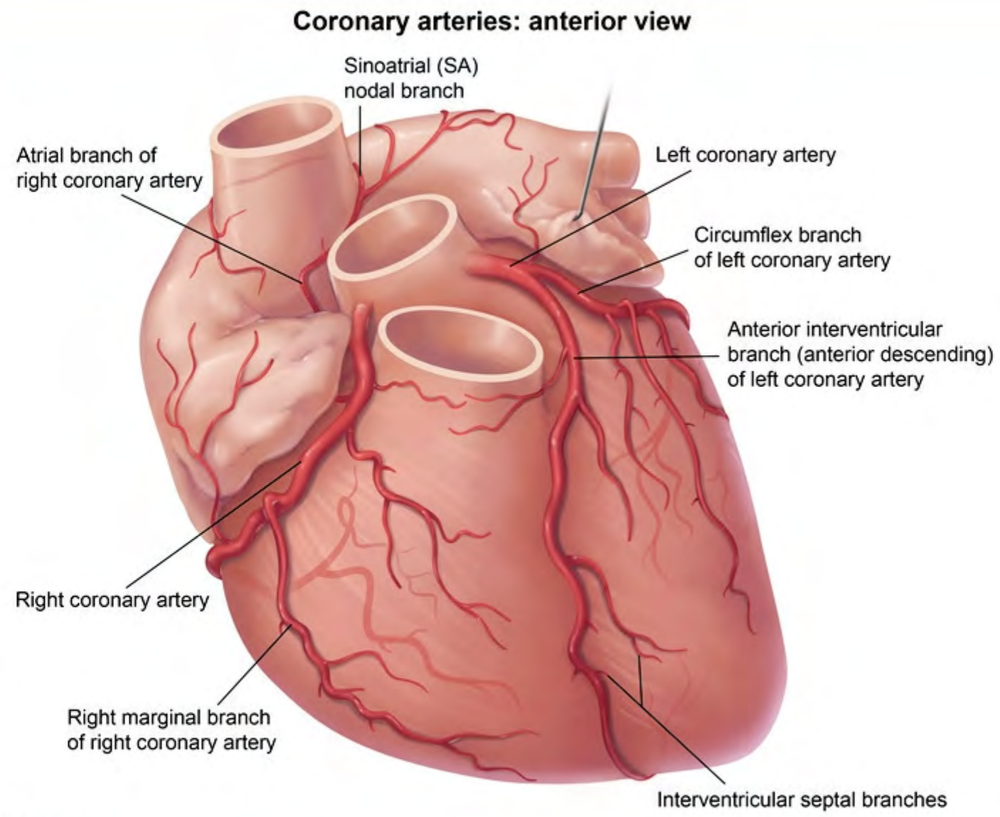

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Circumflex branch
of left coronary artery

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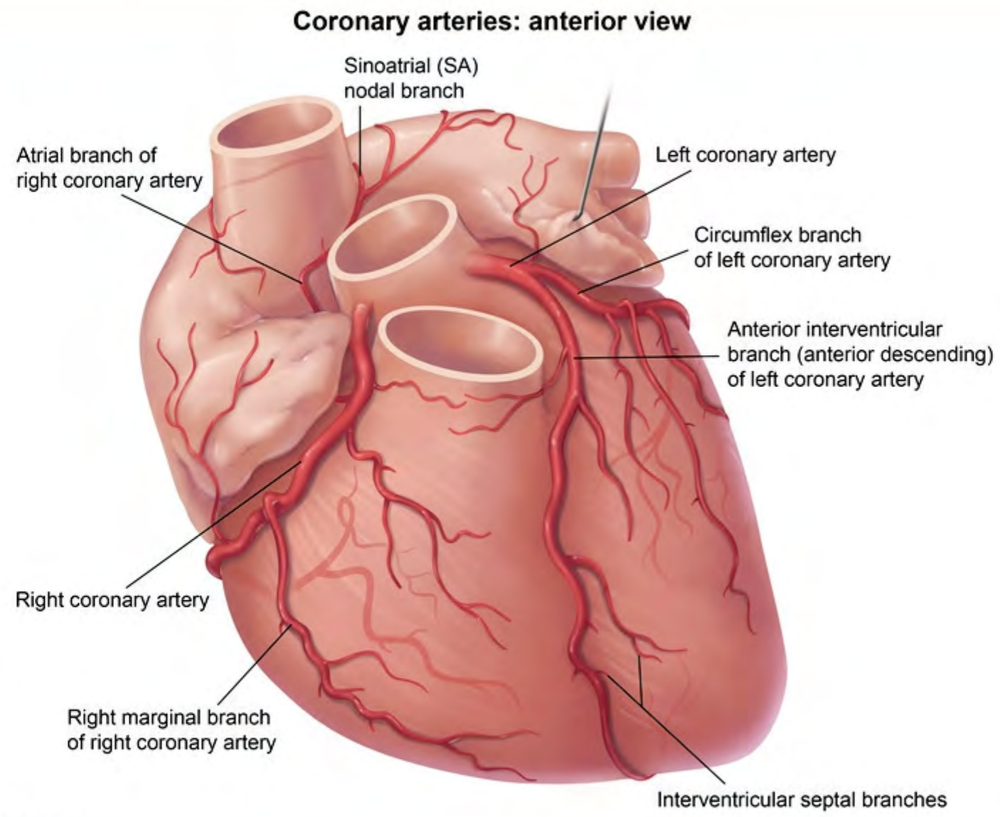

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(Choice B) The left circumflex coronary artery usually supplies the lateral and posterior superior walls of the left

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Right coronary artery

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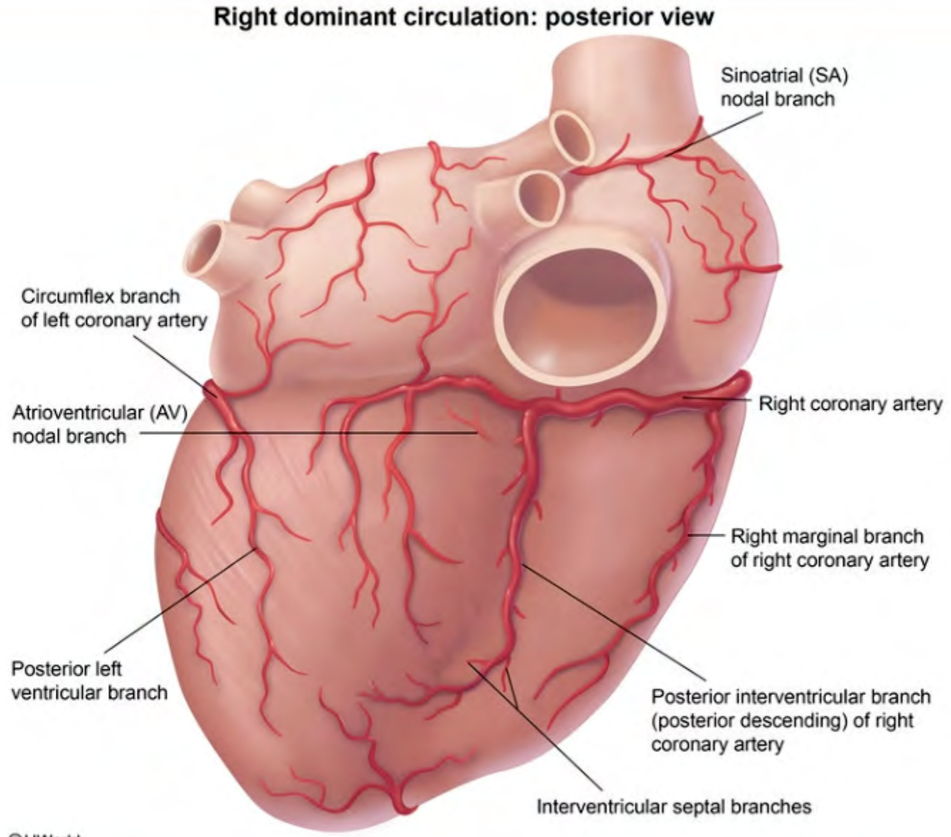

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(Choice C) In approximately 10% of individuals, the posterior descending artery derives from the circumflex branch of the left main coronary artery (left dominant circulation); therefore, the left main coronary artery is not the

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Click here for posterior view

The right and left main coronary arteries arise directly from the root of the aorta and provide the blood supply to the heart. The left main coronary artery divides into the left anterior descending (LAD) and circumflex coronary arteries, which supply most of the anterior and left lateral surfaces of the heart.

In 85%-90% of individuals, the **right coronary artery** gives rise to the **posterior descending artery**. Such patients are said to have **right dominant** coronary circulation. The posterior descending artery runs down the posterior interventricular groove and supplies the posterior one third of the interventricular septum and most of the inferior wall of the left ventricle.

The **inferior (diaphragmatic) surface** of the heart is formed by the left ventricle (two thirds) and right ventricle (one third), which are separated by the posterior interventricular groove. Because most individuals have right dominant circulation, occlusion of the right coronary artery is most likely to cause inducible ischemia of the inferior surface of the heart.

(Choice A) The LAD artery normally supplies the anterior two thirds of the interventricular septum (septal branches), the anterior wall of the left ventricle (diagonal branches), and part of the anterior papillary muscle.

(Choice B) The left circumflex coronary artery usually supplies the lateral and posterior superior walls of the left ventricle via obtuse marginal branches.

(Choice C) In approximately 10% of individuals, the posterior descending artery derives from the circumflex branch of the left main coronary artery (left dominant circulation); therefore, the left main coronary artery is not the artery most likely occluded in this patient.

(Choice E) The right marginal branch of the right coronary artery supplies the wall of the right ventricle and may provide collateral circulation in patients with LAD occlusion.

Educational objective:
The inferior wall of the left ventricle forms most of the inferior (diaphragmatic) surface of the heart and is supplied by the posterior descending artery. In 85%-90% of individuals, the posterior descending artery derives from the

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References

- Anatomy, histology, and pathology of coronary arteries: A review relevant to new interventional and imaging

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A 74-year-old man comes to the office for a follow-up visit for hypertension. His last visit was a year ago. He has no chest pain, shortness of breath, leg swelling, or dizziness. The patient is compliant with his medications and can tolerate a moderate level of physical activity. Blood pressure is 145/75 mm Hg and pulse is 67/min and regular. Auscultation findings at the base of the heart are depicted in the exhibit below.

Which of the following is the most likely cause of this patient's physical findings?

- A. Ankylosing spondylitis
- B. Chordal rupture
- C. Coronary artery disease
- D. Cystic medial necrosis
- E. Infective endocarditis
- F. Rheumatic fever
- G. Right ventricular failure
- H. Valvular calcification

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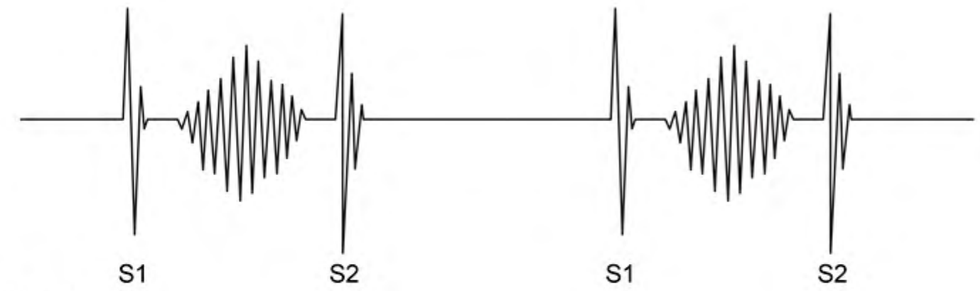

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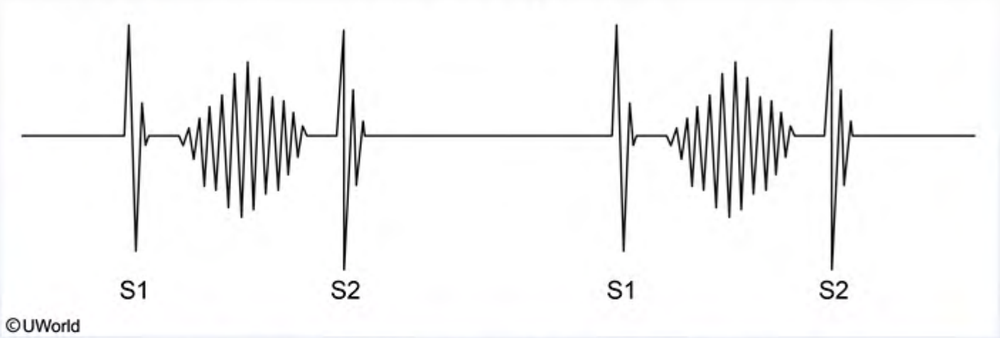

Which of the following is the most likely cause of this patient's physical findings?

- A. Ankylosing spondylitis (0%)
- B. Chordal rupture (3%)
- C. Coronary artery disease (2%)
- D. Cystic medial necrosis (1%)
- E. Infective endocarditis (1%)
- F. Rheumatic fever (5%)
- G. Right ventricular failure (1%)
- ✓ H. Valvular calcification (84%)

Correct

84%
Answered correctly

54 secs
Time Spent

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The diagram above depicts a **crescendo-decrescendo systolic murmur** peaking in midsystole, characteristic of **aortic or pulmonic stenosis**. The most common cause of aortic stenosis (AS) in elderly patients (age ≥70) is degenerative calcification of the aortic valve leaflets. **Calcific AS** is caused by chronic hemodynamic stress or atherosclerotic inflammation that eventually leads to progressive aortic valve leaflet thickening, calcification, and stenosis. The murmur of valvular AS is best heard over **the right second interspace** (where the aorta leaves the base of the heart) with radiation to neck and carotid arteries. A longer and late-peaking murmur usually indicates severe AS, whereas a brief and early-peaking murmur indicates less severe stenosis. However, the intensity of the murmur does not correlate with the severity of stenosis.

(Choice A) Ankylosing spondylitis is a chronic inflammatory disease of the axial skeleton characterized by progressive pain and stiffness of the spine, sacroiliitis, and increased risk of aortic regurgitation.

(Choice B) Chordal rupture would lead to flail mitral leaflet with mitral valve prolapse and mitral regurgitation. Mitral regurgitation associated with chordal rupture would lead to a holosystolic decrescendo murmur.

(Choice C) Coronary artery disease is not associated with a systolic murmur on auscultation.

(Choice D) Cystic medial necrosis predisposes to the formation of thoracic aortic aneurysms with an increased risk of aortic dissection. It can also cause dilation of the aortic root leading to aortic regurgitation.

(Choice E) Infectious endocarditis of the aortic or mitral valve typically causes aortic or mitral regurgitation, respectively. Aortic regurgitation leads to an early diastolic murmur, best heard over the lower left sternal border or right second interspace with the patient sitting and leaning forward with breath held in full expiration. Mitral regurgitation causes a holosystolic murmur best heard at the cardiac apex in the left lateral decubitus position.

(Choice F) Rheumatic heart disease is the most common cause of AS in the developing world and is invariably accompanied by rheumatic mitral valve disease (mitral stenosis and/or regurgitation). It is an uncommon cause of AS in developed nations.

(Choice G) Right ventricular failure leads to jugular venous distension, along with hepatomegaly, ascites, and peripheral edema. Significant right ventricular dilation can cause tricuspid insufficiency, which is characterized by a

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(Choice G) Right ventricular failure leads to jugular venous distension, along with hepatomegaly, ascites, and peripheral edema. Significant right ventricular dilation can cause tricuspid insufficiency, which is characterized by a holosystolic murmur at the left lower sternal border.

Educational objective:
The murmur of valvular aortic stenosis is typically an ejection or midsystolic murmur of crescendo-decrescendo configuration with maximum intensity over the right second interspace and radiation to neck and carotid arteries. The most common cause of aortic stenosis in elderly patients (age ≥70) is degenerative calcification of the aortic valve leaflets.

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A 56-year-old woman is evaluated for fatigue and dyspnea on exertion. Past medical history includes hypertension and systemic sclerosis. Blood pressure is 135/80 mm Hg and pulse is 68/min. Cardiac examination reveals loud second heart sounds with no murmurs. Lungs are clear to auscultation. Further evaluation with a catheterization procedure is performed, during which a balloon-tipped catheter is advanced into the pulmonary artery. A branch of the pulmonary artery is occluded by the balloon, and the pressure beyond the point of occlusion is measured. The pressure reading from the procedure most likely corresponds to which of the following pressures?

- A. Intrapleural pressure
- B. Left atrial pressure
- C. Mean airway pressure
- D. Mean arterial pressure
- E. Pulse pressure
- F. Right atrial pressure
- G. Right ventricular systolic pressure

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- A. Intrapleural pressure (2%)
- ✓ B. Left atrial pressure (78%)
- C. Mean airway pressure (1%)
- D. Mean arterial pressure (2%)
- E. Pulse pressure (0%)
- F. Right atrial pressure (6%)
- G. Right ventricular systolic pressure (8%)

Correct | 78% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Pulmonary artery catheters (PACs; also called Swan-Ganz or right heart catheters) are used to diagnose pulmonary hypertension and occasionally for management of critically ill patients. During pulmonary artery catheterization, the balloon at the distal tip of the catheter is inflated, and the catheter is advanced forward through the right atrium, right ventricle, and pulmonary artery and finally into a branch of the pulmonary artery. Once

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Explanation

Pulmonary artery catheters (PACs; also called Swan-Ganz or right heart catheters) are used to diagnose pulmonary hypertension and occasionally for management of critically ill patients. During pulmonary artery catheterization, the balloon at the distal tip of the catheter is inflated, and the catheter is advanced forward through the right atrium, right ventricle, and pulmonary artery and finally into a branch of the pulmonary artery. Once lodged in a pulmonary artery branch, the inflated balloon obstructs forward blood flow, creating a continuous static column of blood between the catheter tip and left atrium. The pressure measured at the catheter tip at this time is called the **pulmonary artery occlusion pressure** (PAOP; or pulmonary capillary wedge pressure [PCWP]) and closely reflects left atrial and left ventricular end-diastolic pressures.

(Choices A and C) Direct measurement of intrapleural pressure (pleural manometry) can be obtained by placing a catheter in the pleural space during thoracentesis, whereas mean airway pressure is measured during mechanical ventilation. PACs do not measure intrapleural or mean airway pressure.

(Choice D) Mean arterial pressure is measured by placing an arterial catheter directly in central arterial circulation (common femoral or radial artery) and reflects the average aortic pressure (perfusion pressure).

(Choice E) Pulse pressure refers to the difference between systemic arterial systolic and diastolic blood pressure.

(Choices F and G) Right atrial and right ventricular systolic pressures can be measured directly by PACs by placing the catheter in the respective chambers.

Educational objective:
Pulmonary artery occlusion pressure is measured at the distal tip of the pulmonary artery catheter after an inflated balloon occludes blood flow through a pulmonary artery branch. It closely corresponds to left atrial and left ventricular end-diastolic pressure.

References

- Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter.

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Explanation

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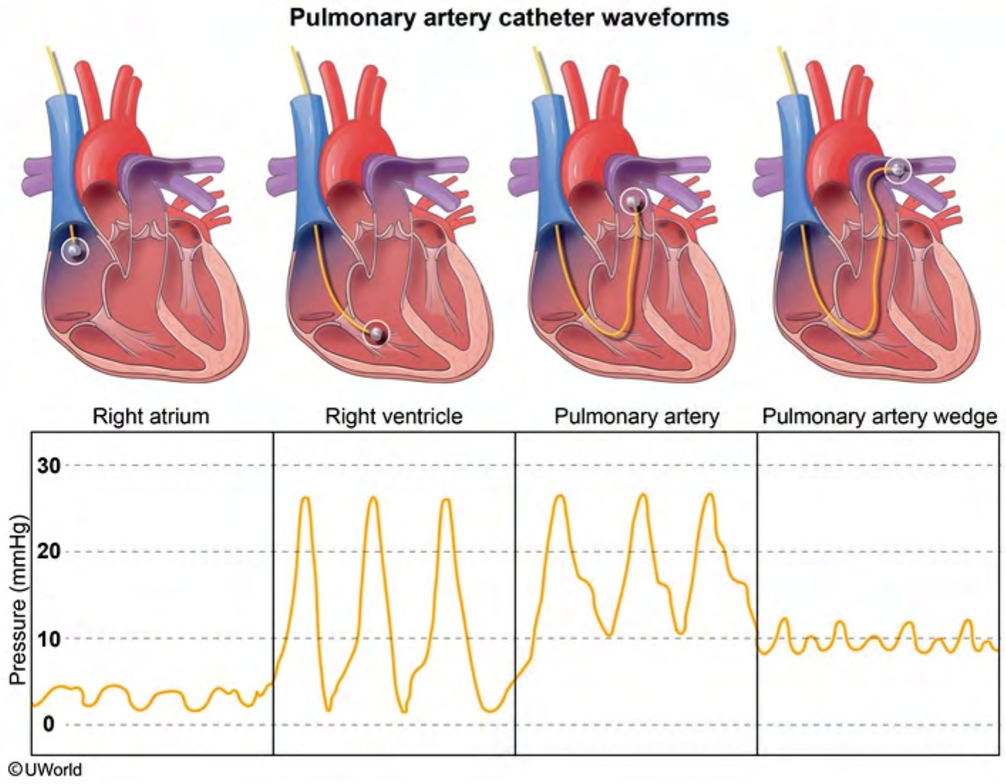

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- Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter.

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A 62-year-old man comes to the office for follow-up of hypertension. He was diagnosed with hypertension 10 years ago and has been treated with a number of different medications. However, the patient has had to discontinue several medications due to side effects such as dizziness, palpitations, and headaches. Currently he takes ramipril and chlorthalidone and is tolerating them well. Blood pressure is 160/92 mm Hg and was 158/89 mm Hg at his most recent prior visit. ECG shows sinus bradycardia (55/min) with PR interval prolongation (280 msec). Which of the following medications would be most effective for lowering this patient's blood pressure without worsening his ECG abnormalities?

- A. Diltiazem
- B. Metoprolol
- C. Nifedipine
- D. Sotalol
- E. Verapamil

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- A. Diltiazem (5%)
- ✗ B. Metoprolol (9%)
- ✓ C. Nifedipine (71%)
- D. Sotalol (7%)
- E. Verapamil (6%)

Incorrect
Correct answer
C

71%
Answered correctly

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Explanation

Effects of calcium channel blockers		
Drug	**Location of action**	**Physiologic effects**
Dihydropyridines (amlodipine, felodipine,	Vascular smooth muscle	Peripheral vasodilation

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Effects of calcium channel blockers		
Drug	**Location of action**	**Physiologic effects**
Dihydropyridines (amlodipine, felodipine, nifedipine)	Vascular smooth muscle	Peripheral vasodilation
Verapamil	Cardiac muscle	Reduced heart rate & myocardial contractility
Diltiazem	Vascular smooth muscle & cardiac muscle	Combined effects

Calcium channel blockers are frequently used in the treatment of several cardiovascular disorders, including hypertension, angina pectoris, and certain cardiac arrhythmias. They inhibit the L-type calcium channel on vascular smooth muscle and cardiac cells and are divided into 2 major categories based on their predominant actions:

1. **Dihydropyridines** (nifedipine, amlodipine, felodipine) primarily affect **arterial smooth muscle**, causing vasodilation with little or no effect on cardiac conduction or contractility.
2. **Nondihydropyridines** (verapamil, diltiazem) affect the **myocardium**, slowing heart rate (negative chronotropic effect) and reducing contractility (negative inotropic effect).

This patient's ECG shows sinus bradycardia (rate <60/min) with first-degree atrioventricular (AV) block (PR interval >200 msec). Of the choices above, nifedipine is the most appropriate agent to manage hypertension as it has minimal effect on cardiac conduction. Dihydropyridines can cause reflex tachycardia in response to peripheral

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vascular smooth muscle and cardiac cells and are divided into 2 major categories based on their predominant actions:

1. **Dihydropyridines** (nifedipine, amlodipine, felodipine) primarily affect **arterial smooth muscle**, causing vasodilation with little or no effect on cardiac conduction or contractility.
2. **Nondihydropyridines** (verapamil, diltiazem) affect the **myocardium**, slowing heart rate (negative chronotropic effect) and reducing contractility (negative inotropic effect).

This patient's ECG shows sinus bradycardia (rate <60/min) with first-degree atrioventricular (AV) block (PR interval >200 msec). Of the choices above, nifedipine is the most appropriate agent to manage hypertension as it has minimal effect on cardiac conduction. Dihydropyridines can cause reflex tachycardia in response to peripheral vasodilation and are therefore useful in hypertensive patients with resting bradycardia.

(Choices A and E) Verapamil is a nondihydropyridine calcium channel blocker with a significant depressive effect on cardiac conduction. Diltiazem has combined effects and acts at both the myocardium and peripheral arterial beds. These medications can worsen bradycardia and AV block and should not be used in patients with underlying conduction abnormalities.

(Choices B and D) Metoprolol is a cardioselective beta blocker with negative chronotropic and dromotropic (decreased AV conduction) effects. Sotalol is a class III antiarrhythmic drug with significant beta-blocking properties. These drugs can worsen bradycardia and AV conduction defects and would not be recommended for this patient.

Educational objective:
Calcium channel blockers inhibit the L-type calcium channel on vascular smooth muscle and cardiac cells. Dihydropyridines (eg, nifedipine, amlodipine) primarily affect peripheral arteries and cause vasodilation. Nondihydropyridines (eg, verapamil, diltiazem) affect the myocardium and can cause bradycardia and slowed atrioventricular conduction.

References

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A 50-year-old man comes to the emergency department due to intermittent palpitations over the last 6 months. The patient describes them as "very uncomfortable" and says the episodes occur randomly, last for several hours, and resolve spontaneously or with sleep. The current episode started an hour ago and has been accompanied by lightheadedness. He has no medical history. Temperature is normal and blood pressure is 122/75 mm Hg. BMI is 30 kg/m^2. Chest examination reveals no murmurs and the lungs are clear on auscultation. There is trace peripheral edema. Cardiac rhythm strip obtained in the emergency department is shown in the exhibit. Aberrant electrical activity in which of the following anatomic structures is the most likely trigger for this patient's current condition?

- A. Atrioventricular node
- B. Crista terminalis
- C. Papillary muscles
- D. Pulmonary veins
- E. Right ventricular outflow tract
- F. Tricuspid valve annulus

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- A. Atrioventricular node (34%)
- ✗ B. Crista terminalis (13%)
- C. Papillary muscles (1%)
- ✓ D. Pulmonary veins (37%)
- E. Right ventricular outflow tract (2%)
- F. Tricuspid valve annulus (10%)

Incorrect
Correct answer
D

37%
Answered correctly

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Explanation

Pulmonary vein ablation for atrial fibrillation

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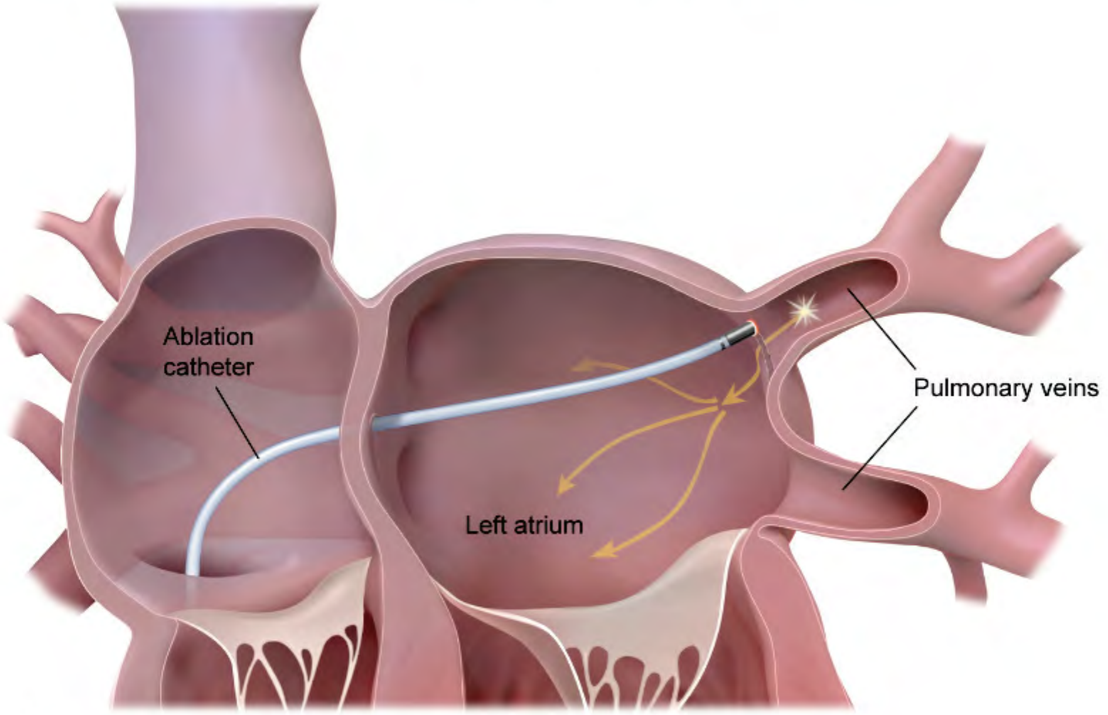
Pulmonary vein ablation for atrial fibrillation



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This patient's presentation with intermittent palpitations and cardiac rhythm strip showing an **irregularly irregular rhythm** with varying R-R intervals and **no discernible P waves** is consistent with **paroxysmal atrial fibrillation** (AF).

The development of AF involves structural and electrical changes that predispose to the initiation and maintenance of electrical reentrant circuits and/or ectopic foci. These pathologic changes, termed **atrial remodeling**, can occur as a result of normal aging or diseases that cause atrial dilation (eg, hypertension, mitral regurgitation). The remodeling leads to shortening of the atrial refractory period and heterogeneity in localized electrical conduction between atrial myocytes, creating a **substrate** for the development of AF.

In predisposed patients, AF can spawn from specific triggers such as focal electrical discharges, increased atrial stretch, elevated sympathetic activity (eg, sepsis, emotional stress), and ingestion of alcohol. The most common trigger is aberrant electrical foci in the **pulmonary veins** near their ostia into the left atrium; therefore, catheter ablation of pulmonary vein trigger sites (pulmonary vein isolation) is used for the treatment of symptomatic, paroxysmal AF.

(Choice A) Aberrant electrical conduction in the atrioventricular node is the cause of atrioventricular nodal reentrant tachycardia (AVNRT). ECG during AVNRT shows a rapid rate with regular R-R intervals and often nonvisible P waves because they are buried within the QRS complexes.

(Choices B and F) The cavotricuspid isthmus is the area of right atrial tissue between the crista terminalis (ridge that runs along the vena cava) and the tricuspid annulus, and is usually part of the reentry circuit in atrial flutter. ECG during atrial flutter typically shows a saw-tooth pattern of flutter waves, and the ventricular rhythm can be regular or irregular.

(Choices C and E) Monomorphic ventricular tachycardia most commonly originates from areas of ischemic myocardium; however, it can sometimes originate from nonischemic sites in the papillary muscles or right ventricular outflow tract. ECG of ventricular tachycardia typically demonstrates regular R-R intervals and wide QRS complexes.

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Educational objective:
Atrial fibrillation is recognized by an irregularly irregular rhythm with variable R-R intervals and absence of P waves on ECG. The development of AF most commonly involves ectopic electrical foci in the pulmonary veins that trigger fibrillatory conduction in abnormal (remodeled) atrial tissue.

References

- Atrial fibrillation: classification, pathophysiology, mechanisms, and drug treatment.

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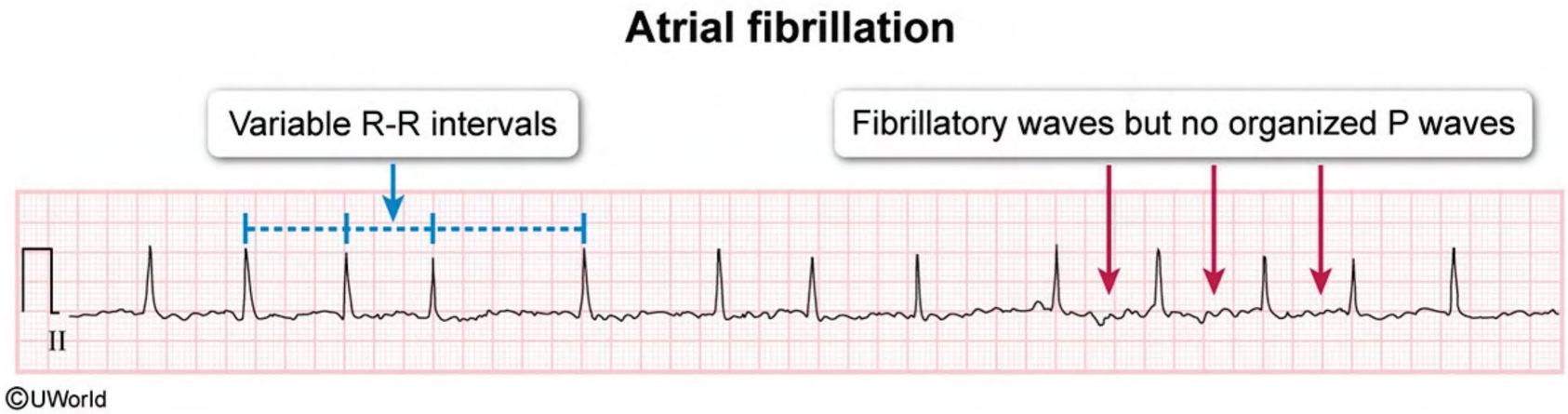

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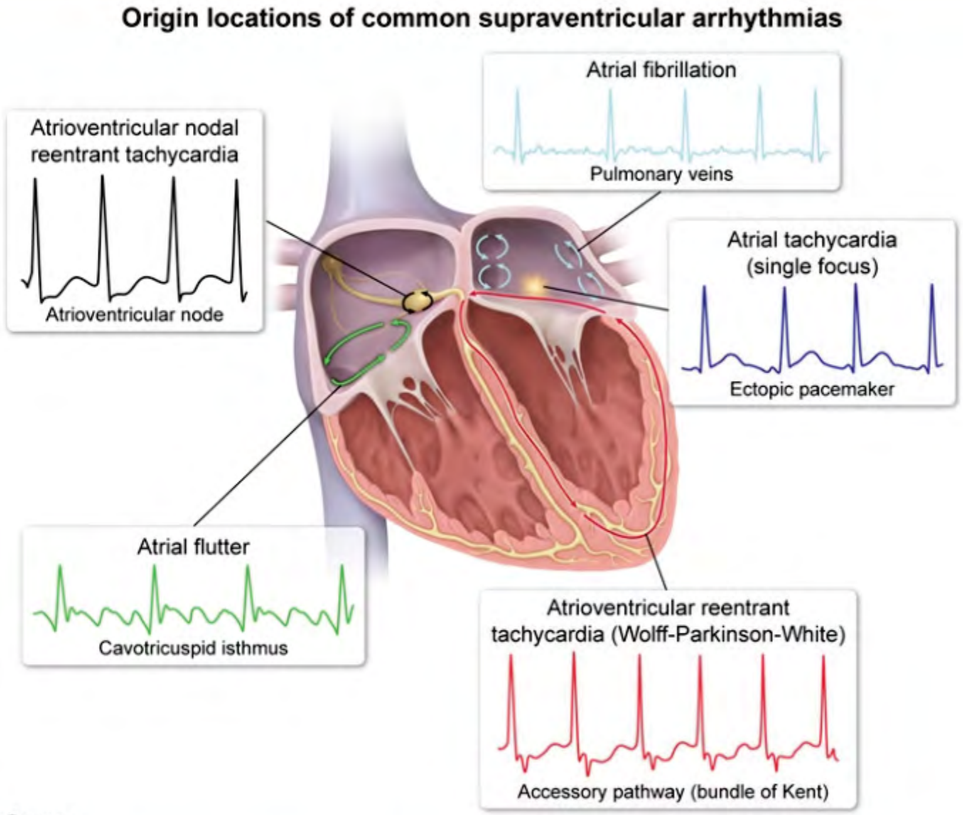

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Atrioventricular nodal reentrant tachycardia (AVNRT)

Pathophysiology	Key features
• Dual AV node conduction pathways (fast & slow) creating a reentrant circuit • Triggered by a premature atrial contraction (PAC)	• 120-180 bpm • Regular R-R intervals • P waves buried within the QRS complexes • Narrow QRS complexes

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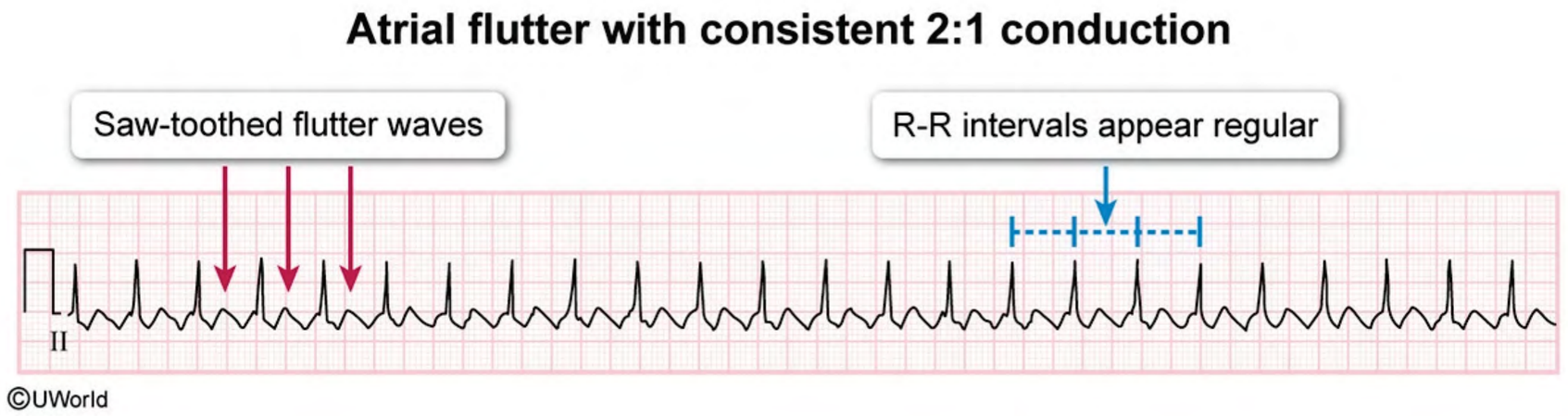

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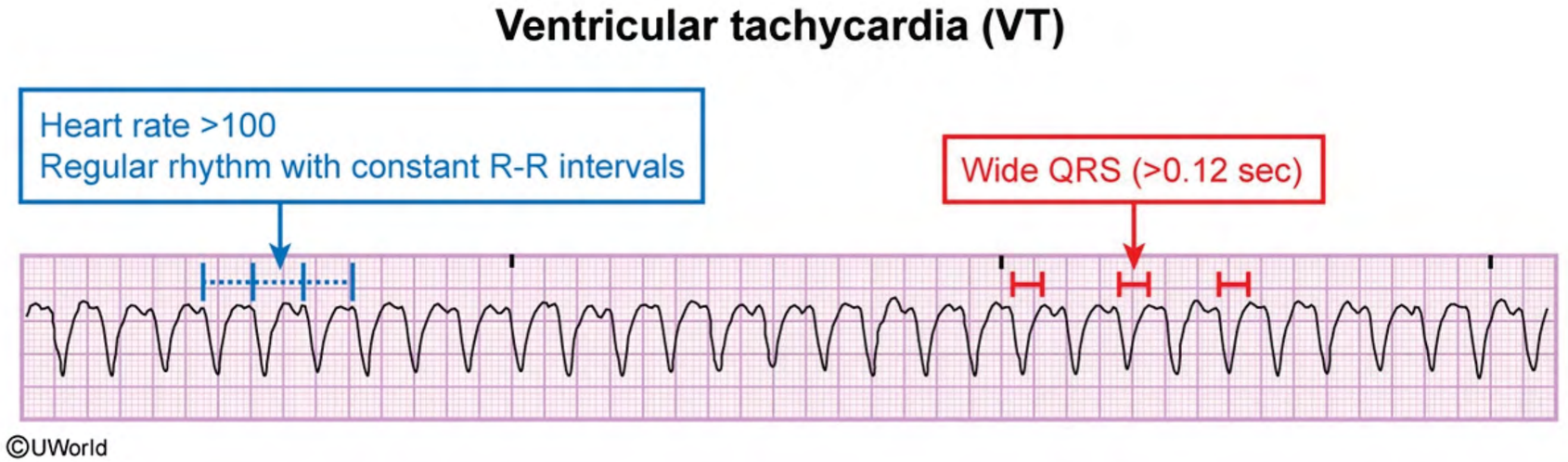

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A 64-year-old man with a long-standing history of hypertension is brought to the emergency department due to a dry cough and shortness of breath. The patient has been unable to sleep in a flat position for the past 2 days. Blood pressure is 192/102 mm Hg and pulse is 92/min and regular. Physical examination reveals an S4 and bibasilar crackles. He is started on a high-dose intravenous nitroglycerin infusion, and soon after he reports significant symptomatic relief. Which of the following physiologic changes most likely occurred in this patient as a result of the infusion?

	Left ventricular end-diastolic pressure	Peripheral venous capacitance	Systemic vascular resistance
○ A.	No effect	↑	↓
○ B.	↑	↓	No effect
○ C.	↓	↑	↓
○ D.	↓	↓	↓
○ E.	↑	No effect	↓
○ F.	↑	↑	↑

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	Left ventricular end-diastolic pressure	Peripheral venous capacitance	Systemic vascular resistance
✗ A.	No effect	↑	↓
B.	↑	↓	No effect
✓ C.	↓	↑	↓
D.	↓	↓	↓
E.	↑	No effect	↓
F.	↑	↑	↑

Incorrect
Correct answer
C

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Explanation

This patient's clinical presentation is consistent with **acute pulmonary edema** due to severely elevated blood pressure (**hypertensive emergency**). Intravenous vasodilators (eg, nitroglycerin, sodium nitroprusside) are often used in the management of acute heart failure due to severe hypertension.

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F. ↑ ↑ ↑

Incorrect
Correct answer
C

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Explanation

This patient's clinical presentation is consistent with **acute pulmonary edema** due to severely elevated blood pressure (**hypertensive emergency**). Intravenous vasodilators (eg, nitroglycerin, sodium nitroprusside) are often used in the management of acute heart failure due to severe hypertension.

Nitrates (eg, nitroglycerin, isosorbide dinitrate, isosorbide mononitrate) are primarily **venodilators** that increase peripheral venous capacitance, thereby reducing cardiac preload and left ventricular end-diastolic pressure (LVEDP) and volume. This is the primary mechanism of action for symptom relief in patients with acute pulmonary edema. **Lower LVEDP** also leads to a reduction in LV systolic wall stress and a decrease in myocardial oxygen demand, resulting in relief of angina symptoms.

At **high doses**, nitrates also cause some degree of **arteriolar dilation** (in addition to venodilation), which decreases systemic vascular resistance and may sometimes cause a precipitous decline in blood pressure. Sodium nitroprusside has slightly different effects than nitrates, acting as a balanced vasodilator of venules and arterioles at all dose levels.

Educational objective:
Nitrates are primarily venodilators and increase peripheral venous capacitance, thereby reducing cardiac preload and left ventricular end-diastolic volume and pressure. At high doses, nitrates also cause some degree of arteriolar dilation with a resulting decrease in systemic vascular resistance.

Pharmacology	Cardiovascular System	Nitrates
Subject	System	Topic

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A 53-year-old woman comes to the office due to progressive distension and tightness in her right lower extremity for the past year. She has had no shortness of breath, fever, or skin rash. The patient was diagnosed with melanoma of the right thigh approximately 2 years ago and underwent surgical resection. Two of her inguinal lymph nodes tested positive for metastasis, and she subsequently underwent inguinal lymphadenectomy and received adjuvant therapy. Recent imaging revealed no evidence of recurrent malignancy. The patient's medical history also includes hypertension and hyperlipidemia. Vital signs are within normal limits. Physical examination shows clear lungs and normal heart sounds without murmurs or gallops. No jugular venous distension is present. The right lower extremity is significantly larger in diameter than the left. Edema is present up to the thigh, with mild skin indentation after application of pressure. The skin overlying the involved area is thickened and dry. Laboratory testing, including complete blood count, serum metabolic panel, and urinalysis, is within normal limits. Treatment of this patient's symptoms should include which of the following strategies?

- A. Blockade of estrogen receptors
- B. Compression to increase lymphatic flow
- C. Dissolution of intravascular thrombus
- D. Inhibition of vascular endothelial growth factor
- E. Reduction of intravascular volume

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- ✗ A. Blockade of estrogen receptors (0%)
- ✓ B. Compression to increase lymphatic flow (73%)
- C. Dissolution of intravascular thrombus (11%)
- D. Inhibition of vascular endothelial growth factor (5%)
- E. Reduction of intravascular volume (7%)

Incorrect
Correct answer
B

73%
Answered correctly

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Explanation

Chronic lymphedema	
	• Physical disruption of lymphatic drainage ◦ Lymphadenectomy, radiation

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Explanation

Chronic lymphedema	
Etiology	• Physical disruption of lymphatic drainage ◦ Lymphadenectomy, radiation ◦ Malignant obstruction ◦ Chronic inflammation (eg, recurrent cellulitis) ◦ Parasitic infection (eg, filariasis) ◦ Congenital (eg, Turner syndrome)
Clinical presentation	• Swelling, heaviness, discomfort • Early: soft skin, pitting edema • Late: firm, dry & thickened skin; nonpitting edema
Treatment	• Weight loss, limb elevation • Compression bandages & physiotherapy • Diuretics contraindicated

This patient with chronic swelling and thickened skin of the right leg most likely has **chronic lymphedema**. Lymphedema can be congenital, but it most commonly results from an acquired **disruption of lymphatic drainage** that allows for accumulation of lymphatic fluid in the interstitial tissue. Malignancy and its treatment (eg, radiation, **lymphadenectomy**) are frequent underlying causes; other causes include chronic inflammation (eg, recurrent cellulitis, connective tissue disease) and parasitic infection (ie, filariasis due to *Wuchereria bancrofti*). Obesity is often a strong contributing factor.

Lymphedema usually presents with swelling, heaviness, and discomfort in one or more extremities. In early disease, the edema is pitting and the skin remains soft. However, over time there is progressive **deposition of subcutaneous collagen** and adipose tissue, which leads to firm, dry, and **thickened skin** and **nonpitting edema**. Treatment is difficult and typically involves conservative management to increase lymphatic drainage via **compression bandages** or physiotherapy (ie, manual lymphatic drainage).

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Lymphedema usually presents with swelling, heaviness, and discomfort in one or more extremities. In early disease, the edema is pitting and the skin remains soft. However, over time there is progressive **deposition of subcutaneous collagen** and adipose tissue, which leads to firm, dry, and **thickened skin** and **nonpitting edema**. Treatment is difficult and typically involves conservative management to increase lymphatic drainage via **compression bandages** or physiotherapy (ie, manual lymphatic drainage).

(Choice A) Selective estrogen receptor modulators (eg, tamoxifen) block estrogen receptors in breast tissue and can be useful in treating breast cancer. Breast cancer may present with upper extremity lymphedema, but lower extremity lymphedema would be extremely unusual.

(Choice C) Anticoagulants (eg, heparin) facilitate dissolution of acute deep venous thrombosis (DVT); however, acute DVT is less likely without active malignancy and would not explain this patient's year-long duration of symptoms.

(Choice D) Inhibition of vascular endothelial growth factor is useful in the treatment of lymphangiosarcoma, which is a rare complication of lymphedema. Lymphangiosarcoma typically presents as purple maculopapular skin lesions, which are not present in this patient.

(Choice E) Diuretics reduce intravascular volume and are useful in treating lower extremity edema that results from elevated venous hydrostatic pressure in true volume overloaded states (eg, heart failure, cirrhosis). However, they are ineffective and contraindicated for lymphedema because lymphedematous fluid cannot be easily mobilized from the interstitial space into the vasculature. When used for lymphedema, diuretics are likely to cause intravascular volume depletion and consequent acute kidney injury.

Educational objective:
Chronic lymphedema is most commonly caused by an acquired disruption of lymphatic drainage (eg, due to malignancy or lymphadenectomy), and typically presents with swelling and thickened skin in one or more extremities. Treatment is usually conservative and involves compression bandages and physiotherapy; diuretics are ineffective and contraindicated.

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A 67-year-old man admitted for right lower lobe pneumonia subsequently develops hypotension and lactic acidosis. He is started on a norepinephrine intravenous drip. A few hours later, the antecubital vein being used for the infusion blanches and the tissues surrounding the IV site become cold, hard, and pale. Local injection of the affected tissues with which of the following agents is most likely to be of greatest benefit?

- A. Calcium gluconate
- B. Phentolamine
- C. Heparin
- D. Isoproterenol
- E. Lidocaine

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- A. Calcium gluconate (9%)
- ✓ B. Phentolamine (49%)
- C. Heparin (15%)
- D. Isoproterenol (14%)
- E. Lidocaine (9%)

Correct | 49% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Development of **venous blanching** along with **induration** and **pallor** of the tissues surrounding the norepinephrine infusion site are signs of **norepinephrine extravasation**. The norepinephrine leak causes intense **α_1 receptor** mediated vasoconstriction which can lead to local **tissue necrosis**. Such necrosis can be prevented by infiltration (using a syringe with a fine hypodermic needle) throughout the affected area with **phentolamine**, an α receptor blocker leading to vasodilatation (thus counteracting the vasoconstrictive effects of norepinephrine). This antidote must be given within 12 hours of extravasation to be effective.

(Choice A) Calcium gluconate is administered for severe hypocalcemia and for severe hyperkalemia (acts as a cardioprotective agent that prevents arrhythmias); it would not lead to the desired effect of vasodilation.

(Choice C) Heparin is indicated if there is an arterial thrombosis (cold, pulseless, painful limb), but this patient does not have any risk factors for arterial thrombosis (eg, atrial fibrillation, arterial catheterization) and

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(Choice A) Calcium gluconate is administered for severe hypocalcemia and for severe hyperkalemia (acts as a cardioprotective agent that prevents arrhythmias); it would not lead to the desired effect of vasodilation.

(Choice C) Heparin is indicated if there is an arterial thrombosis (cold, pulseless, painful limb), but this patient does not have any risk factors for arterial thrombosis (eg, atrial fibrillation, arterial catheterization) and norepinephrine extravasation is a much more likely cause of his limb symptoms.

(Choice D) Isoproterenol can mediate vasodilation via β_2 receptor activation, particularly in striated muscle, renal, and mesenteric vascular beds, leading to decreased peripheral vascular resistance with increased cardiac output. Subcutaneous tissue blood vessels have a relatively low β_2 receptor density compared to α1 receptors, thus, an alpha blocker would likely be more effective in reversing α_1 receptor mediated vasoconstriction.

(Choice E) Lidocaine can relax vascular smooth muscle via blockade of Na^+ current mediated action potentials. However, in this case, extravasation of norepinephrine is responsible for the vasoconstriction; nerve blockade would not reverse the ischemia.

Educational objective:
Blanching of a vein into which norepinephrine is being infused together with induration and pallor of the tissues surrounding the IV site are signs of norepinephrine extravasation and resulting vasoconstriction. Tissue necrosis is best prevented by local injection of an α receptor blocking drug, such as phentolamine.

Pharmacology	Cardiovascular System	Sympathomimetic agents
Subject	System	Topic

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A 73-year-old man is evaluated for exertional dyspnea. His exercise tolerance has decreased over the last year, and he can barely walk 2-3 blocks without stopping. The patient also reports occasional episodes of lightheadedness and palpitations. He has had no chest pain or syncope. He was diagnosed with hypertension in the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?

- A. A
- B. B
- C. C
- D. D
- E. E

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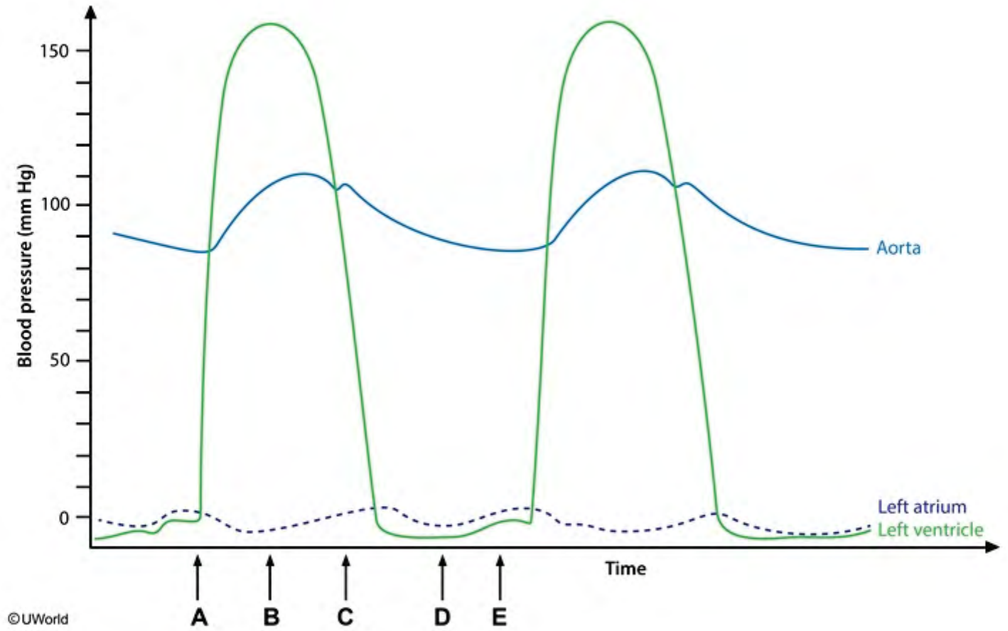

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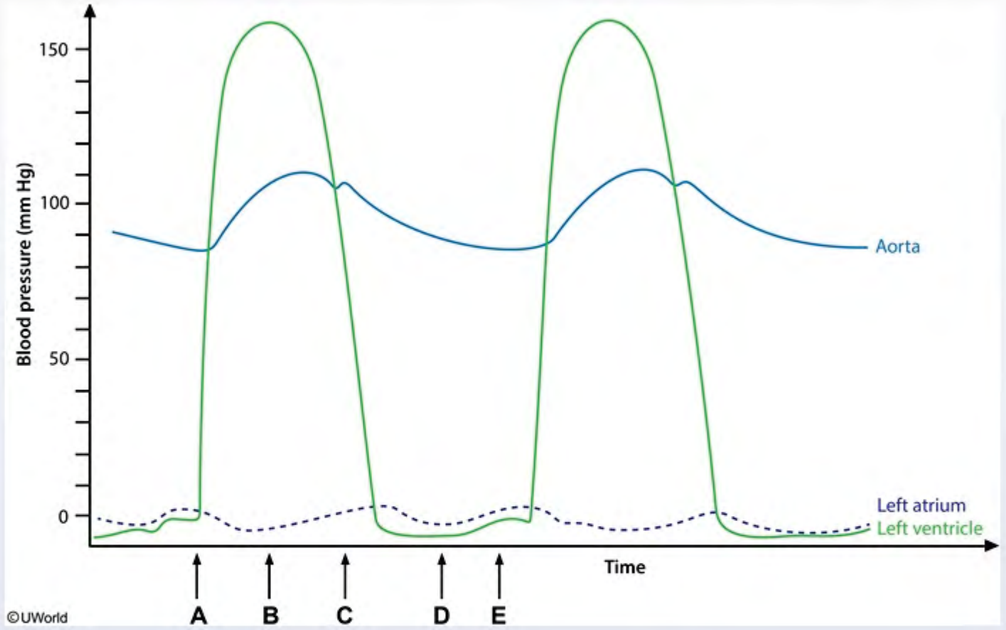

- A. A
- B. B
- C. C
- D. D
- E. E

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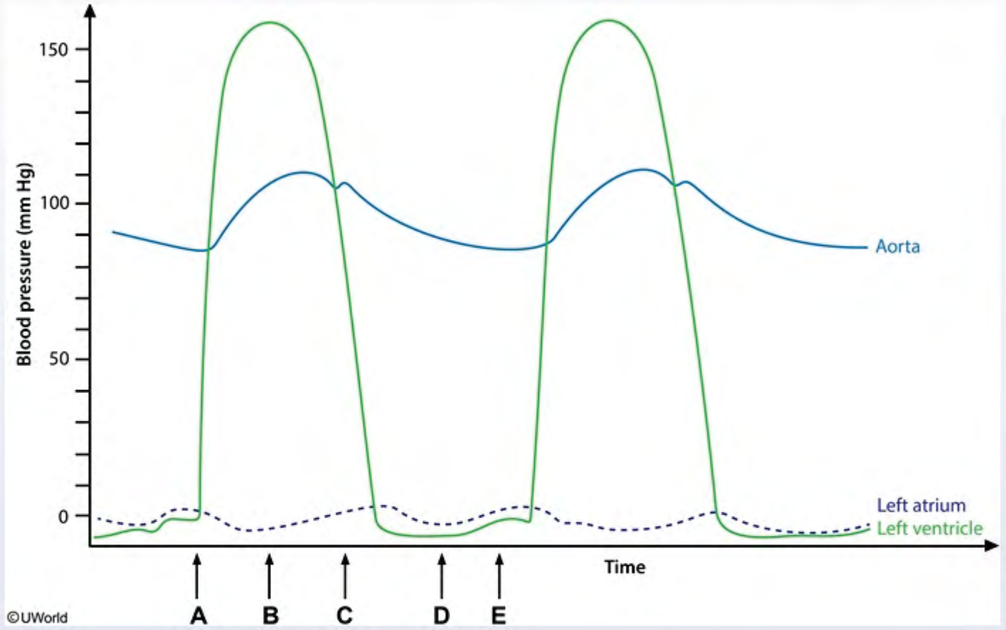
the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?



- A. A (15%)
- ✓ B. B (57%)
- C. C (17%)
- D. D (5%)
- E. E (3%)

Correct

57% Answered correctly

12 secs Time Spent

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Explanation

In the absence of any aortic valve pathology, the pressure in the aorta is similar to left ventricular pressure during systole. However, this patient's hemodynamic profile shows an abnormal pressure gradient between the left ventricular and aortic pressure tracings during systole (compare to normal), indicating significant aortic stenosis (AS). The intensity of the murmur of AS is directly related to the magnitude of the left ventricle to aorta pressure gradient. The maximum pressure difference between the left ventricular and aortic pressure tracings is noted at point B and corresponds to the peak intensity of a cardiac murmur during auscultation.

(Choices A and C) The murmur of AS is a systolic ejection-type crescendo-decrescendo murmur that starts after the first heart sound following the opening of the aortic valve (point A). It typically ends before the A2 component of the second heart sound (point C). At points A and C, the left ventricular and aortic pressures are nearly equal with no or minimal flow across the aortic valve, and the murmur of AS would not be appreciated.

(Choices D and E) These points occur during diastole, when the aortic valve is closed and the mitral valve is open. There is no abnormally elevated pressure gradient between the left atrium and left ventricle. This is consistent with normal, unobstructed diastolic filling of the left ventricle, and no murmur would be heard during diastole.

Educational objective:
The murmur of aortic stenosis (AS) is a systolic ejection-type, crescendo-decrescendo murmur that starts after the first heart sound and typically ends before the A2 component of the second heart sound. The intensity of the murmur is proportional to the magnitude of the left ventricle to aorta pressure gradient during systole.

References

- The patient with a systolic murmur: severe aortic stenosis may be missed during cardiovascular examination.

Pathophysiology	Cardiovascular System	Aortic stenosis
Subject	System	Topic

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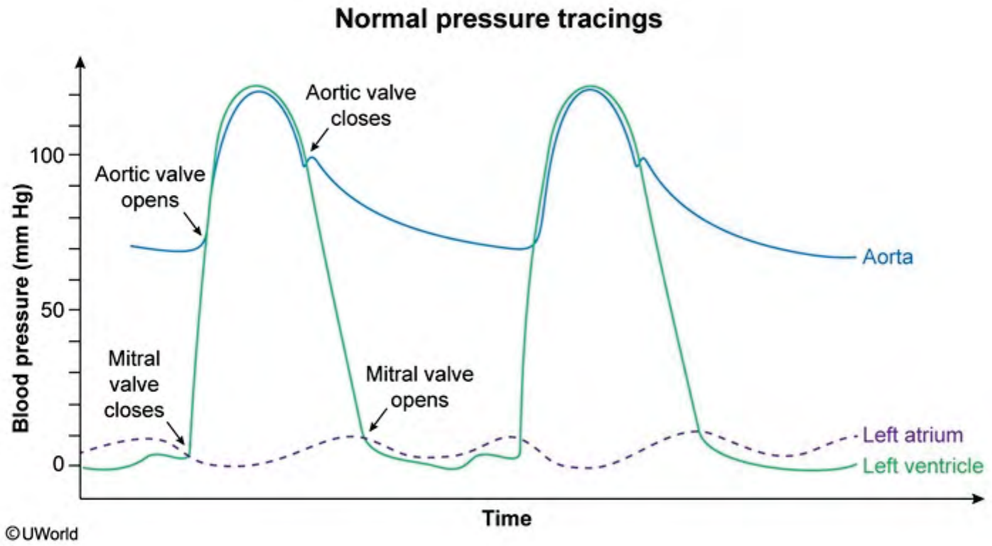

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https://t.me/USMLEWorldStep1

A 22-year-old woman comes to the office due to worsening dyspnea. The patient has also experienced low-grade fevers, a 6.8-kg (15-lb) weight loss, and syncopal episodes over the last 3 months. Her shortness of breath worsens when sitting and improves when lying down. Medical history is insignificant. The patient does not use tobacco, alcohol, or illicit drugs. Temperature is 37.4 C (99.3 F), blood pressure is 115/72 mm Hg, pulse is 75/min and regular, and respirations are 12/min. The lungs are clear. Cardiac auscultation reveals a low-pitched, mid-diastolic rumble at the cardiac apex. The remainder of the physical examination is normal. ECG reveals left atrial enlargement, and echocardiography shows a large, pedunculated mass in the left atrium. Histologic analysis of this mass will most likely reveal which of the following?

- A. Coarse, filamented, branching septate hyphae
- B. Gram-positive cocci in clusters
- C. Malignant myeloid cells
- D. Scattered cells within a mucopolysaccharide stroma
- E. Solid mass of platelets and fibrin

Submit

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- A. Coarse, filamented, branching septate hyphae (1%)
- ✕ B. Gram-positive cocci in clusters (5%)
- C. Malignant myeloid cells (8%)
- ✓ D. Scattered cells within a mucopolysaccharide stroma (73%)
- E. Solid mass of platelets and fibrin (10%)

Incorrect
Correct answer
D

73%
Answered correctly

03 secs
Time Spent

2023
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Explanation

Cardiac myxoma

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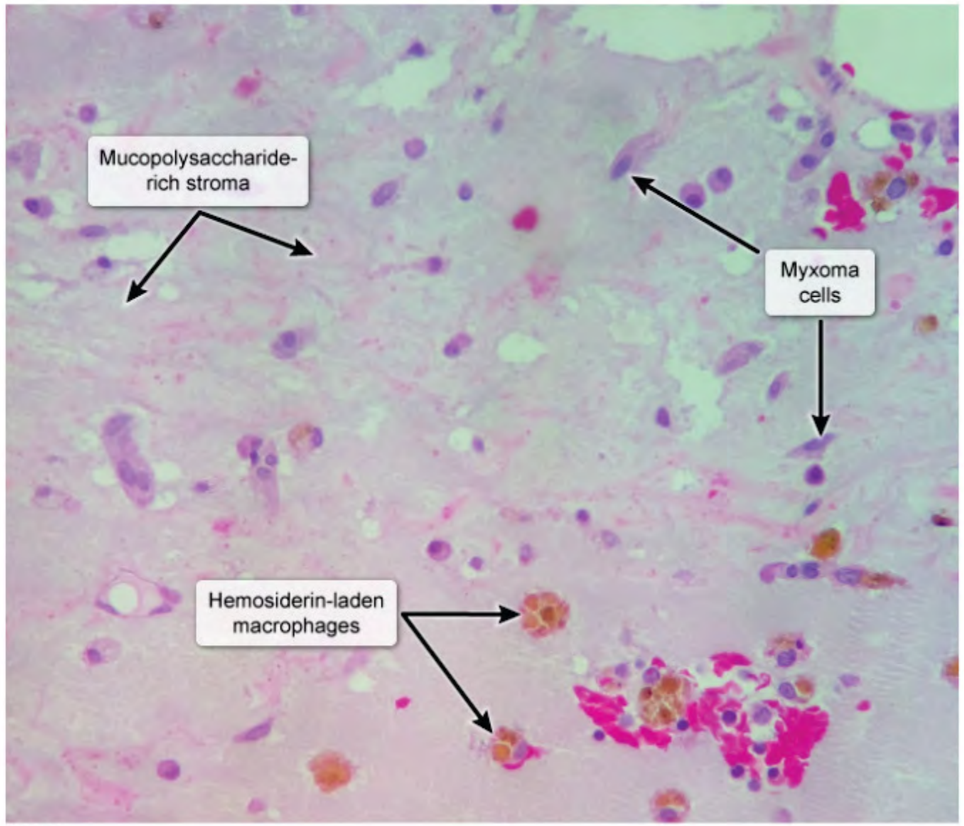
Cardiac myxoma



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Mucopolysaccharide-rich stroma

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This patient with a large, pedunculated mass in the left atrium most likely has an **atrial myxoma**. Myxomas are the most common primary cardiac neoplasm, and approximately 80% arise within the **left atrium**.

Left atrial myxomas frequently obstruct blood flow from the left atrium to the left ventricle, leading to a **murmur** mimicking that of mitral stenosis (ie, mid-diastolic rumble at the apex). The obstruction can also lead to a decrease in cardiac output that manifests as dyspnea, lightheadedness, or syncope. Because the mass is typically mobile, **obstructive symptoms** may be transient and **influenced by position** (ie, upright posture exacerbates mitral obstruction, whereas lying down alleviates it). In some patients, fragments of the mass may embolize into the systemic circulation (eg, resulting in stroke or acute limb ischemia). In addition, some myxomas can produce cytokines (eg, interleukin-6) that lead to **constitutional symptoms** including fever and weight loss.

Histologically, these tumors demonstrate scattered myxoma cells within a **mucopolysaccharide stroma** and blood vessels, which may be encircled by myxoma cells. Myxomas produce a large amount of vascular endothelial growth factor, which contributes to the angiogenesis, hemorrhaging (seen as brown hemosiderin deposits), and friability characterizing these tumors.

(Choices A and B) Coarse, filamented, branching septate hyphae are found in fungal endocarditis, whereas gram-positive cocci in clusters are consistent with bacterial endocarditis. Both are unlikely in this young woman with no history of valvular disease or intravenous drug use. In addition, endocarditis typically causes a regurgitant murmur, with valvular vegetations seen on echocardiography.

(Choice C) Acute myeloid leukemia (AML) rarely may involve the heart. Signs and symptoms related to anemia (eg, weakness, fatigue), neutropenia (eg, infections), and thrombocytopenia (eg, ecchymoses, epistaxis), are typically present with AML.

(Choice E) Nonbacterial thrombotic endocarditis involves the valvular deposition of sterile platelets and fibrin and most commonly occurs in the setting of advanced malignancy or systemic lupus erythematosus. The disease usually presents with systemic embolization, and heart murmurs are often absent; if a murmur is present, it is typically regurgitant rather than stenotic.

Educational objective:
Myxomas are the most common primary cardiac neoplasm and usually arise within the left atrium. The tumors

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obstructive symptoms may be transient and **influenced by position** (ie, upright posture exacerbates mitral obstruction, whereas lying down alleviates it). In some patients, fragments of the mass may embolize into the systemic circulation (eg, resulting in stroke or acute limb ischemia). In addition, some myxomas can produce cytokines (eg, interleukin-6) that lead to **constitutional symptoms** including fever and weight loss.

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Educational objective:
Myxomas are the most common primary cardiac neoplasm and usually arise within the left atrium. The tumors typically cause position-dependent obstruction of the mitral valve, leading to a mid-diastolic murmur and symptoms of decreased cardiac output (eg, dyspnea, syncope). Constitutional symptoms (eg, fever, weight loss) may also be present. Histologically, the tumors demonstrate scattered cells within a mucopolysaccharide stroma and blood vessels with hemorrhage.

Pathology	Cardiovascular System	Atrial myxoma
Subject	System	Topic

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 23-year-old woman comes to the office due to exertional dyspnea. She occasionally has a dry cough but has had no chest pain, palpitations, or syncope. The patient emigrated from Cambodia 3 years ago. She had bilateral knee swelling as a child. Cardiac auscultation findings over the cardiac apex are given below. Which of the following is the most likely cause of this patient's symptoms?

Play Media

- A. Ascending aortic aneurysm
- B. Atrial septal defect
- C. Bicuspid aortic valve
- D. Mitral regurgitation
- E. Mitral stenosis

Submit

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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

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Play Media

- A. Ascending aortic aneurysm (0%)
- B. Atrial septal defect (3%)
- C. Bicuspid aortic valve (5%)
- ✓ D. Mitral regurgitation (61%)
- E. Mitral stenosis (28%)

Correct

61% Answered correctly

55 secs Time Spent

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Explanation

Cardiac murmurs

	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed

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A. Ascending aortic aneurysm (0%)

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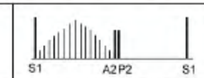
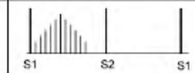
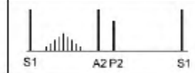
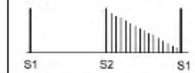
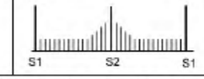
Cardiac murmurs

Systolic	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid-left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation*		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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This patient's auscultatory findings reveal a blowing holosystolic murmur best heard over the cardiac apex (typically with radiation to the axilla), which is classic for **mitral regurgitation** (MR). Given her emigration from Southeast Asia and knee swelling as a child, the MR is most likely due to **rheumatic heart disease** (rheumatic heart disease is most commonly seen in Latin America, Africa, and Asia).

Patients with rheumatic heart disease almost always have **mitral valve involvement**; MR is the more common manifestation in young patients (eg, age <25), whereas mitral stenosis is more common in middle-aged patients (eg, age >30). With increasing age, mixed mitral valve disease (ie, components of both regurgitation and stenosis) becomes increasingly frequent.

(Choice A) Patients with an ascending aortic aneurysm may develop aortic regurgitation secondary to aortic root dilation; an early decrescendo diastolic murmur is expected, best heard at the right upper sternal border.

(Choice B) The typical murmur of an atrial septal defect is not caused by blood flow through the defect itself but results from an elevation in right-sided blood volume with increased blood flow across the pulmonic valve (ie, pulmonic flow murmur). It is recognized as a faint midsystolic murmur best heard at the left upper sternal border and with fixed splitting of S2.

(Choice C) A bicuspid aortic valve is commonly associated with aortic stenosis, which classically causes a crescendo-decrescendo systolic murmur best heard at the right upper sternal border and with radiation to the carotids. Aortic stenosis due to a bicuspid aortic valve typically affects patients age >40.

(Choice E) Rheumatic heart disease is the most common cause of mitral stenosis. With mitral stenosis, auscultation at the cardiac apex is expected to reveal an opening snap followed by a low-pitch, middiastolic, rumbling murmur. The murmur may accentuate in late diastole due to atrial contraction against the stenotic valve.

Educational objective:
Mitral regurgitation causes a blowing holosystolic murmur best heard at the cardiac apex and with radiation to the axilla. Rheumatic heart disease is a common cause of both mitral regurgitation and mitral stenosis and is most commonly seen in patients who grew up in Latin America, Africa, or Asia.

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This patient's auscultatory findings reveal a blowing holosystolic murmur best heard over the cardiac apex

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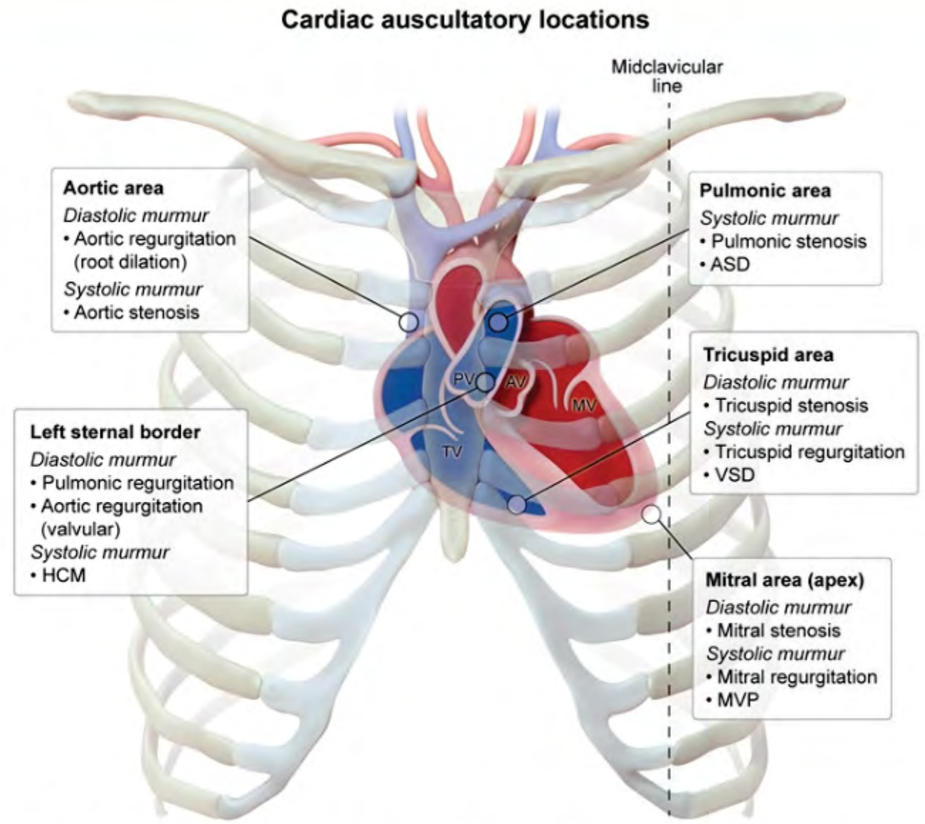

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A multinational research institute conducting experiments on human circulatory physiology enrolls a healthy 30-year-old male volunteer to assess the oxygen consumption rate of various organs. During the study, the blood oxygen content of the aorta and several other vessels is measured at rest. The greatest difference in these measurements will most likely be between the aorta and which of the following blood vessels?

- A. Brachial vein
- B. Coronary sinus
- C. Internal jugular vein
- D. Portal vein
- E. Pulmonary artery
- F. Renal vein

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- A. Brachial vein (1%)
- ✓ B. Coronary sinus (29%)
- C. Internal jugular vein (8%)
- D. Portal vein (4%)
- E. Pulmonary artery (52%)
- F. Renal vein (3%)

Correct | 29% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

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A multinational research institute conducting experiments on human circulatory physiology enrolls a healthy 30 year-o oxyge measu

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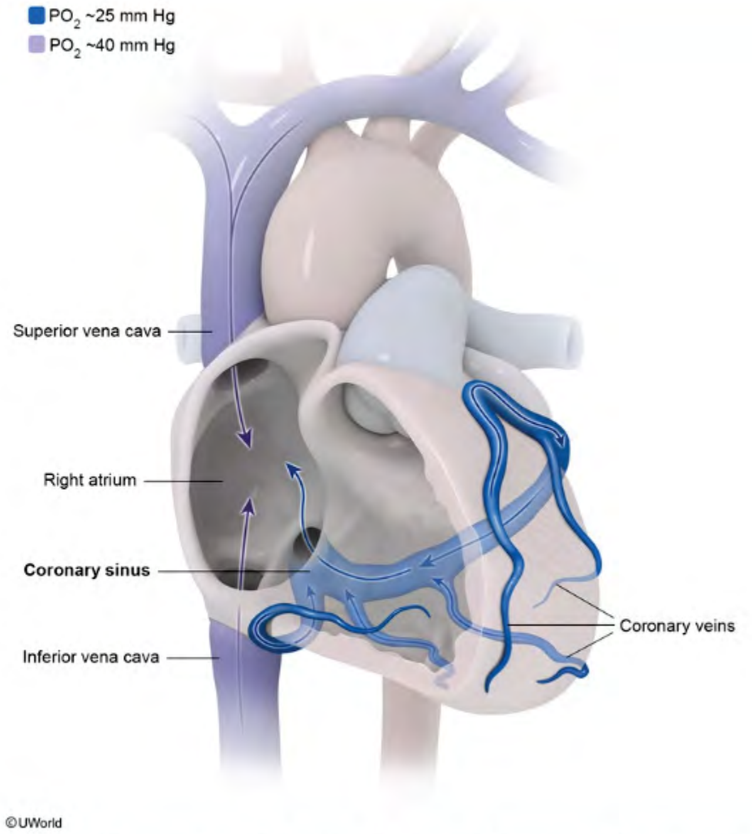

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Arterial blood supply to the cardiac muscle (myocardium) is provided by the right and left coronary arteries arising directly from the aortic root. Most cardiac venous blood drains into the right atrium via the **coronary sinus**, with the remainder draining directly into the other chambers of the heart. Three specific features distinguish cardiac circulation from blood flow to skeletal muscle and viscera:

1. **The left ventricle is perfused only during diastole.** Myocardial contraction during systole leads to compression of the coronary vessels and disruption of blood flow. Wall tension is highest near the endocardium, making the subendocardial region the most prone to ischemia.

2. **Myocardial oxygen extraction is very high.** The heart has a capillary density far exceeding that of skeletal muscle. Oxygen extraction from arterial blood is very effective within the heart as the resting myocardium extracts 60%-75% of oxygen from blood. This amount is higher than that extracted by any other tissue or organ in the body. As a result, the cardiac venous blood in the coronary sinus, before it reaches the right atrium and mixes with blood returning from the systemic circulation, is the most deoxygenated blood in the body **(Choices A, C, D, and F)**.

3. **Myocardial oxygen demand and coronary blood flow are tightly coupled.** Because oxygen extraction by the resting heart is already very high, there is little capacity to increase myocardial oxygen extraction during periods of increased oxygen demand (eg, during exercise). Therefore, increased oxygen delivery to the heart can be achieved only through increased **coronary blood flow**. Adenosine and nitric oxide are the most important vasodilators responsible for increasing coronary flow.

(Choice E) Deoxygenated systemic blood returning via the vena cava contains more oxygen than coronary sinus venous blood. Because the pulmonary artery contains an admixture of blood returning via coronary sinus and systemic venous circulation, its oxygen content is also higher than pure coronary venous return.

Educational objective:
Myocardial oxygen extraction exceeds that of any other tissue or organ; therefore, the cardiac venous blood in the coronary sinus is the most deoxygenated blood in the body. Due to the high degree of oxygen extraction, increases in myocardial oxygen demand can only be met by an increase in coronary blood flow.

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An 8-day-old boy is being evaluated in the neonatal intensive care unit. The patient was born at 27 weeks gestation and intubated at birth due to surfactant deficiency. Temperature is 37 C (98.6 F), blood pressure is 66/12 mm Hg, and pulse is 182/min. A continuous harsh murmur is heard at the left infraclavicular region. There are bounding femoral and palmar pulses. A drug with which of the following mechanisms of action is most appropriate for treating the cause of this patient's murmur?

- A. Activation of phospholipase A_2
- B. Activation of prostacyclin receptors
- C. Activation of prostaglandin E_2 receptors
- D. Inhibition of cyclooxygenase
- E. Inhibition of lipoxygenase
- F. Inhibition of prostaglandin D_2 receptors

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- A. Activation of phospholipase A_2 (2%)
- ✗ B. Activation of prostacyclin receptors (2%)
- C. Activation of prostaglandin E_2 receptors (33%)
- ✓ D. Inhibition of cyclooxygenase (46%)
- E. Inhibition of lipoxygenase (1%)
- F. Inhibition of prostaglandin D_2 receptors (13%)

Incorrect
Correct answer
D

46%
Answered correctly

03 secs
Time Spent

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Explanation

This newborn has a continuous murmur, widened pulse pressure, and bounding pulses, findings characteristic of a **patent ductus arteriosus** (PDA). After birth, **closure** of the ductus arteriosus normally occurs within 24-48 hours and is **initiated by loss of** placental prostaglandin E_2 (**PGE_2**) and the vasoconstrictive effects of increased systemic oxygen levels.

Persistence of the ductus arteriosus (ie, a PDA) most commonly occurs in **premature infants** and may be associated with elevated postnatal PGE_2 or increased sensitivity to prostaglandins. Left-to-right shunting from the

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Persistence of the ductus arteriosus (ie, a PDA) most commonly occurs in **premature infants** and may be associated with elevated postnatal PGE_2 or increased sensitivity to prostaglandins. Left-to-right shunting from the aorta to the pulmonary artery takes place, causing a continuous machinery-like murmur. Patients with a large PDA can develop decreased diastolic blood pressure (due to reduced systemic blood flow) and pulmonary overcirculation with left ventricular volume overload, leading to a compensatory increase in stroke volume that causes bounding pulses and widened pulse pressure.

Treatment of PDA includes nonsteroidal anti-inflammatory drugs (**NSAIDs**) (eg, indomethacin, ibuprofen), which inhibit cyclooxygenase. Because cyclooxygenase normally synthesizes prostaglandins from arachidonic acid, NSAIDs **inhibit PGE_2 synthesis**, thereby inducing ductal smooth muscle contraction and promoting PDA closure.

(Choice A) Phospholipase A_2 synthesizes arachidonic acid from phospholipids; therefore, phospholipase A_2 activation would increase arachidonic acid metabolite production, including PGE_2, and could encourage patency of the ductus arteriosus.

(Choices B and C) Activation of prostacyclin (prostaglandin I_2) and PGE_2 receptors promotes vasodilation and patency, not closure, of the ductus arteriosus. This mechanism is used in treating ductal-dependent congenital heart defects (eg, transposition of the great arteries) in which maintaining ductal patency is required for adequate systemic oxygenation.

(Choices E and F) Lipoxygenase converts arachidonic acid to leukotrienes, which are potent bronchoconstrictors. Similarly, the primary effect of prostaglandin D_2 (PGD_2), which is released by mast cells, is bronchoconstriction. Inhibitors of either lipoxygenase (eg, zileuton) or PGD_2 may be used as bronchodilators in asthma treatment, but they are not useful in facilitating PDA closure.

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(Choices E and F) Lipoxygenase converts arachidonic acid to leukotrienes, which are potent bronchoconstrictors. Similarly, the primary effect of prostaglandin D_2 (PGD_2), which is released by mast cells, is bronchoconstriction. Inhibitors of either lipoxygenase (eg, zileuton) or PGD_2 may be used as bronchodilators in asthma treatment, but they are not useful in facilitating PDA closure.

Educational objective:
A patent ductus arteriosus (PDA) classically presents with a continuous murmur, widened pulse pressure, and bounding pulses. Because physiologic closure of the ductus arteriosus is normally initiated by loss of placental prostaglandin E_2 (PGE_2), treatment of a symptomatic PDA includes cyclooxygenase inhibitors (eg, indomethacin), which decrease PGE_2 production.

References

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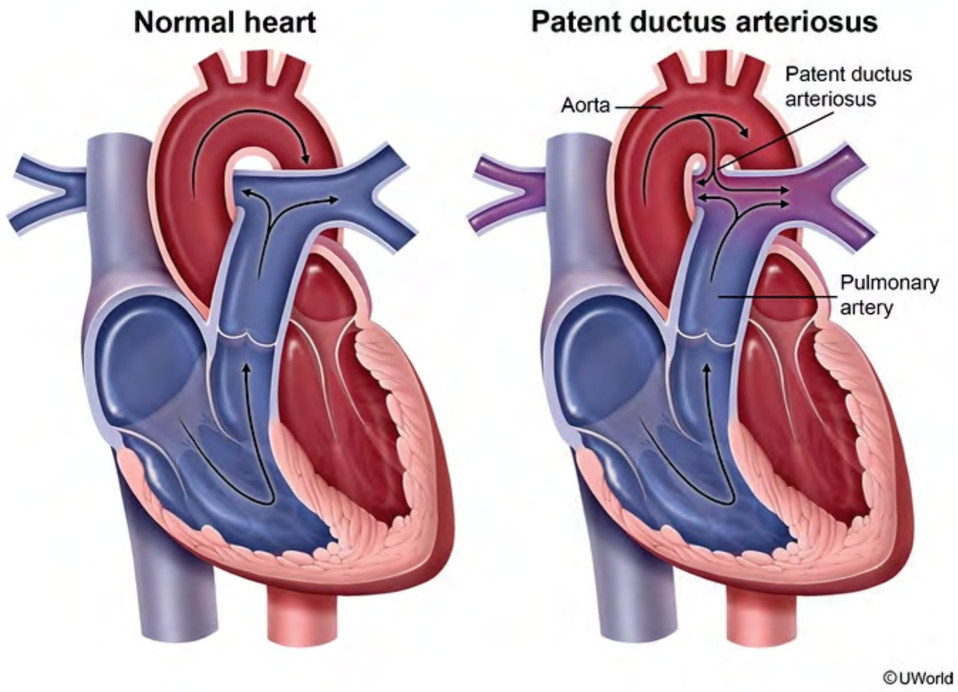

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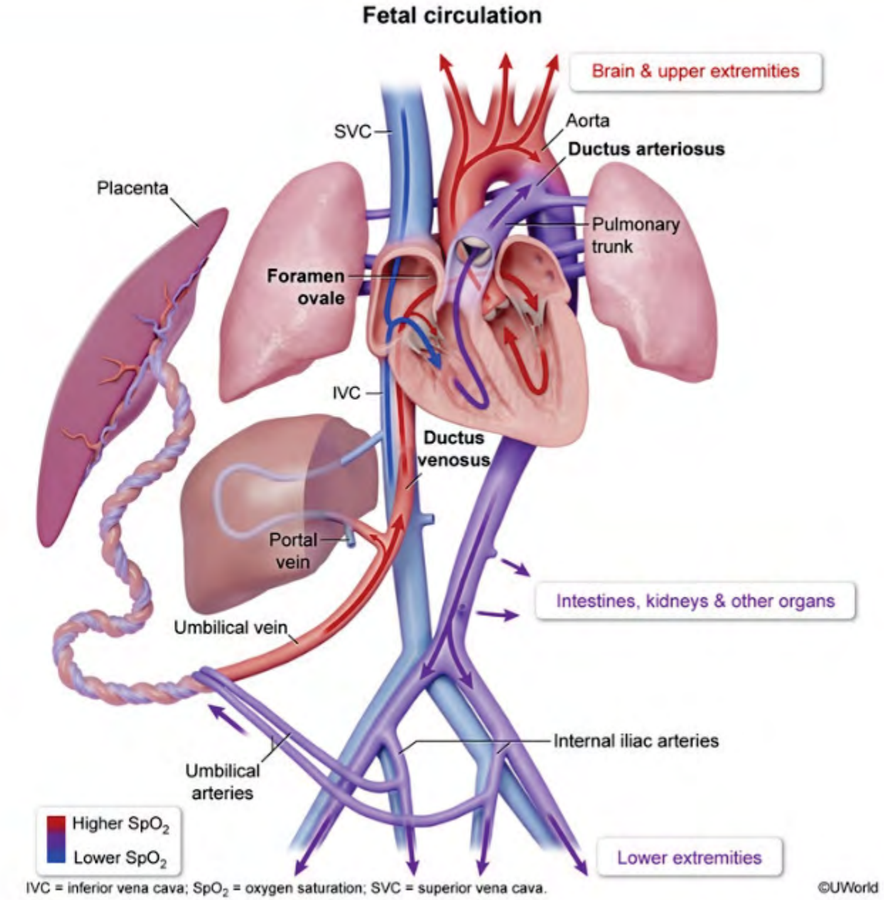

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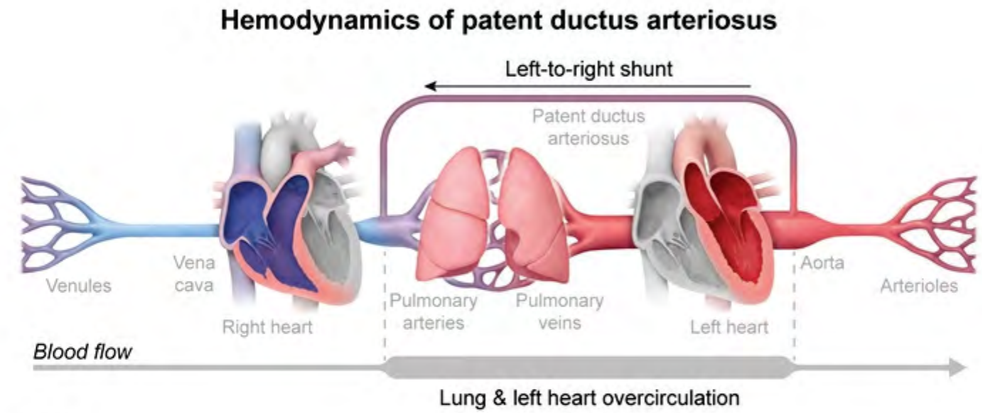

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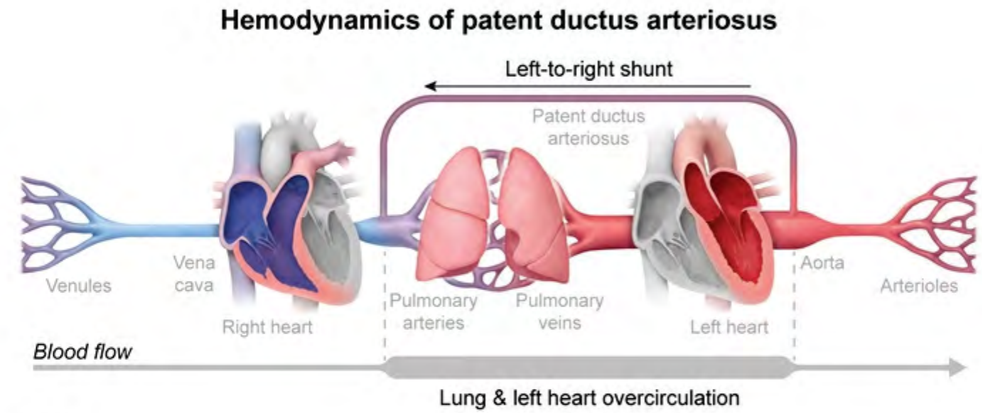

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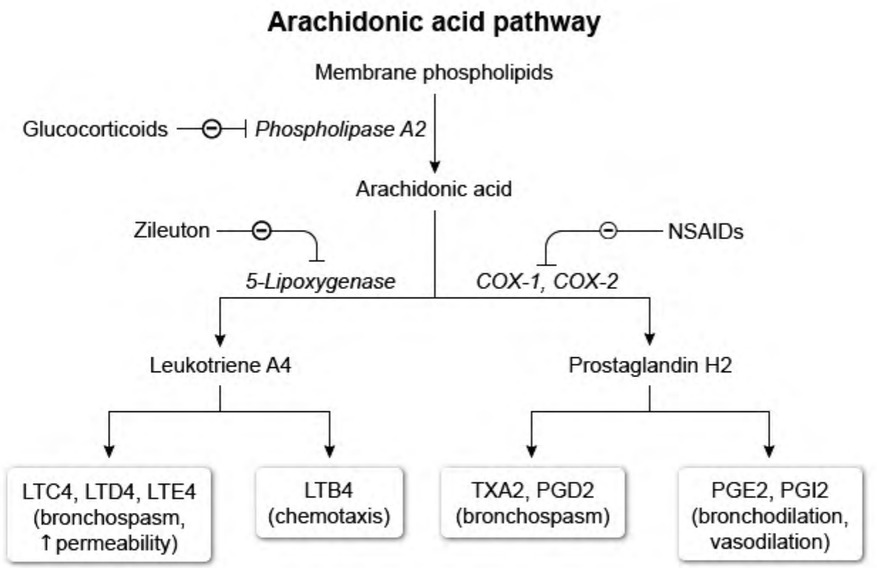

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A 54-year-old man comes to the emergency department due to severe fatigue and dyspnea. He has a long history of progressively worsening heart failure that has been resistant to treatment with medications, including diuretics. He was treated with chest radiation 10 years ago for non-Hodgkin lymphoma and has been in remission since then. The patient is admitted to the hospital, but his condition continues to deteriorate despite aggressive therapy. He dies 3 days later, and an autopsy is performed. Gross inspection of the heart shows dense, thick, fibrous tissue in the pericardial space between the visceral and parietal pericardium. Which of the following signs would most likely have been detected during a physical examination of this patient just prior to his death?

- A. Kussmaul sign
- B. Loud P2
- C. Paradoxical splitting of S2
- D. S3 gallop
- E. Sustained left parasternal lift

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- ✓ A. Kussmaul sign (64%)
- ✗ B. Loud P2 (3%)
- C. Paradoxical splitting of S2 (11%)
- D. S3 gallop (13%)
- E. Sustained left parasternal lift (6%)

Incorrect
Correct answer
A

64%
Answered correctly

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Explanation

Constrictive pericarditis	
Etiology	• Idiopathic or viral pericarditis • Cardiac surgery or radiation therapy • Tuberculosis (in endemic areas)

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Constrictive pericarditis	
Etiology	• Idiopathic or viral pericarditis • Cardiac surgery or radiation therapy • Tuberculosis (in endemic areas)
Pathogenesis	• Rigid pericardium prevents ventricular expansion and restricts diastolic filling • Predominantly right-sided manifestations
Physical examination	• ↑ Jugular venous pressure • Pericardial knock • Pulsus paradoxus • Kussmaul sign

The autopsy finding of thick, fibrous tissue in the pericardial space is consistent with **constrictive pericarditis**, a potential complication of chest radiation therapy for non-Hodgkin lymphoma. This dense, rigid pericardial tissue encases the heart and **restricts ventricular filling**, causing low cardiac output (manifesting with fatigue and dyspnea on exertion) and progressive **right-sided heart failure** (manifesting with hepatomegaly and peripheral edema).

Physical examination in constrictive pericarditis typically shows **elevated jugular venous pressure (JVP)** with prominent x and y descents and a **pericardial knock** (early diastolic sound that occurs before S3) and may also demonstrate **pulsus paradoxus** (>10 mm Hg drop in systolic blood pressure during inspiration). In addition, **Kussmaul sign** may be present. Under normal circumstances, the decrease in intrathoracic pressure during inspiration increases venous return to the right side of the heart and lowers JVP. However, in constrictive pericarditis, the rigid pericardium prevents the right side of the heart from accommodating increased venous return, which leads to a **paradoxical rise in JVP during inspiration**, referred to as Kussmaul sign.

(Choice B) A loud pulmonic component of the second heart sound (P2) is heard in patients with pulmonary

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prominent x and y descents and a **pericardial knock** (early diastolic sound that occurs before S3) and may also demonstrate **pulsus paradoxus** (>10 mm Hg drop in systolic blood pressure during inspiration). In addition, **Kussmaul sign** may be present. Under normal circumstances, the decrease in intrathoracic pressure during inspiration increases venous return to the right side of the heart and lowers JVP. However, in constrictive pericarditis, the rigid pericardium prevents the right side of the heart from accommodating increased venous return, which leads to a **paradoxical rise in JVP during inspiration**, referred to as Kussmaul sign.

(Choice B) A loud pulmonic component of the second heart sound (P2) is heard in patients with pulmonary hypertension.

(Choice C) During inspiration, venous return to the right side of the heart is increased, resulting in later closure of the pulmonic valve and physiologic splitting of the aortic (A2) and pulmonic components of S2. Paradoxical splitting occurs when cardiac pathology (eg, aortic stenosis, left bundle branch block) delays closure of the aortic valve, causing A2 to occur noticeably later than P2.

(Choice D) An S3 gallop occurs due to the sudden deceleration of blood as it enters a dilated ventricle; it is typically heard in patients with dilated cardiomyopathy or severe mitral regurgitation.

(Choice E) A sustained left parasternal lift is most commonly a sign of right ventricular hypertrophy, such as occurs with chronic right ventricular pressure overload (eg, pulmonary hypertension, pulmonic stenosis) or volume overload (eg, tricuspid regurgitation).

Educational objective:
In constrictive pericarditis, normal pericardium is replaced by dense, rigid pericardial tissue that restricts ventricular filling, leading to low cardiac output and progressive right-sided heart failure. Physical examination findings in such patients include elevated jugular venous pressure (JVP), pericardial knock, pulsus paradoxus, and a paradoxical rise in JVP with inspiration (Kussmaul sign).

References

- Constrictive pericarditis: diagnostic aspects and surgical results in 23 cases.

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A 10-year-old boy is brought to the clinic by his parents due to chronic fatigue, shortness of breath, and failure to gain weight. The child was recently adopted from an orphanage in Asia. He weighs 20 kg (44.2 lb). Vital signs are within normal limits. Physical examination reveals a loud systolic murmur. Echocardiogram shows pulmonary stenosis and subaortic ventricular septal defect with deviation of the origin of the aorta to the right. The mother describes occasional episodes of severe dyspnea and cyanosis, during which the child quickly assumes a squatting position. Which of the following mechanisms during squatting relieves this child's symptoms?

- A. Decreasing left ventricular workload
- B. Decreasing pulmonary blood flow
- C. Decreasing pulmonary vascular resistance
- D. Improving lung compliance
- E. Increasing systemic vascular resistance

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- ✗ A. Decreasing left ventricular workload (9%)
- B. Decreasing pulmonary blood flow (3%)
- C. Decreasing pulmonary vascular resistance (10%)
- D. Improving lung compliance (0%)
- ✓ E. Increasing systemic vascular resistance (75%)

Incorrect
Correct answer
E

75%
Answered correctly

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Explanation

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C. Decreasing pulmonary vascular resistance (10%)

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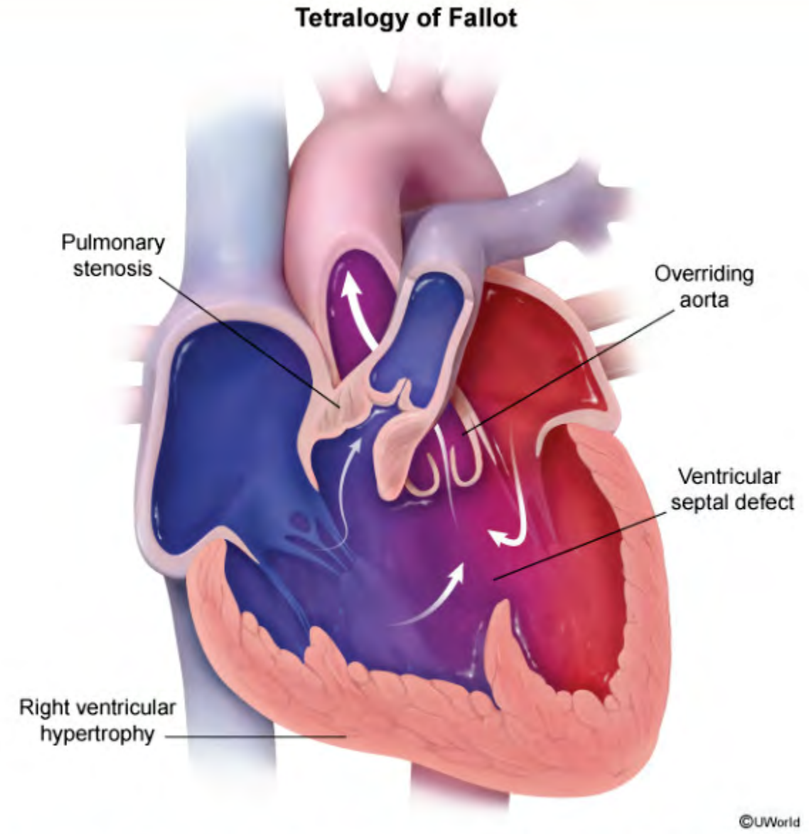

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This child has **Tetralogy of Fallot** (ToF) with ventricular septal defect, overriding aorta, pulmonic stenosis, and right ventricular hypertrophy. The pulmonic stenosis and overriding aorta cause a relatively low ratio of systemic vascular resistance (SVR) to pulmonary vascular resistance (PVR). This low SVR:PVR ratio allows the **deoxygenated** right ventricular output to take the low-resistance route to the **systemic** circulation, leading to acute hypoxemia ("Tet spell"). Tet spells are most common in infancy but can occur at any age in children with unrepaired ToF. The mechanism of Tet spells has not been clearly defined but may be caused by acute changes of the infundibular septum leading to more right ventricular outflow tract obstruction.

Children with ToF quickly learn to squat to relieve Tet spells (infants who cannot squat can be placed in the knee-to-chest position by their caregiver). This posture quickly **increases SVR** without changing PVR, increasing the SVR:PVR ratio. The increased SVR forces a higher proportion of right ventricular output to enter the pulmonary circulation and oxygenate in the pulmonary capillary beds, increasing arterial oxygen concentration and relieving the Tet spell.

(Choices A and D) Squatting restricts chest wall expansion and slightly increases work of breathing. Lung compliance is not changed by this positioning.

(Choices B and C) Squatting does not significantly impact PVR as the compressed vessels are primarily in the abdomen and lower extremities. Pulmonary blood flow increases – not decreases – due to decreased right-to-left shunt with increased SVR.

Educational objective:
In patients with Tetralogy of Fallot, squatting during a Tet spell increases systemic vascular resistance and decreases right-to-left shunting, thereby increasing pulmonary blood flow and improving oxygenation status.

Physiology	Cardiovascular System	Tetralogy of fallot
Subject	System	Topic

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A 44-year-old man reports exertional shortness of breath and palpitations. On examination, he has a systolic murmur at the left sternal border and cardiac apex, which gets louder when he stands up. He is diagnosed with obstructive hypertrophic cardiomyopathy and paroxysmal atrial fibrillation, and is started on a medication. Changes in the action potential of ventricular muscle cells before and after administration of medication are shown in the image below

Black curve = Before administration of medication
Red curve = After administration of medication

The patient is most likely being treated with which of the following medications?

- A. Adenosine
- B. Digoxin
- C. Diltiazem
- D. Disopyramide
- E. Flecainide
- F. Lidocaine
- G. Propranolol

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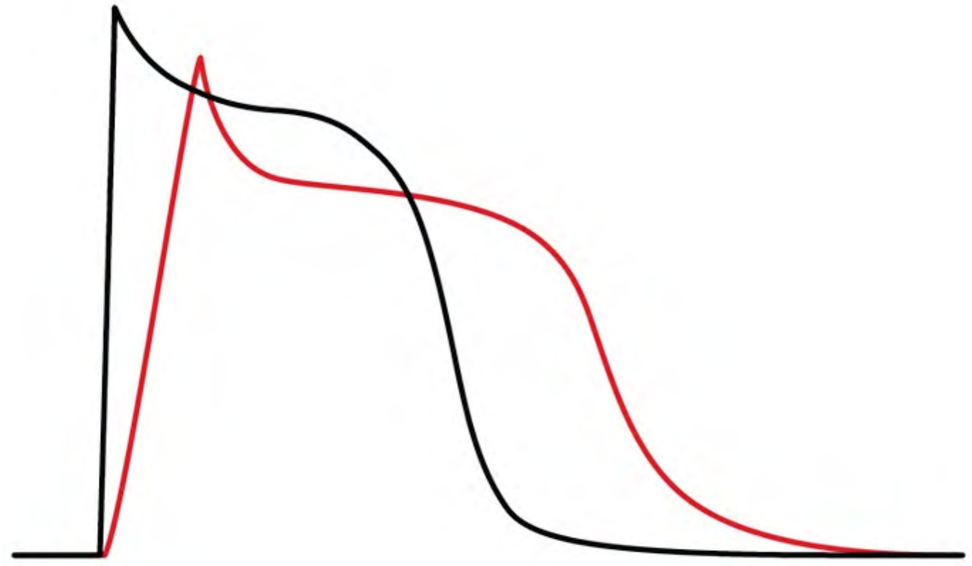

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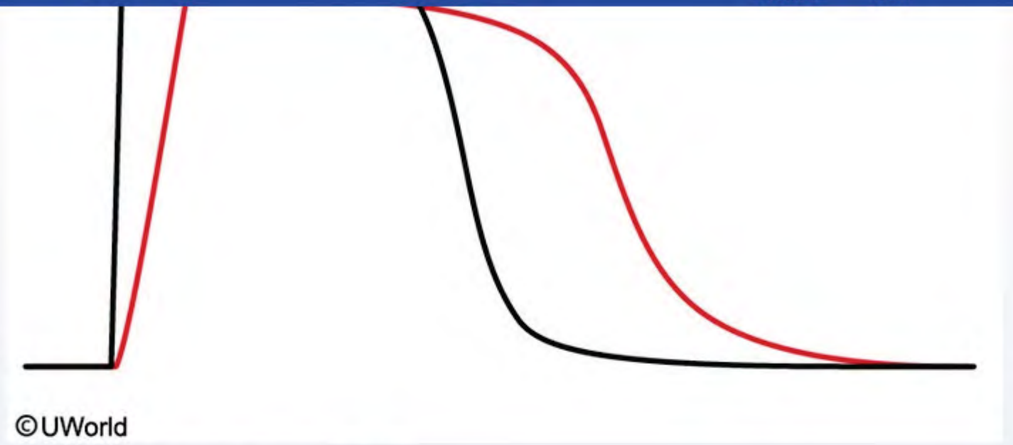

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Black curve = Before administration of medication

Red curve = After administration of medication

The patient is most likely being treated with which of the following medications?

- A. Adenosine (3%)
- ✗ B. Digoxin (5%)
- C. Diltiazem (6%)
- ✓ D. Disopyramide (55%)
- E. Flecainide (15%)
- F. Lidocaine (10%)
- G. Propranolol (3%)

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Correct answer
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55%
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Class I (sodium channel blocking) antiarrhythmics			
	Specific agents	**Inhibition of phase 0 depolarization**	**Effect on length of action potential**
Class IA	Quinidine, procainamide, disopyramide	Intermediate	Prolonged
Class IB	Lidocaine, mexiletine	Weak	Shortened
Class IC	Flecainide, propafenone	Strong	No change

The illustration shows predominant slowing of phase 0 depolarization and prolongation of phase 3 repolarization (red curve) of ventricular action potential after administration of the drug. This action is usually seen with the use of class IA antiarrhythmics drugs (quinidine, procainamide, and disopyramide).

Class I antiarrhythmic drugs block **sodium channels** and inhibit initial phase 0 depolarization of action potential, thereby slowing conduction. Class IA agents also have moderate potassium channel-blocking activity, leading to a slow rate of repolarization and prolongation of action potential duration in the cardiac myocytes.

(Choice A) Adenosine activates potassium channels and increases potassium conductance by interacting with A1 receptors on the surface of cardiac cells. This causes membrane hyperpolarization, resulting in transient slowing of the sinus rate and an increase in atrioventricular (AV) nodal conduction delay. Adenosine does not modulate the ventricular myocyte action potential.

(Choice B) Digoxin exerts its antiarrhythmic effects by increasing vagal output to the AV node and conduction system, thereby slowing conduction in these tissues. It does not alter the ventricular myocyte action potential, but

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(Choice A) Adenosine activates potassium channels and increases potassium conductance by interacting with A1 receptors on the surface of cardiac cells. This causes membrane hyperpolarization, resulting in transient slowing of the sinus rate and an increase in atrioventricular (AV) nodal conduction delay. Adenosine does not modulate the ventricular myocyte action potential.

(Choice B) Digoxin exerts its antiarrhythmic effects by increasing vagal output to the AV node and conduction system, thereby slowing conduction in these tissues. It does not alter the ventricular myocyte action potential, but it does increase intracellular calcium in ventricular myocytes, leading to increased cardiac contractility.

(Choice C) Calcium channel blockers are class IV antiarrhythmic drugs (eg, verapamil, diltiazem) that inhibit L-type calcium channels, slowing depolarization in the sinoatrial and AV nodes and decreasing calcium entry into cardiomyocytes. Calcium channel blockers slow the sinus rate, prolong AV node conduction, and depress myocardial contractility.

(Choice E) Flecainide is a class IC antiarrhythmic that strongly blocks sodium channels. It exhibits a slow rate of receptor dissociation that makes it more effective at higher rates of depolarization (use dependence). Class IC drugs slow phase 0 depolarization but do not affect action potential duration.

(Choice F) Lidocaine is a class IB antiarrhythmic drug that has weak sodium channel-blocking activity at rest; however, it is quite effective in blocking sodium channels in depolarized cardiac myocytes. Class IB drugs also shorten phase 3 repolarization (blockade of "plateau" Na+ current), thereby decreasing action potential duration.

(Choice G) Propranolol is a beta-adrenergic blocking agent that slows conduction through the AV node and prolongs phase 4 depolarization in cardiac pacemaker cells. Beta blockers do not affect ventricular myocyte action potential; their primary site of action is on the AV node and cells with automaticity.

Educational objective:
The class IA antiarrhythmics (quinidine, procainamide, and disopyramide) are sodium channel-blocking agents that depress phase 0 depolarization. They also prolong repolarization due to moderate potassium channel- blocking activity, increasing action potential duration in cardiac myocytes.

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Class IA	Quinidine, procainamide	Intermediate	Prolonged

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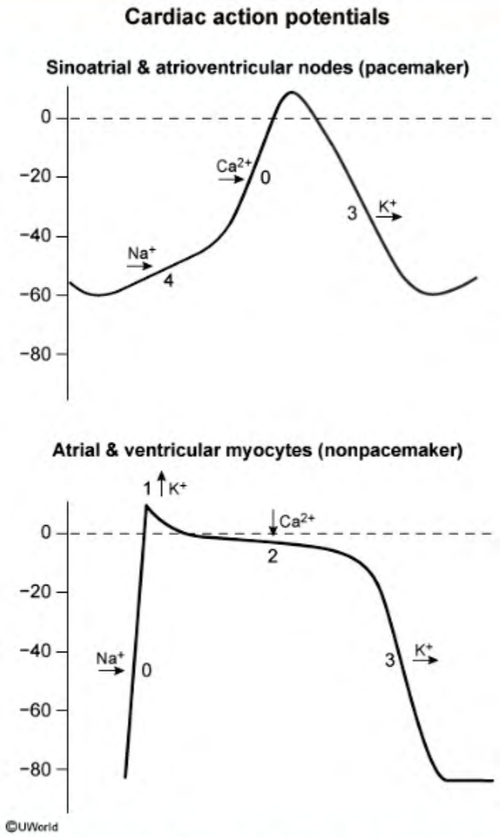

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The illustration shows predominant slowing of phase 0 depolarization and prolongation of phase 3 repolarization

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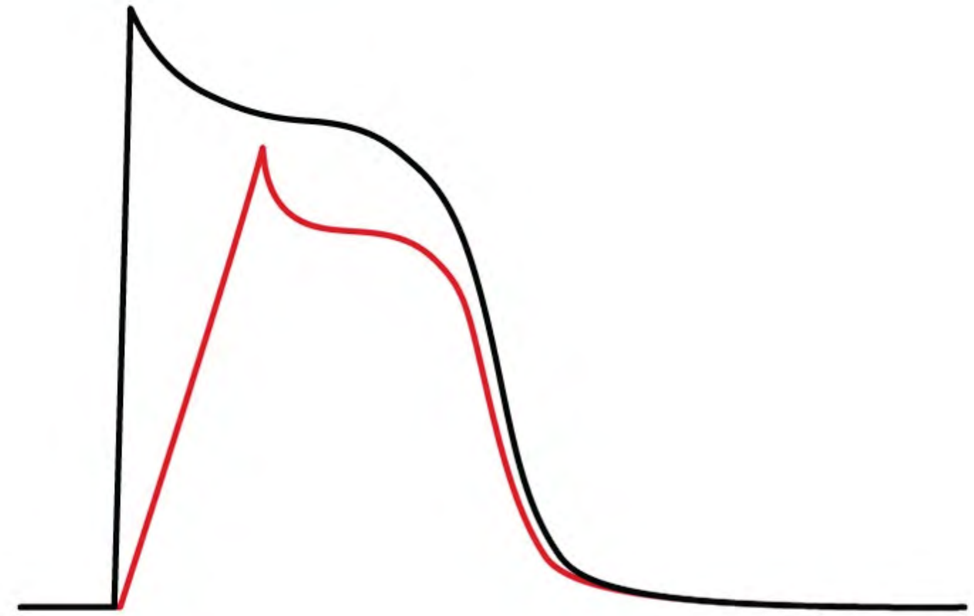

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(Choice G) Propranolol is a beta-adrenergic blocking agent that slows conduction through the AV node and

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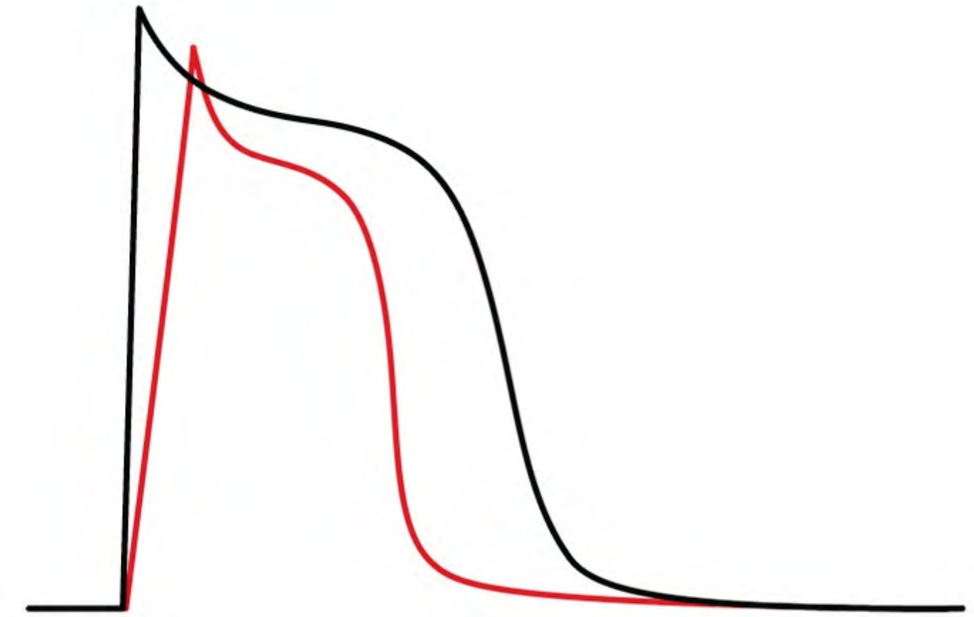

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(Choice G) Propranolol is a beta-adrenergic blocking agent that slows conduction through the AV node and

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A 45-year-old woman comes to the office due to 3 days of progressive pain in the right upper quadrant, nausea, and vomiting. In the preceding year, the patient has had fatigue, weight loss, joint pain, and intermittent abdominal pain. Laboratory studies show increased blood urea nitrogen and serum creatinine. Right-upper quadrant ultrasound shows no evidence of gallstones, but an inflamed gallbladder is present. The patient undergoes cholecystectomy, and subsequent biopsy of the submucosal gallbladder vessels is shown in the exhibit (2 images). Which of the following is the most likely diagnosis?

- A. Angiodysplasia
- B. Antiphospholipid-antibody syndrome
- C. Granulomatosis with polyangiitis
- D. Polyarteritis nodosa
- E. Thromboangiitis obliterans

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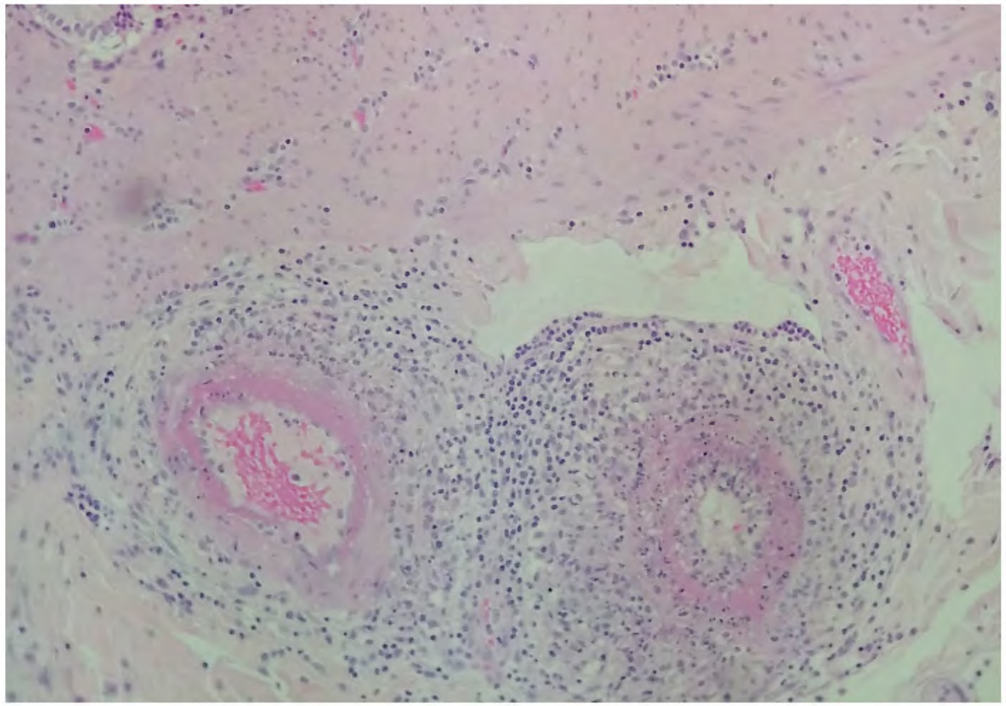

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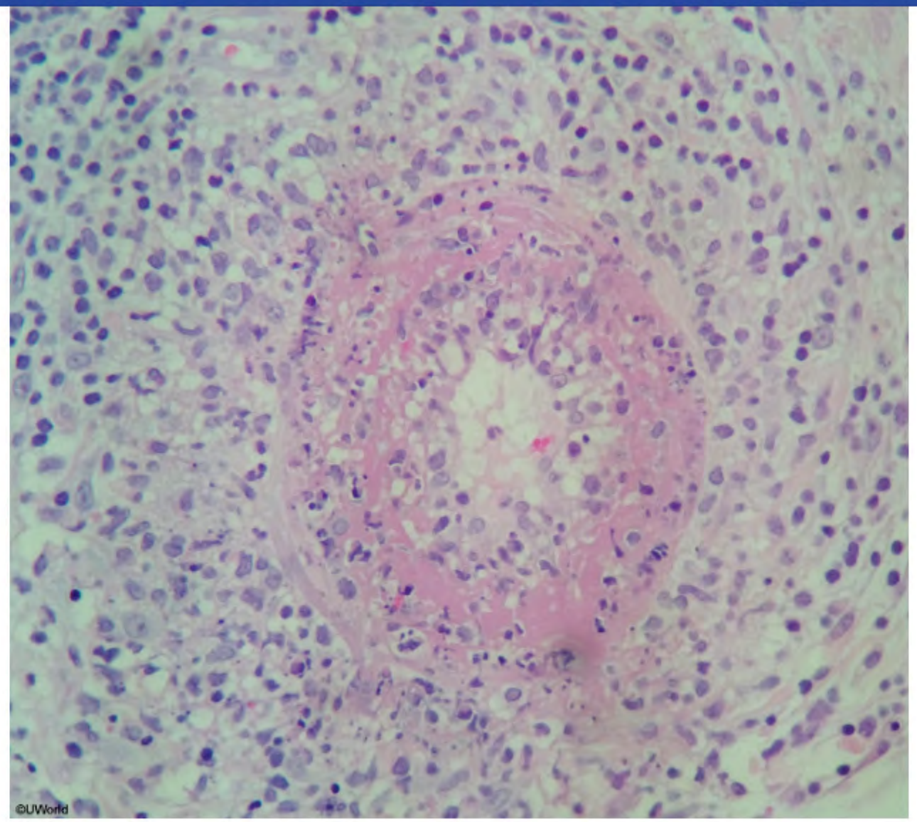

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- A. Angiodysplasia (5%)
- B. Antiphospholipid-antibody syndrome (10%)
- C. Granulomatosis with polyangiitis (21%)
- ✓ D. Polyarteritis nodosa (54%)
- ✗ E. Thromboangiitis obliterans (7%)

Incorrect
Correct answer
D

54%
Answered correctly

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Explanation

Polyarteritis nodosa	
Pathophysiology	• Correlation with underlying hepatitis B/C (immune complexes) • Fibrinoid necrosis of arterial wall → luminal narrowing & thrombosis → tissue ischemia • Internal/external elastic lamina damage → microaneurysm formation → rupture & bleeding

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Polyarteritis nodosa	
Pathophysiology	• Correlation with underlying hepatitis B/C (immune complexes) • Fibrinoid necrosis of arterial wall → luminal narrowing & thrombosis → tissue ischemia • Internal/external elastic lamina damage → microaneurysm formation → rupture & bleeding
Clinical features	• Constitutional: fever, weight loss, malaise • Skin: nodules, livedo reticularis, ulcers, purpura • Renal: hypertension, renal insufficiency, arterial aneurysms • Nervous: headache, seizures, mononeuritis multiplex • Gastrointestinal: mesenteric ischemia/infarction • Musculoskeletal: myalgias, arthritis
Diagnosis	• Negative ANCA & ANA • Angiography: microaneurysms & segmental/distal narrowing • Tissue biopsy: nongranulomatous transmural inflammation
ANA = antinuclear antibodies; **ANCA** = antineutrophil cytoplasmic antibodies.	

This patient's vomiting, right upper quadrant pain, and gallbladder inflammation with no evidence of gallstones indicates **acute acalculous cholecystitis**. Although a variety of conditions cause acute acalculous cholecystitis, the presence of long-standing, nonspecific symptoms (eg, **fatigue**, weight loss, **arthralgias**) and biopsy findings of fibrinoid necrosis of the submucosal arteries of the gallbladder raise strong suspicion for **polyarteritis nodosa** (PAN).

PAN is a systemic vasculitis of **medium-sized muscular arteries**; biopsy of affected areas generally reveals the following:

- Segmental fibrinoid necrosis (amorphic, eosinophilic areas) of the vessel wall

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ANA = antinuclear antibodies; **ANCA** = antineutrophil cytoplasmic antibodies.

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PAN is a systemic vasculitis of **medium-sized muscular arteries**; biopsy of affected areas generally reveals the following:

- Segmental fibrinoid necrosis (amorphic, eosinophilic areas) of the vessel wall
- Infiltration of the vessel wall with mononuclear cells and neutrophils
- Internal and external **elastic laminae damage**, which may cause microaneurysms

In contrast to many other vasculitides (eg, granulomatosis with polyangiitis), there is **no granulomatous inflammation** in PAN.

PAN typically attacks arteries of the **kidneys** (eg, increased BUN/creatinine), skin, neurologic system, and **gastrointestinal tract**; most cases present due to symptoms of **tissue ischemia** from artery lumen narrowing/thrombosis or **bleeding** from microaneurysm rupture. In this case, damage to the submucosal gallbladder arteries likely caused gallbladder ischemia, which lead to acalculous cholecystitis. Other common gastrointestinal findings in PAN include bead-like microaneurysm formation in the mesenteric vessels, mesenteric ischemia, and bowel necrosis/rupture.

(Choice A) Angiodysplasia is thought to arise from intermittent obstruction of submucosal veins at the muscularis propria of the gastrointestinal tract. Biopsy shows dilated small vessels lined by thin-walled endothelium; infiltration with inflammatory cells would not be seen. In addition, patients with angiodysplasia generally have gastrointestinal bleeding, not acalculous cholecystitis.

(Choice B) Antiphospholipid-antibody syndrome is characterized by small-vessel thrombosis due to the formation

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gastrointestinal tract; most cases present due to symptoms of **tissue ischemia** from artery lumen narrowing/thrombosis or **bleeding** from microaneurysm rupture. In this case, damage to the submucosal gallbladder arteries likely caused gallbladder ischemia, which lead to acalculous cholecystitis. Other common gastrointestinal findings in PAN include bead-like microaneurysm formation in the mesenteric vessels, mesenteric ischemia, and bowel necrosis/rupture.

(Choice A) Angiodysplasia is thought to arise from intermittent obstruction of submucosal veins at the muscularis propria of the gastrointestinal tract. Biopsy shows dilated small vessels lined by thin-walled endothelium; infiltration with inflammatory cells would not be seen. In addition, patients with angiodysplasia generally have gastrointestinal bleeding, not acalculous cholecystitis.

(Choice B) Antiphospholipid-antibody syndrome is characterized by small-vessel thrombosis due to the formation of autoantibodies against phospholipid-binding proteins. It is often triggered by underlying systemic lupus erythematosus, which can cause long-standing nonspecific symptoms. However, acute biopsy would show thrombosed vessels, not vessels with fibrinoid necrosis and neutrophilic inflammation.

(Choice C) Granulomatosis with polyangiitis is an antineutrophil cytoplasmic antibody–positive vasculitis that generally affects the upper airways, lungs, and kidneys. Gastrointestinal involvement is uncommon.

(Choice E) Thromboangiitis obliterans primarily attacks vessels in the extremities. It is marked by inflammatory thrombi with relative sparing of the vessel wall and intact internal elastic lamina. Fibrinoid necrosis would not be seen. Most cases are seen in young smokers.

Educational objective:
Polyarteritis nodosa is characterized by segmental, transmural, necrotizing inflammation of medium-sized muscular arteries. Manifestations arise due to tissue ischemia from arterial lumen narrowing/thrombosis or bleeding from microaneurysms. The vessels of the kidneys, skin, peripheral nerves, and gastrointestinal tract are affected most commonly; the lung is typically spared.

Pathology	Cardiovascular System	Polyarteritis nodosa
Subject	System	Topic

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- Negative ANCA & ANA

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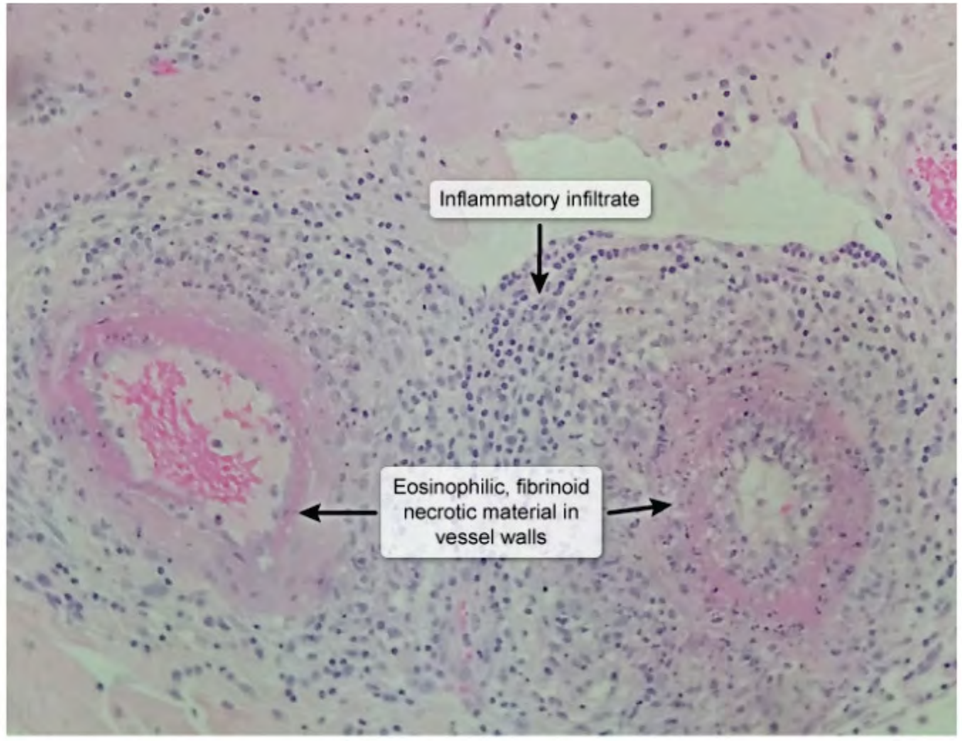
Polyarteritis nodosa



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Gallbladder - vasculitis

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(Choice A) Angiodysplasia is thought to arise from intermittent obstruction of submucosal veins at the muscularis propria of the gastrointestinal tract. Biopsy shows dilated small vessels lined by thin-walled endothelium; infiltration

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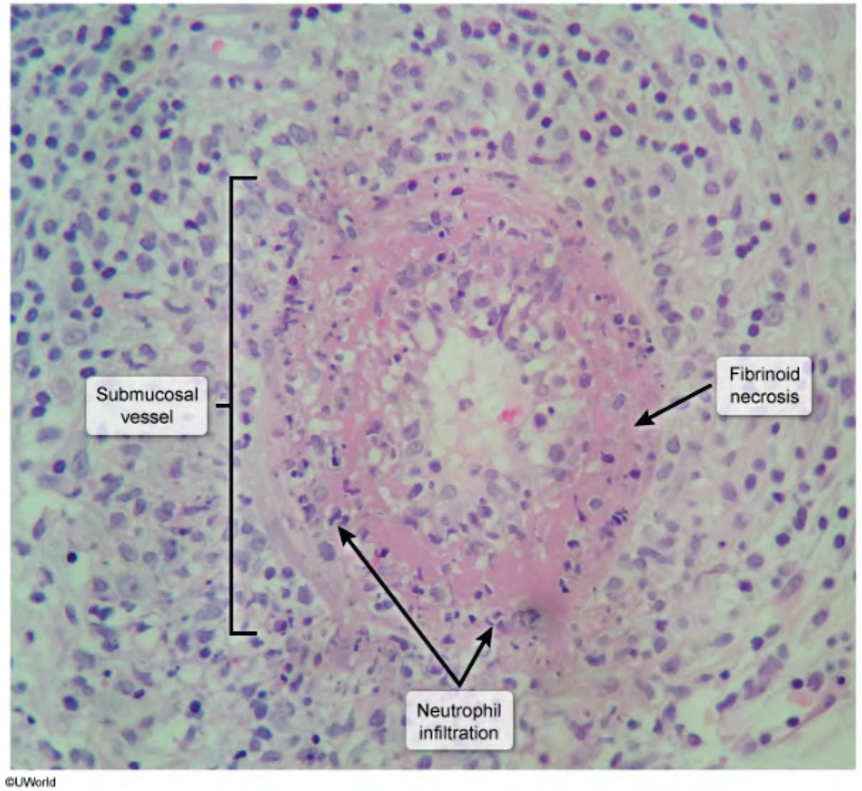
Polyarteritis nodosa



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A 65-year-old man is admitted to the hospital with an acute ST-segment elevation myocardial infarction. The patient undergoes coronary angiography with stent placement in the left circumflex artery, and he is started on therapy with a beta blocker and antiplatelet agents. On day 3 of hospitalization he experiences chest pain. The pain, described as sharp and radiating to his neck and shoulders, is exacerbated by coughing and swallowing. He has no shortness of breath, lightheadedness, or abdominal pain. Temperature is 37.5 C (99.5 F), blood pressure is 130/80 mm Hg, pulse is 90/min and regular, and respirations are 20/min. The lungs are clear on auscultation. There is no peripheral edema. Which of the following is the most likely cause of this patient's chest pain?

- A. Pericardial inflammation due to autoimmune reaction to necrotic tissue
- B. Pericardial inflammation due to concomitant viral infection
- C. Pericardial inflammation overlying the necrotic segment of myocardium
- D. Recurrent thrombosis of the culprit coronary vessel
- E. Thrombosis of a nonculprit coronary vessel

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- ✗ A. Pericardial inflammation due to autoimmune reaction to necrotic tissue (14%)
- B. Pericardial inflammation due to concomitant viral infection (2%)
- ✓ C. Pericardial inflammation overlying the necrotic segment of myocardium (69%)
- D. Recurrent thrombosis of the culprit coronary vessel (8%)
- E. Thrombosis of a nonculprit coronary vessel (5%)

Incorrect
Correct answer
C

69%
Answered correctly

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Explanation

Acute pericarditis	
Etiology	• Viral or idiopathic • Autoimmune disease (eg, SLE) • Uremia (acute or chronic renal failure) • Post myocardial infarction

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Acute pericarditis	

Etiology	• Viral or idiopathic
	• Autoimmune disease (eg, SLE)
	• Uremia (acute or chronic renal failure)
	• Post myocardial infarction
	  ◦ Early: peri-infarction pericarditis
	  ◦ Late: Dressler syndrome

Clinical features & diagnosis	• Pleuritic chest pain (↓ when sitting up) ± fever • Pericardial friction rub • ECG: diffuse ST-segment elevation • Pericardial effusion on echocardiogram
SLE = systemic lupus erythematosus.	

The **sharp** and **pleuritic** nature of this patient's new chest pain suggests pericardial involvement. The exacerbation with swallowing indicates that the posterior pericardium may be involved, and the radiation into the neck suggests involvement of the inferior pericardium, which is adjacent to phrenic nerve afferents supplying the diaphragm. The patient's low-grade fever indicates that this is an inflammatory process.

A fibrinous or serofibrinous pericarditis, known as **peri-infarction pericarditis** (PIP), develops in about 10%-20% of patients between **2 and 4 days** following a **transmural myocardial infarction** (MI). This pericarditis is a reaction to necrosis of the myocardium near the epicardial surface; delayed treatment presentation increases the risk of PIP due to higher levels of necrosis. Inflammation of the visceral and parietal pericardium is usually localized to the areas overlying the necrotic myocardial segment. The condition is generally short-lived and resolves with several days of supportive care; sometimes therapy with high-dose aspirin is needed.

(Choice A) Dressler syndrome (postcardiac injury syndrome) is an autoimmune-mediated pericarditis that is likely

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neck suggests involvement of the inferior pericardium, which is adjacent to phrenic nerve afferents supplying the diaphragm. The patient's low-grade fever indicates that this is an inflammatory process.

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(Choice A) Dressler syndrome (postcardiac injury syndrome) is an autoimmune-mediated pericarditis that is likely provoked by antigens exposed or created by infarction and necrosis of the cardiac muscle. It is less common and has later onset than PIP, typically occurring a week to a few months after an MI. The pericardium is often diffusely affected in Dressler syndrome.

(Choice B) Viral pericarditis usually follows an antecedent upper respiratory infection. However, a viral cause for this patient's pericarditis is less likely than PIP as a direct complication of his transmural infarction.

(Choices D and E) Recurrent thrombosis of the recently stented vessel or thrombosis of another coronary vessel would be expected to cause similar pain to the patient's original, anginal type of pain. The pain of myocardial ischemia is not typically sharp or pleuritic, but rather constant, substernal, and "crushing."

Educational objective:
In contrast to angina, the chest pain of pericarditis is sharp and pleuritic and may be exacerbated by swallowing or coughing. Peri-infarction pericarditis (PIP) occurs between 2 and 4 days following a transmural myocardial infarction (MI). PIP is an inflammatory reaction to cardiac muscle necrosis that occurs in the adjacent pericardium.

Pathology	Cardiovascular System	Acute pericarditis
Subject	System	Topic

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A 27-year-old man comes to the emergency department due to acute-onset chest pain and shortness of breath. The pain started suddenly an hour ago while he was lifting a box of books. The patient has no chronic medical conditions, and he was in his usual state of health prior to the onset of symptoms. He does not use tobacco, alcohol, or illicit drugs. Height is 182 cm (71.7 in) and weight is 72 kg (158.7 lb). On physical examination, the patient is in acute respiratory distress. Temperature is 37 C (98.6 F), blood pressure is 90/60 mm Hg, pulse is 120/min and regular, and respirations are 26/min. Pulse oximetry shows 86% saturation on room air. Cardiopulmonary examination reveals pectus excavatum, bilateral pulmonary crackles, and a decrescendo diastolic murmur at the right upper sternal border. The abdomen is soft and nontender. There is no peripheral edema. Subsequently, the patient develops cardiac arrest and dies. Which of the following histologic changes is most likely to be seen on autopsy?

- A. Atherosclerotic thickening of the intima
- B. Coagulative necrosis of the left ventricular wall
- C. Fragmentation of the elastic lamellae in the aortic media
- D. Necrosis of the papillary muscles
- E. Perivascular inflammation around the vasa vasorum

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- A. Atherosclerotic thickening of the intima (2%)
- ✗ B. Coagulative necrosis of the left ventricular wall (2%)
- ✓ C. Fragmentation of the elastic lamellae in the aortic media (87%)
- D. Necrosis of the papillary muscles (3%)
- E. Perivascular inflammation around the vasa vasorum (4%)

Incorrect
Correct answer
C

87%
Answered correctly

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Explanation

Aortic wall

Normal **Marfan syndrome**

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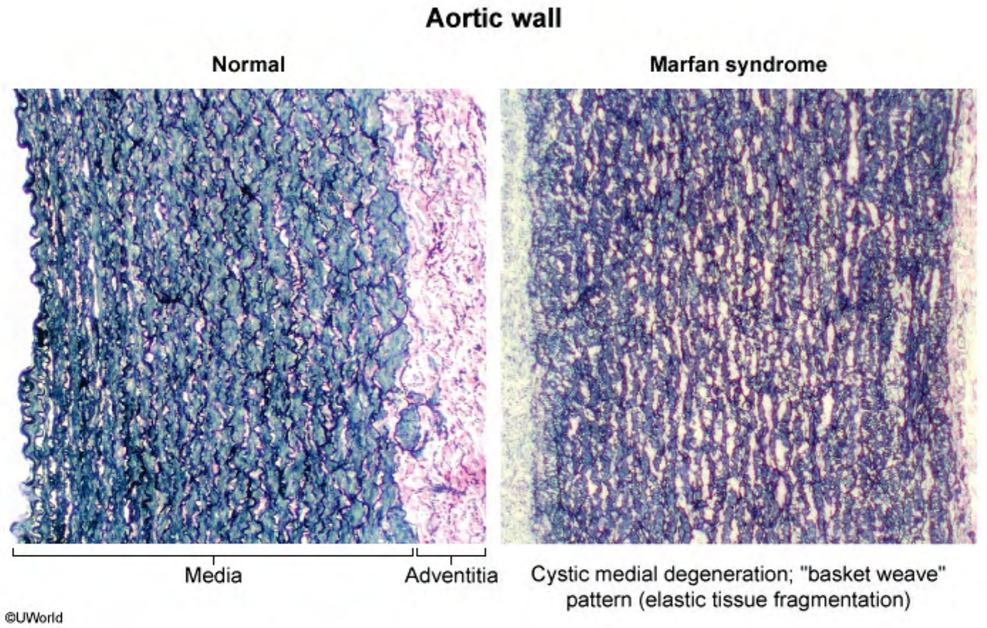

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Normal Marfan syndrome

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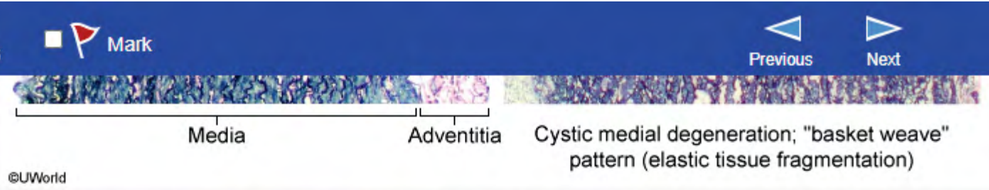

This young patient likely had Marfan syndrome complicated by aortic root disease, leading to **aortic dissection** with **acute aortic regurgitation** and heart failure. Aortic dissection typically presents with sudden-onset, severe chest or back pain. In Marfan syndrome, the dissection almost always affects the ascending aorta; it can propagate proximally to impair aortic valve closure, causing acute aortic regurgitation (evidenced by a decrescendo diastolic murmur) and heart failure (evidenced by pulmonary edema).

Marfan syndrome results from a mutation that disrupts the synthesis, secretion, and incorporation into the extracellular matrix of **fibrillin**, a protein that provides the glycoprotein scaffolding for elastin structure. Although affected patients are often tall in stature, the syndrome is more reliably recognized by an increased ratio of limb length to trunk length.

The most common histologic findings in Marfan-related aortic root disease include **fragmentation** and loss of the **elastic lamellae** with fibrosis and cystic medial degeneration (replacement of collagen, elastin, and smooth muscle by a basophilic mucoid extracellular matrix with irregular fiber cross-linkages and cystic collections of mucopolysaccharide). Cystic medial degeneration also occurs with normal aging, but is accelerated in Marfan syndrome.

(Choices A and B) Fatty streaking (the early stages of atherosclerosis) is often present in male patients in their 20s, but significant atherosclerotic thickening of the intima is not expected. Coagulative necrosis of the left ventricular wall typically results from atherosclerotic plaque rupture leading to myocardial infarction, which is unlikely in the absence of significant atherosclerotic disease.

(Choice D) Myocardial infarction can cause papillary muscle necrosis and rupture. This mechanical complication typically occurs acutely or within 3-5 days of myocardial infarction and leads to acute mitral regurgitation with a holosystolic – rather than diastolic decrescendo – murmur.

(Choice E) Inflammation of the vasa vasorum can lead to aortitis and aneurysm formation in syphilis and certain

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(Choice E) Inflammation of the vasa vasorum can lead to aortitis and aneurysm formation in syphilis and certain vasculitides (eg, Behçet syndrome). However, such diseases are associated with multiple other manifestations (eg, rash, ulcer, airway disease) and would not explain this patient's pectus excavatum.

Educational objective:
Marfan syndrome involves a deleterious mutation in fibrillin that mainly affects the structural integrity of the cardiovascular and musculoskeletal systems. Aortic root disease predisposes to aortic dissection, which can present with sudden-onset chest or back pain, acute aortic regurgitation, and heart failure. Common histologic findings in aortic disease include fragmentation and loss of the elastic lamellae with fibrosis and cystic medial degeneration.

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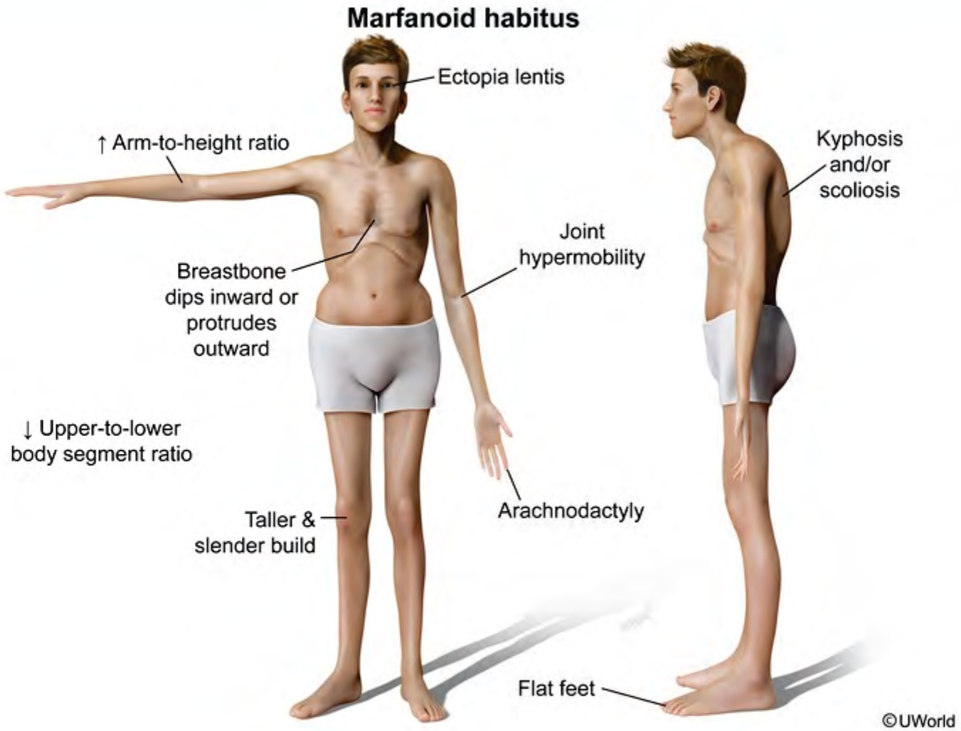

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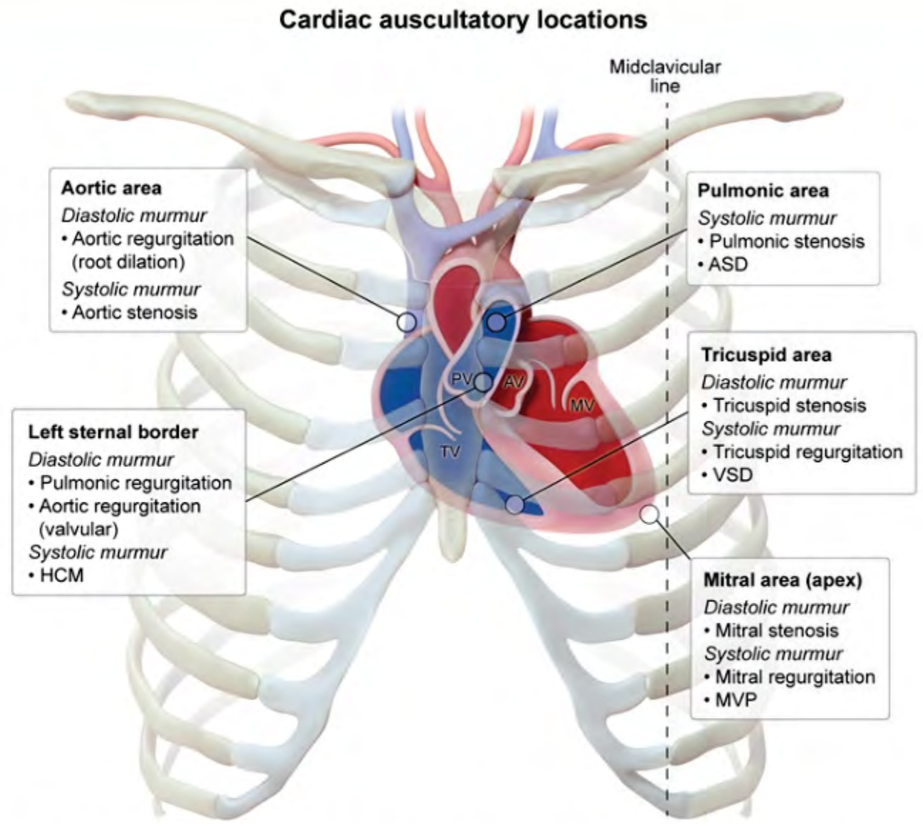

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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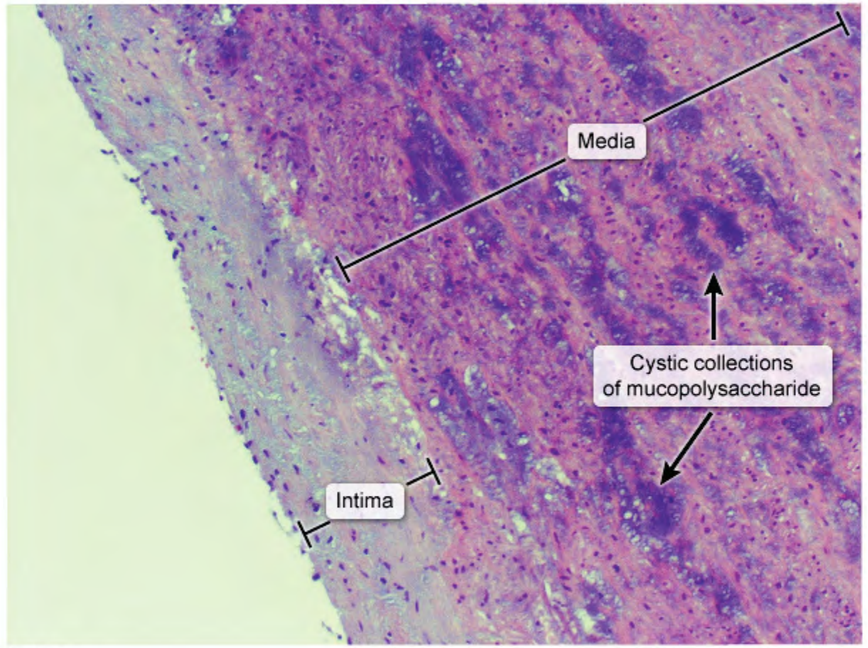
Exhibit Display

Cystic medial degeneration of aorta



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Atherosclerotic plaque

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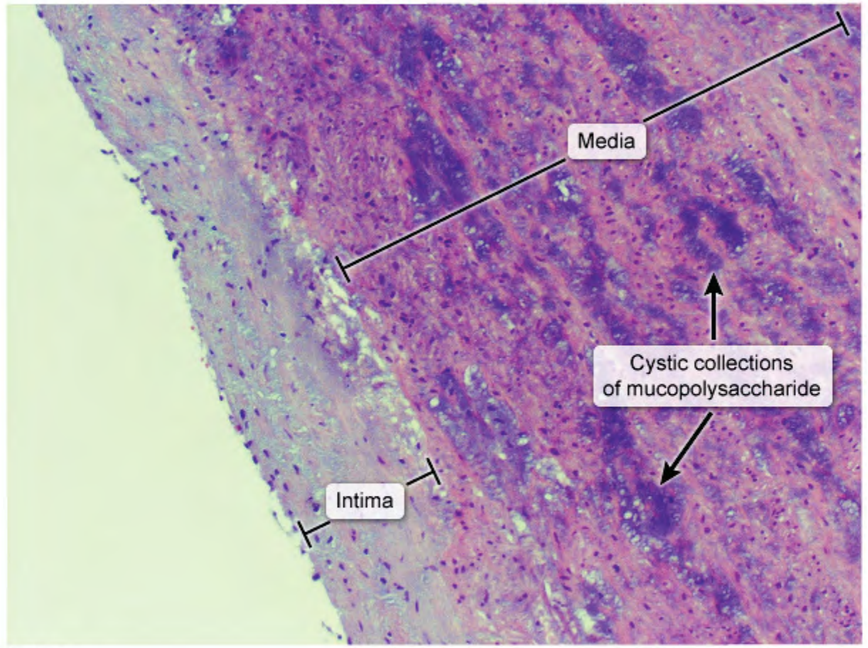
Cystic medial degeneration of aorta



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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 54-year-old man comes to the office due to light-headedness when walking his dog in the morning. He has had no chest pain, shortness of breath, weakness, or headaches. Cardiac auscultation findings at the right sternal border are heard in the exhibit. Which of the following is the most likely cause of this patient's symptoms?

Play Media

- A. Bicuspid aortic valve
- B. Infective endocarditis
- C. Marfan syndrome
- D. Mitral valve prolapse
- E. Ventricular septal defect

Submit

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Explanation

Cardiac auscultation in this patient reveals a harsh, crescendo-decrescendo systolic ejection murmur which is classic for **aortic stenosis** (AS). The murmur is typically best heard at the right upper sternal border with radiation to the carotids. Patients with AS usually remain asymptomatic for a long time and **gradually develop symptoms** (eg, exertional dyspnea, angina, syncope) as the stenosis becomes severe. This patient's light-headedness while walking likely represents **exertional presyncope** due to inadequate cardiac output from severe AS.

Rheumatic heart disease is the most common cause of AS worldwide. However, in the United States, AS most commonly results from age-related calcification of a normal trileaflet or a congenitally abnormal bicuspid aortic valve. Calcification of a **bicuspid aortic valve** is accelerated, causing patients to develop AS at a younger age compared to those with a normal trileaflet valve.

(Choice B) Infective endocarditis (IE) usually causes a regurgitant, rather than a stenotic, murmur. IE affecting the mitral valve can cause a systolic murmur of mitral regurgitation, but the murmur is usually holosystolic and best heard at the cardiac apex. In addition, IE presenting with exertional presyncope would be unusual; fever and fatigue are more typical.

(Choice C) Marfan syndrome is associated with aortic root dilation, which can cause aortic regurgitation. However, the murmur of aortic regurgitation is a decrescendo diastolic murmur.

(Choice D) Mitral valve prolapse produces a midsystolic click that may be followed by a late systolic murmur of mitral regurgitation, best heard at the cardiac apex.

(Choice E) A ventricular septal defect (VSD) typically causes a holosystolic murmur best heard at the left lower sternal border. A VSD first presenting in an adult age >50 would be rare and would typically present with symptoms of heart failure (eg, dyspnea) rather than exertional presyncope.

Educational objective:
A bicuspid aortic valve is a common cause of aortic stenosis in the United States. The classic auscultatory finding in aortic stenosis is a harsh, crescendo-decrescendo systolic ejection murmur best heard at the right upper sternal

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classic for **aortic stenosis** (AS). The murmur is typically best heard at the right upper sternal border with radiation to the carotids. Patients with AS usually remain asymptomatic for a long time and **gradually develop symptoms** (eg, exertional dyspnea, angina, syncope) as the stenosis becomes severe. This patient's light-headedness while walking likely represents **exertional presyncope** due to inadequate cardiac output from severe AS.

Rheumatic heart disease is the most common cause of AS worldwide. However, in the United States, AS most commonly results from age-related calcification of a normal trileaflet or a congenitally abnormal bicuspid aortic valve. Calcification of a **bicuspid aortic valve** is accelerated, causing patients to develop AS at a younger age compared to those with a normal trileaflet valve.

(Choice B) Infective endocarditis (IE) usually causes a regurgitant, rather than a stenotic, murmur. IE affecting the mitral valve can cause a systolic murmur of mitral regurgitation, but the murmur is usually holosystolic and best heard at the cardiac apex. In addition, IE presenting with exertional presyncope would be unusual; fever and fatigue are more typical.

(Choice C) Marfan syndrome is associated with aortic root dilation, which can cause aortic regurgitation. However, the murmur of aortic regurgitation is a decrescendo diastolic murmur.

(Choice D) Mitral valve prolapse produces a midsystolic click that may be followed by a late systolic murmur of mitral regurgitation, best heard at the cardiac apex.

(Choice E) A ventricular septal defect (VSD) typically causes a holosystolic murmur best heard at the left lower sternal border. A VSD first presenting in an adult age >50 would be rare and would typically present with symptoms of heart failure (eg, dyspnea) rather than exertional presyncope.

Educational objective:
A bicuspid aortic valve is a common cause of aortic stenosis in the United States. The classic auscultatory finding in aortic stenosis is a harsh, crescendo-decrescendo systolic ejection murmur best heard at the right upper sternal border with radiation to the carotids.

Pathophysiology Cardiovascular System Aortic stenosis

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D. Mitral valve prolapse (7%)

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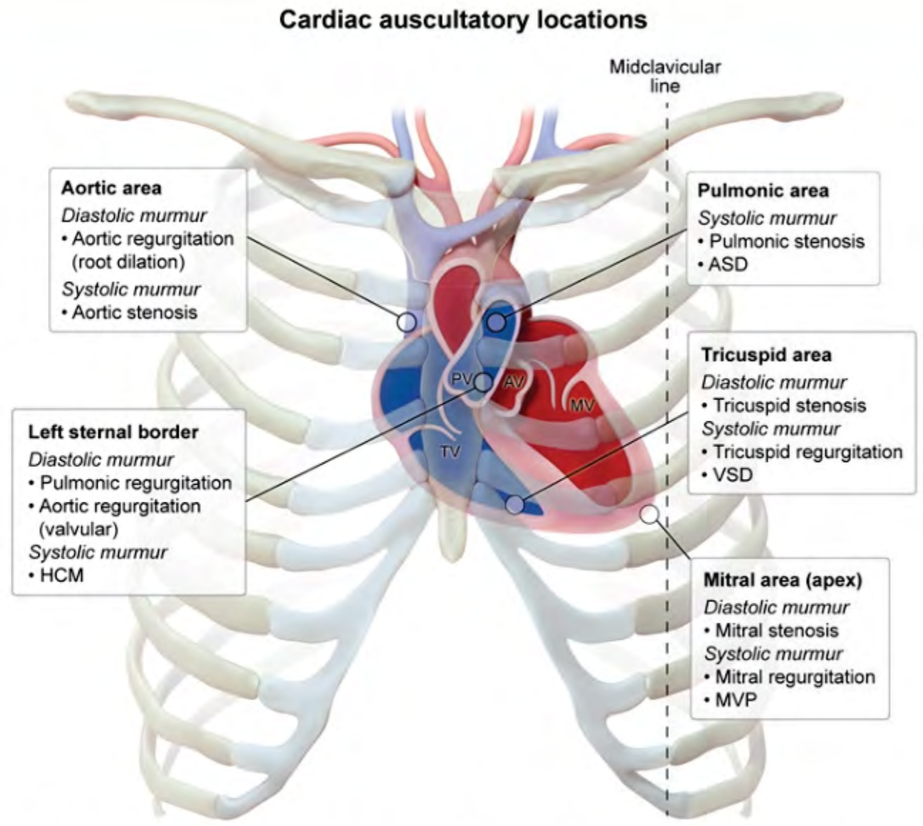

ASD = atrial septal defect; **AV** = aortic valve; **HCM** = hypertrophic cardiomyopathy; **MV** = mitral valve; **MVP** = mitral valve prolapse; **PV** = pulmonic valve; **TV** = tricuspid valve; **VSD** = ventricular septal defect.
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A 32-year-old woman comes to the emergency department due to sudden-onset, left-sided facial droop and arm weakness. The patient has also had right leg swelling after a recent road trip. She has no chronic medical conditions, and her only medication is an oral contraceptive pill. CT scan of the head reveals an acute infarction in the right middle cerebral artery territory. Lower extremity ultrasonography demonstrates a right femoral vein thrombus. No obvious cardiac structural abnormalities are detected on echocardiography. Agitated normal saline is injected into a peripheral vein to evaluate for abnormal right-to-left shunting. No bubbles are seen in the left side of the heart after several minutes. Which of the following is most appropriate for increasing right-sided heart pressure to reveal an obscured intracardiac shunt?

- A. Abrupt standing from the supine position
- B. Amyl nitrite injection
- C. Breath-holding after exhalation
- D. Sustained handgrip
- E. Valsalva maneuver release phase

Submit

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- ✗ A. Abrupt standing from the supine position (6%)
- B. Amyl nitrite injection (6%)
- C. Breath-holding after exhalation (19%)
- D. Sustained handgrip (27%)
- ✓ E. Valsalva maneuver release phase (39%)

Incorrect
Correct answer
E

39%
Answered correctly

09 secs
Time Spent

2023
Version

Explanation

This patient has deep vein thrombosis and has experienced a large stroke, suggesting **paradoxical embolization** of thrombus (ie, passage from the venous circulation to the arterial circulation). Because thrombi are too large to pass through the pulmonary capillaries, paradoxical embolization typically occurs via an **intracardiac right-to-left shunt**. In this case, echocardiography did not reveal overt septal abnormalities (ie, atrial or ventricular septal defect), raising strong suspicion for a **patent foramen ovale** (PFO).

PFO is common, affecting up to 30% of adults. It results from failure of the septum primum and septum secundum

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Explanation

This patient has deep vein thrombosis and has experienced a large stroke, suggesting **paradoxical embolization** of thrombus (ie, passage from the venous circulation to the arterial circulation). Because thrombi are too large to pass through the pulmonary capillaries, paradoxical embolization typically occurs via an **intracardiac right-to-left shunt**. In this case, echocardiography did not reveal overt septal abnormalities (ie, atrial or ventricular septal defect), raising strong suspicion for a **patent foramen ovale** (PFO).

PFO is common, affecting up to 30% of adults. It results from failure of the septum primum and septum secundum to fuse during early childhood, creating a **one-way valve**. This valve is forced closed when left atrial pressure exceeds right atrial pressure (which is most of the time), but it opens when right atrial pressure **exceeds** left atrial pressure, allowing for transient right-to-left shunting.

In testing for a PFO, normal saline bubbles are injected intravenously, and echocardiography is performed to observe whether the bubbles pass from the right into the left atrium. The release phase of the **Valsalva maneuver** increases venous return to the right atrium and consequently increases right atrial pressure to facilitate right-to-left shunting of saline bubbles. In contrast, the strain phase of Valsalva maneuver decreases venous return to the right atrium and discourages shunting through a PFO.

(Choices A, B, and C) Medications and physiologic maneuvers that reduce venous return to the right atrium discourage right-to-left shunting through a PFO. Abrupt standing from a supine position causes blood to pool in the lower extremities, amyl nitrite (an inhaled nitrite) causes venous vasodilation, and exhalation causes increased intrathoracic pressure (resulting in partial collapse of the inferior vena cava at end-expiration), all of which reduce venous return to the right atrium.

(Choice D) Sustained handgrip increases systemic vascular resistance, increasing pressure in the left ventricle and left atrium and discouraging shunting through a PFO.

Educational objective:
Paradoxical embolization, or passage of a thromboembolism from the venous to the arterial circulation, most commonly occurs through a patent foramen ovale (PFO). A PFO is a one-way tissue valve in the atrial septum that opens only when right exceeds left atrial pressure; venous return to the right atrium increases during the release

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of thrombus (ie, passage from the venous circulation to the arterial circulation). Because thrombi are too large to pass through the pulmonary capillaries, paradoxical embolization typically occurs via an **intracardiac right-to-left shunt**. In this case, echocardiography did not reveal overt septal abnormalities (ie, atrial or ventricular septal defect), raising strong suspicion for a **patent foramen ovale** (PFO).

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Educational objective:
Paradoxical embolization, or passage of a thromboembolism from the venous to the arterial circulation, most commonly occurs through a patent foramen ovale (PFO). A PFO is a one-way tissue valve in the atrial septum that opens only when right exceeds left atrial pressure; venous return to the right atrium increases during the release (ie, relaxation) phase of the Valsalva maneuver and encourages the PFO to open.

Physiology	Cardiovascular System	Patent foramen ovale
Subject	System	Topic

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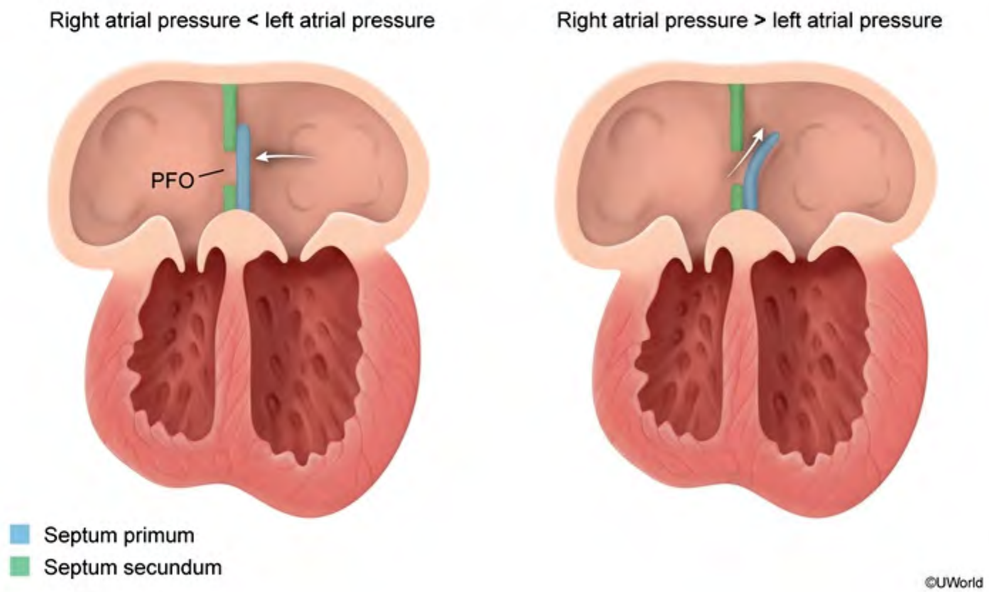
Intermittent right-to-left shunting through a patent foramen ovale (PFO)



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Physiology (Subject) | Cardiovascular System (System) | Patent foramen ovale (Topic)

https://t.me/USMLEWorldStep1

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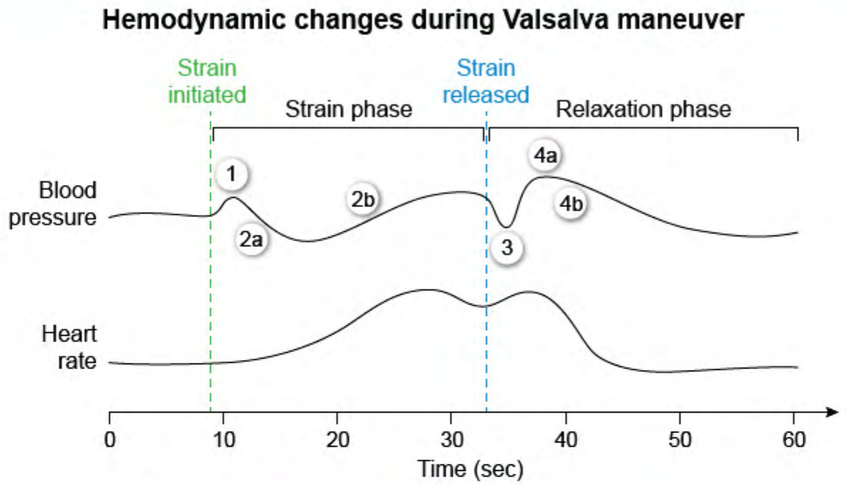

------ Strain initiated (↑ intrathoracic pressure) ------

1. Pressure directly transmitted to aorta
2a. Reduced venous return to RA leads to reduced CO
2b. Compensatory ↑ in SVR & heart rate

----- Strain released (↓ intrathoracic pressure) -----

3. Blood surges into RA & pulmonary circulation
4a. ↑ Venous return ejected into constricted vasculature
4b. ↑ Vagal tone returns hemodynamics to baseline

CO = cardiac output; **RA** = right atrium; **SVR** = systemic vascular resistance.

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Physiology
Subject

Cardiovascular System
System

Patent foramen ovale
Topic

https://t.me/USMLEWorldStep1

A 56-year-old woman presents to your office with chronic cough. She says that the cough is dry and affects quality of her life significantly. She denies chest pain, hemoptysis and shortness of breath. Her past medical history is significant for long-standing hypertension, diabetes and myocardial infarction experienced two months ago. She does not smoke or consume alcohol. Her blood pressure is 130/70 mmHg and heart rate is 70/min. Which of the following is the best next step in the management of this patient?

- A. Careful review of family history
- B. Careful review of past allergic episodes
- C. Careful review of current medications
- D. Careful review of diet and physical activity
- E. Chest x-ray

Submit

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- ✗ A. Careful review of family history (1%)
- B. Careful review of past allergic episodes (1%)
- ✓ C. Careful review of current medications (90%)
- D. Careful review of diet and physical activity (1%)
- E. Chest x-ray (5%)

Incorrect
Correct answer
C

90%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation

With a history of hypertension, diabetes, and coronary heart disease, the likelihood that this patient is on standard ACE inhibitor therapy is very high. A dry, nonproductive, and persistent cough is one of the most well-known side effects of ACE inhibitor therapy.

The mechanism of ACE inhibitor induced cough is believed to involve increased bradykinin, substance P, or prostaglandins. ACE is a very nonspecific enzyme responsible for the breakdown of many substrates. By blocking ACE, ACE inhibitors block degradation of bradykinin and substance P. Bradykinin and substance P accumulation can cause inflammation and stimulate lung irritation.

Because angiotensin receptor blockers (ARBs) do not affect ACE activity, they theoretically should not cause cough.

https://t.me/USMLEWorldStep1

Explanation

With a history of hypertension, diabetes, and coronary heart disease, the likelihood that this patient is on standard ACE inhibitor therapy is very high. A dry, nonproductive, and persistent cough is one of the most well-known side effects of ACE inhibitor therapy.

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Because angiotensin receptor blockers (ARBs) do not affect ACE activity, they theoretically should not cause cough.

(Choice A) Chronic cough is one of the early signs of cancer. Screening family history will allow risk assessment for cancer, but since patient does not complain of any other symptoms of cancer (unexplained fatigue, weight loss, change in bowel habits, etc.), suspicion for cancer is low.

(Choice B) Although cough can occur secondary to post nasal drip in patients with allergies, the patient describes no other allergy type symptoms (itchy and watery eyes, runny nose, sneezing, etc.).

(Choice D) Diet and physical activity are not linked to causing cough.

(Choice E) Cough may be linked to pulmonary infection or disease. Chest x-ray would be helpful in diagnosis if suspicion for pneumonia, TB, bronchitis, or emphysema were high. However, patient has no symptoms associated with any infection and her risk for emphysema is low since she does not smoke.

Educational Objective:
Cough is a very well recognized side effect of ACE inhibitor therapy. Cough secondary to ACE inhibitor therapy is characterized as dry, nonproductive, and persistent. The mechanism behind ACE inhibitor induced cough is accumulation of bradykinin, substance P, or prostaglandins. Because angiotensin receptor blockers (ARBs) do not affect ACE activity, they theoretically should not cause cough.

https://t.me/USMLEWorldStep1

A 2-year-old, previously healthy boy is brought to the emergency department due to a 4-day history of poor feeding, diarrhea, and abdominal pain. The patient has had 5-7 bowel movements every day for the past 4 days. Temperature is 36.8 C (98.2 F) and blood pressure is 65/42 mm Hg. On physical examination, the patient is lethargic. Mucous membranes are dry, and the eyes are sunken. The lungs are clear to auscultation. No murmurs are heard. Abdominal examination shows distension and tenderness; bowel sounds are hyperactive throughout. Which of the following changes is most likely present in this patient?

- A. Extravasation of fluid from systemic capillaries
- B. Increased arteriolar capacitance
- C. Increased central venous pressure
- D. Increased ventilatory rate
- E. Relaxation of venous smooth muscle

Submit

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- A. Extravasation of fluid from systemic capillaries (16%)
- ✕ B. Increased arteriolar capacitance (9%)
- C. Increased central venous pressure (20%)
- ✓ D. Increased ventilatory rate (47%)
- E. Relaxation of venous smooth muscle (6%)

Incorrect
Correct answer
D

47%
Answered correctly

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Time Spent

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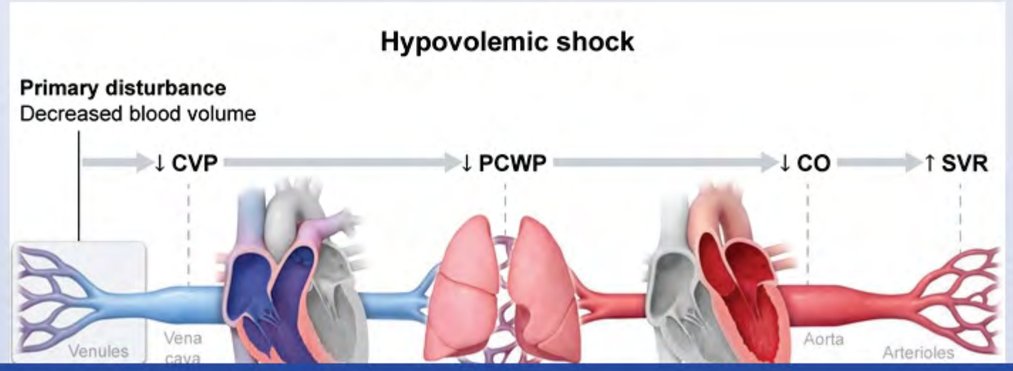
Explanation



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A 2-year-old, previously healthy boy is brought to the emergency department due to a 4-day history of poor
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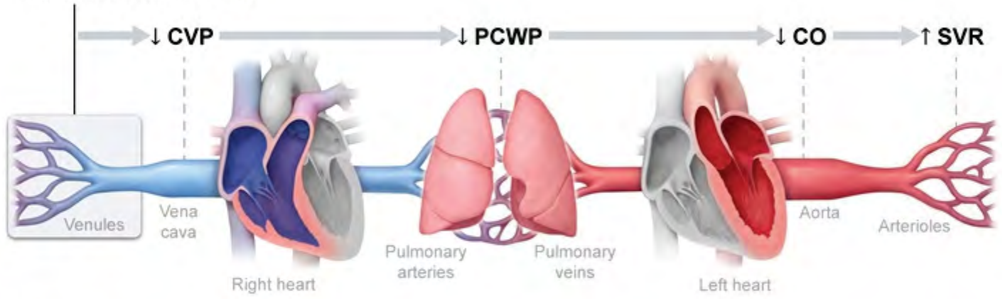
Exhibit Display

Hypovolemic shock

Primary disturbance
Decreased blood volume



CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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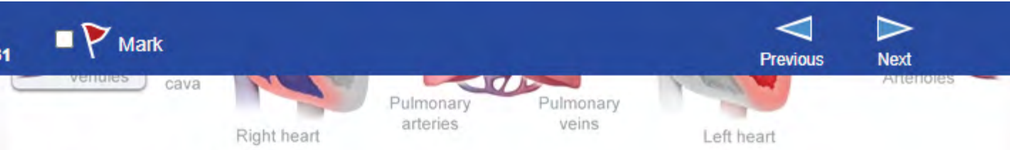

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This patient has had several days of poor oral intake and diarrhea and now has signs of **hypovolemia** (eg, dry mucous membranes, sunken eyes) and **shock** (eg, lethargy, severe hypotension). With shock, there is poor organ and tissue perfusion that leads to **tissue hypoxia** with increased anaerobic metabolism and consequent **lactic acidosis**. In response to the reduction in pH caused by metabolic acidosis there is **increased ventilation** to increase CO_2 exhalation (ie, **compensatory respiratory alkalosis**).

Severe diarrhea causes loss of bicarbonate in the stool, further contributing to metabolic acidosis and a compensatory increase in ventilation in this patient.

(Choices A, B, and E) The reduced cardiac output in hypovolemic shock stimulates arteriolar vasoconstriction to increase systemic vascular resistance and help increase blood pressure. This reduces arteriolar capacitance. There is also contraction of venous smooth muscle to cause venular constriction and help increase venous return to the heart. The constricted vasculature reduces extravasation of intravascular fluid. In contrast, septic shock involves peripheral vasodilation with relaxation of vascular smooth muscle, increased vascular capacitance, and extravasation of intravascular fluid.

(Choice C) Blood volume is markedly decreased in hypovolemic shock, leading to decreased central venous pressure. Central venous pressure is increased in cardiogenic shock and obstructive shock.

Educational objective:
Hypovolemic shock results from markedly decreased blood volume, which reduces central venous pressure and cardiac output and leads to increased systemic vascular resistance (peripheral vasoconstriction). Shock causes reduced organ and tissue perfusion, which leads to lactic acidosis (metabolic acidosis) and a compensatory increase in ventilation (ie, compensatory respiratory alkalosis).

Physiology	Cardiovascular System	Hypovolemic shock
Subject	System	Topic

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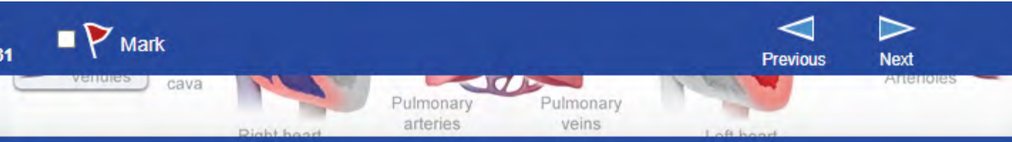

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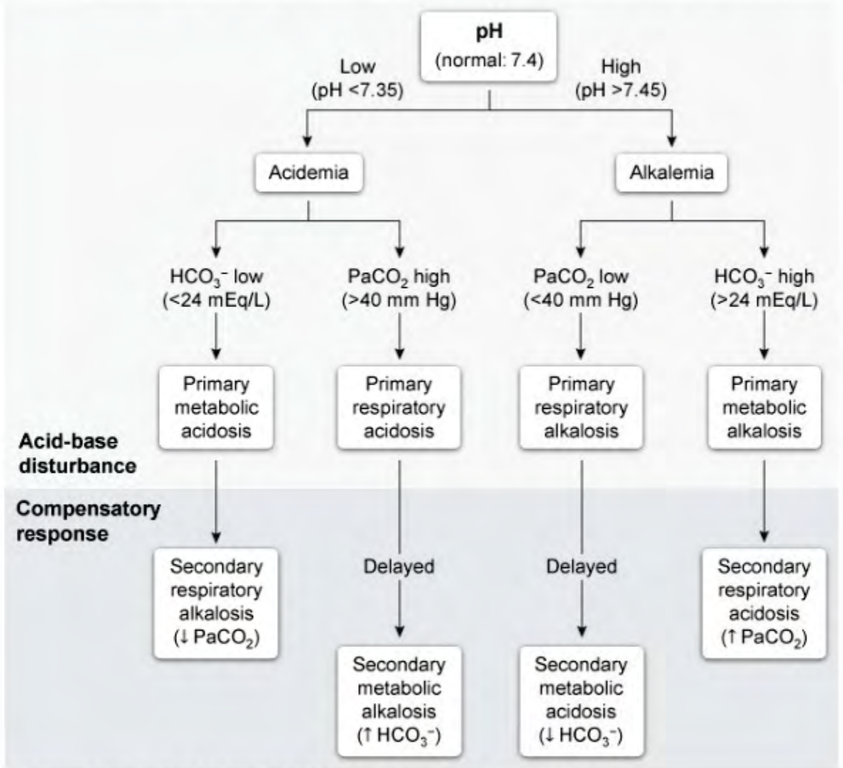
Subject

Arterial blood gas interpretation of acid-base disorders



* The normal ranges for $PaCO_2$ and HCO_3^- vary slightly around 40 mm Hg and 24 mEq/L.
For simplicity, these numbers should be used as a normal baseline for acid-base calculations.

HCO_3^- = bicarbonate; **$PaCO_2$** = partial pressure of carbon dioxide in arterial blood.

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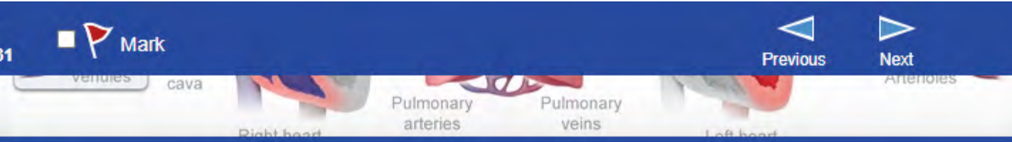

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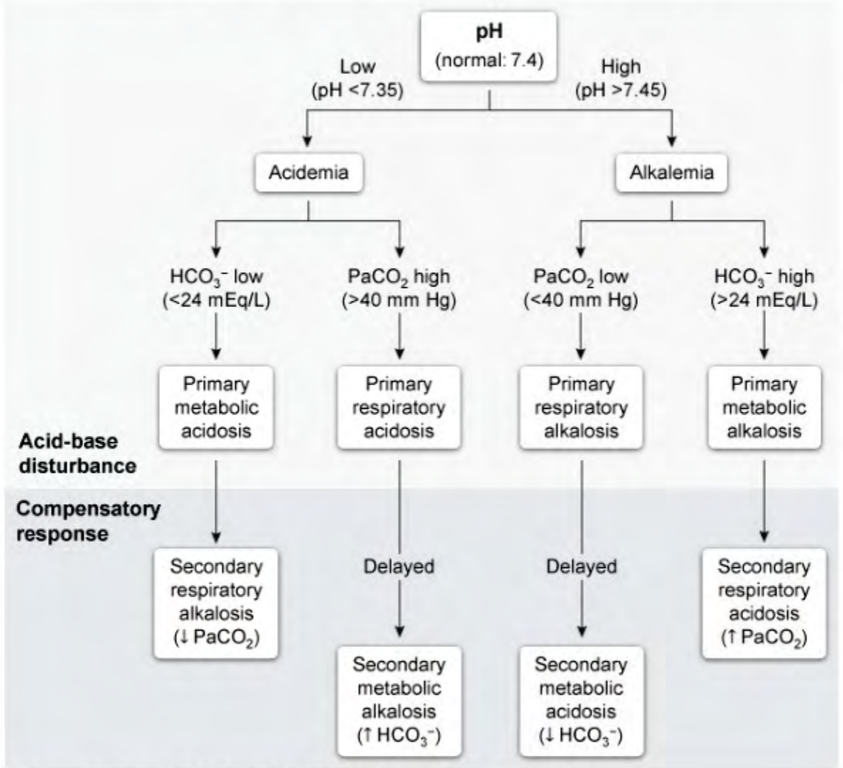
Arterial blood gas interpretation of acid-base disorders



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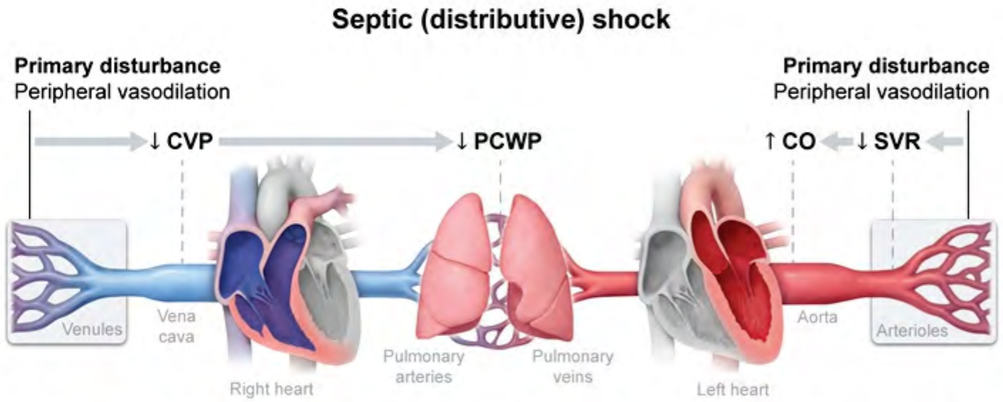

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Cardiogenic shock

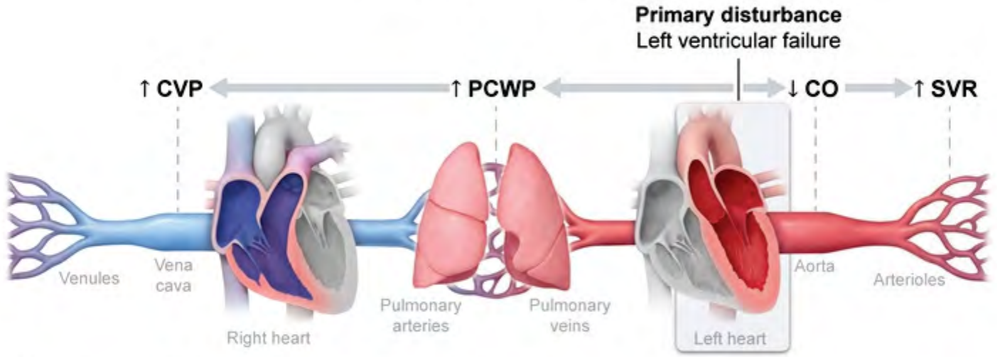

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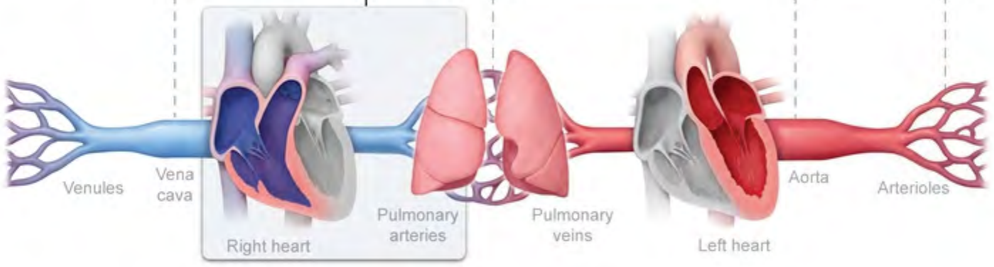
Exhibit Display

Obstructive shock

Primary disturbance
Impeded cardiopulmonary blood flow
(eg, PE, tension PTX)

↑ CVP → ↓ PCWP → ↓ CO → ↑ SVR



CO = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **PE** = pulmonary embolism; **PTX** = pneumothorax; **SVR** = systemic vascular resistance.

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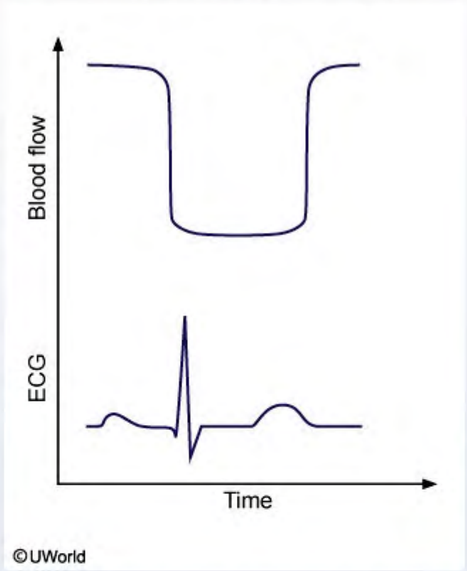
A large pharmaceutical company is conducting research on an experimental drug that affects blood flow in diseased tissue. A healthy 30-year-old man is enrolled in the trial, and patterns of normal blood flow through various tissues are studied. One of the patterns is shown in the image below:



The blood flow curve was most likely obtained from tissue of which of the following?

- A. Adrenal medulla
- B. Brain cortex
- C. Left ventricular myocardium
- D. Renal cortex
- E. Renal medulla
- F. Right ventricular myocardium
- G. Subcortical nuclei

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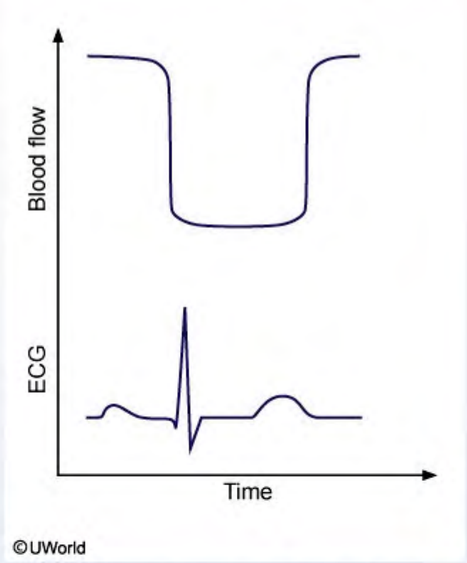
various tissues are studied. One of the patterns is shown in the image below:



The blood flow curve was most likely obtained from tissue of which of the following?

- A. Adrenal medulla (1%)
- B. Brain cortex (1%)
- ✓ C. Left ventricular myocardium (84%)
- ✗ D. Renal cortex (1%)
- E. Renal medulla (1%)
- F. Right ventricular myocardium (9%)
- G. Subcortical nuclei (0%)

Incorrect
Correct answer
C

84%
Answered correctly

08 secs
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Incorrect
Correct answer
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Answered correctly

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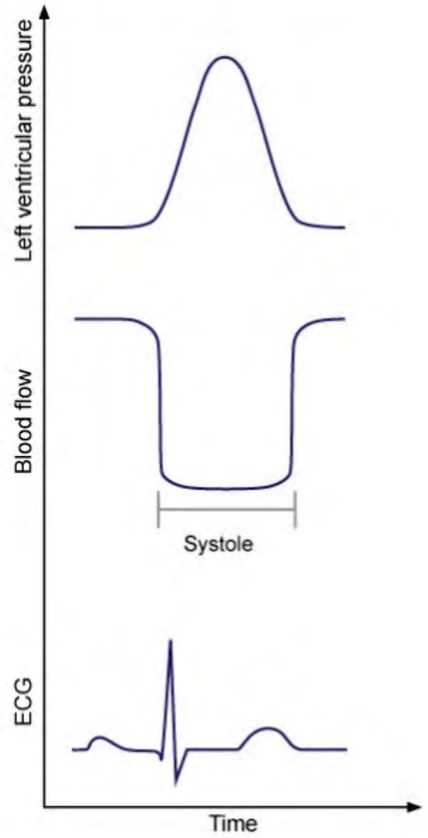

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The blood flow curve shows a cyclical variation in flow during the cardiac cycle, with **maximum flow** occurring

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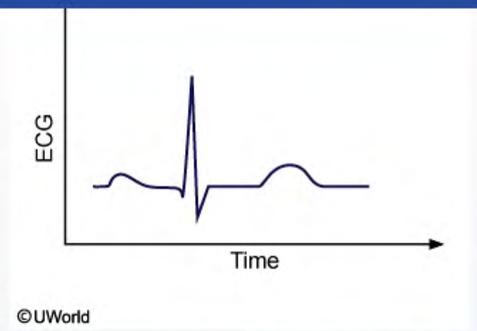

The blood flow curve shows a cyclical variation in flow during the cardiac cycle, with **maximum flow** occurring during **diastole** and minimal flow occurring during ventricular systole. This pattern is unique for the **left ventricular myocardium**. The great majority of blood flows through the vascular beds of the left ventricle during diastole, when the coronary vessels are not compressed by the high ventricular wall pressures created by myocardial contraction. This systolic reduction in blood flow is greatest in the subendocardial myocardium (where wall pressures are the highest), making it the region most prone to ischemia and myocardial infarction.

(Choices A, B, D, E, and G) In all other organs, blood flows continuously throughout the cardiac cycle down the pressure gradient from the arterial source into the venous circulation.

(Choice F) During systole, pressures in the right ventricle are much lower than in the left ventricle (only ~25 mm Hg compared to ~120 mm Hg). As a result, coronary perfusion pressure is able to overcome right ventricular wall pressure throughout the cardiac cycle, leading to relatively constant blood flow to the right ventricular myocardium.

Educational objective:
During ventricular systole, the coronary vessels supplying the left ventricle are compressed by the surrounding muscle. As a result, the majority of left ventricular blood flow occurs during diastole. The systolic reduction in coronary blood flow is greatest in the subendocardial region, making this portion of the left ventricle most prone to ischemia and infarction.

Physiology	Cardiovascular System	Coronary blood flow
Subject	System	Topic

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A 78-year-old man is brought to the emergency department due to lightheadedness that began earlier in the day. This morning, the patient felt unsteady and almost passed out when he tried to get out of bed. He has not had similar symptoms before and he has had no chest pain or shortness of breath. He has a history of coronary artery disease requiring percutaneous coronary intervention. The rhythm strip of the ECG obtained in the emergency department is shown in the exhibit. Which of the following is the most likely cause of this patient's current symptoms?

- A. Degenerative changes involving the atrioventricular node
- B. Degenerative changes involving the sinus node
- C. Ectopic electrical foci in left ventricular myocardium
- D. Reentrant circuit involving the cavotricuspid isthmus
- E. Sporadic depolarization of atrial cardiomyocytes

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A 78-year-old man is brought to the emergency department due to lightheadedness that began earlier in the day.

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- ✓ A. Degenerative changes involving the atrioventricular node (65%)
- B. Degenerative changes involving the sinus node (14%)
- C. Ectopic electrical foci in left ventricular myocardium (5%)
- D. Reentrant circuit involving the cavotricuspid isthmus (2%)
- E. Sporadic depolarization of atrial cardiomyocytes (11%)

Correct

65% Answered correctly

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Explanation

This patient's ECG demonstrates third-degree atrioventricular (AV) block. Third-degree (complete) AV block involves a total lack of communication between the atria and ventricles due to **AV node dysfunction**. It can result from ischemia, infiltrative disease (eg, sarcoidosis), infection (eg, Lyme disease), or **age-related** fibrosis with cellular **degeneration**.

Because conduction signals from the atria cannot communicate with the ventricles, the intrinsic pacemaker of the His bundle or ventricles is triggered, resulting in a junctional or ventricular escape rhythm. On ECG, there is **dissociation of P waves and QRS complexes**, with P waves marching out at the intrinsic rate of the sinoatrial node (~75/min) and QRS complexes at the intrinsic rate of the His bundle or ventricles (~45/min). The QRS-

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The slow ventricular rate in third-degree AV block leads to **reduced cardiac output**, potentially causing dyspnea, fatigue, lightheadedness, and syncope. Definitive management requires placement of a permanent pacemaker.

(Choice B) Degenerative changes involving the sinus node can lead to sick sinus syndrome, characterized by bradycardia with sinus pauses (delayed P waves) and sinus arrest (dropped P waves). However, there would be no dissociation of P waves and QRS complexes (ie, P waves are always promptly followed by a QRS complex).

(Choice C) Ectopic electrical foci in left ventricular (LV) myocardium are a common cause of monomorphic ventricular tachycardia, typically in the setting of myocardial ischemia or LV systolic dysfunction. ECG demonstrates wide QRS complexes that are rapid and regular, and P waves are not visible.

(Choice D) A reentrant circuit involving the cavotricuspid isthmus occurs in atrial flutter. ECG shows saw-toothed flutter waves; QRS complexes may demonstrate a regular or irregular rhythm depending on how consistently flutter waves are conducted through the AV node.

(Choice E) Sporadic depolarization of atrial cardiomyocytes occurs in atrial fibrillation. On ECG, QRS intervals are irregular and P waves are absent.

Educational objective:
Third-degree (complete) atrioventricular block involves dysfunction of the atrioventricular node, resulting in total lack of communication between the atria and ventricles. On ECG, there is dissociation of P waves and QRS complexes, with P waves marching out at the intrinsic rate of the sinoatrial node and QRS complexes at the

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(Choice C) Ectopic electrical foci in left ventricular (LV) myocardium are a common cause of monomorphic ventricular tachycardia, typically in the setting of myocardial ischemia or LV systolic dysfunction. ECG demonstrates wide QRS complexes that are rapid and regular, and P waves are not visible.

(Choice D) A reentrant circuit involving the cavotricuspid isthmus occurs in atrial flutter. ECG shows saw-toothed flutter waves; QRS complexes may demonstrate a regular or irregular rhythm depending on how consistently flutter waves are conducted through the AV node.

(Choice E) Sporadic depolarization of atrial cardiomyocytes occurs in atrial fibrillation. On ECG, QRS intervals are irregular and P waves are absent.

Educational objective:
Third-degree (complete) atrioventricular block involves dysfunction of the atrioventricular node, resulting in total lack of communication between the atria and ventricles. On ECG, there is dissociation of P waves and QRS complexes, with P waves marching out at the intrinsic rate of the sinoatrial node and QRS complexes at the intrinsic rate of the His bundle or ventricles (escape rhythm).

Pathology	Cardiovascular System	Heart block
Subject	System	Topic

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Third degree (complete) atrioventricular block

Pathophysiology	Key features
• Complete blockage of atrioventricular conduction • Atria & ventricles contract independently (atrioventricular dissociation)	• Ventricular rate <50 bpm with faster atrial rate • Regular P-P and R-R intervals • Complete dissociation of P waves & QRS complexes • Wide QRS complexes (junctional escape rhythm)

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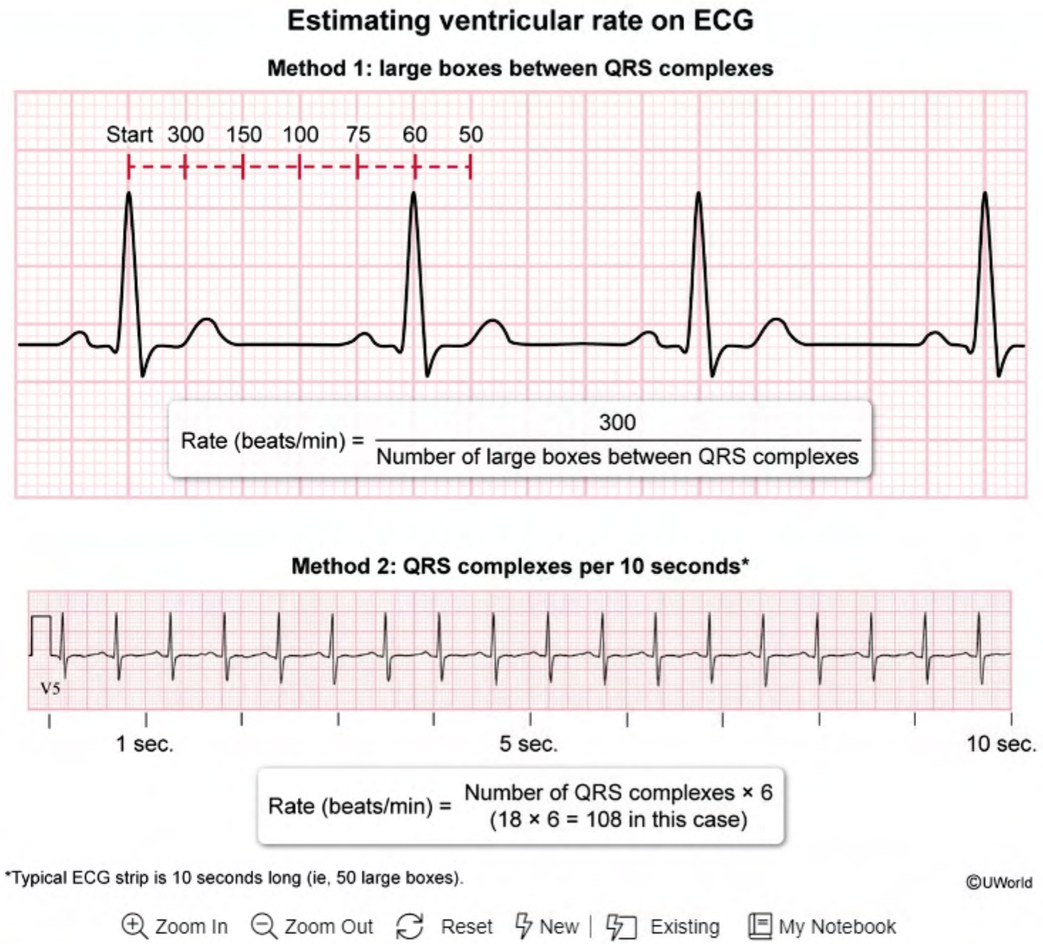

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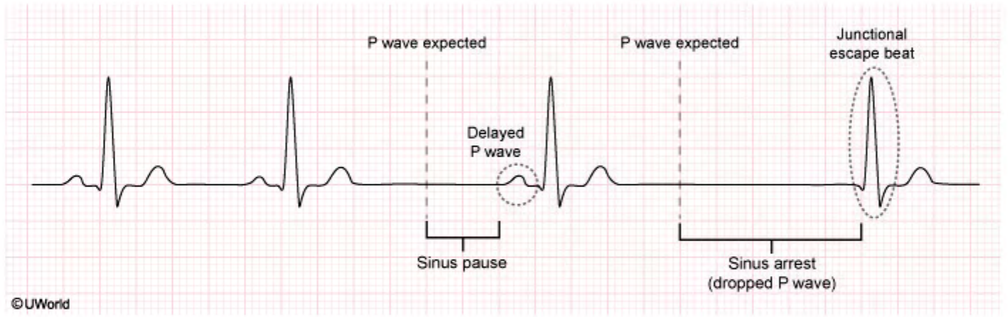

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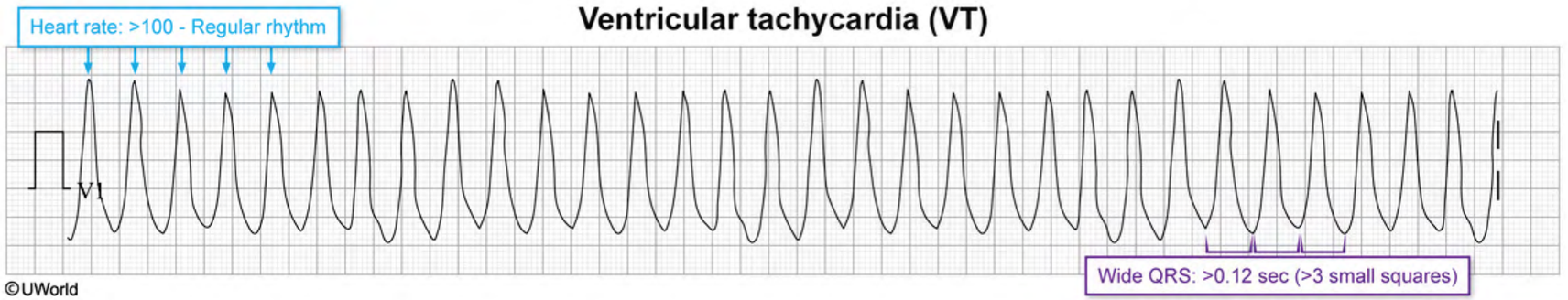

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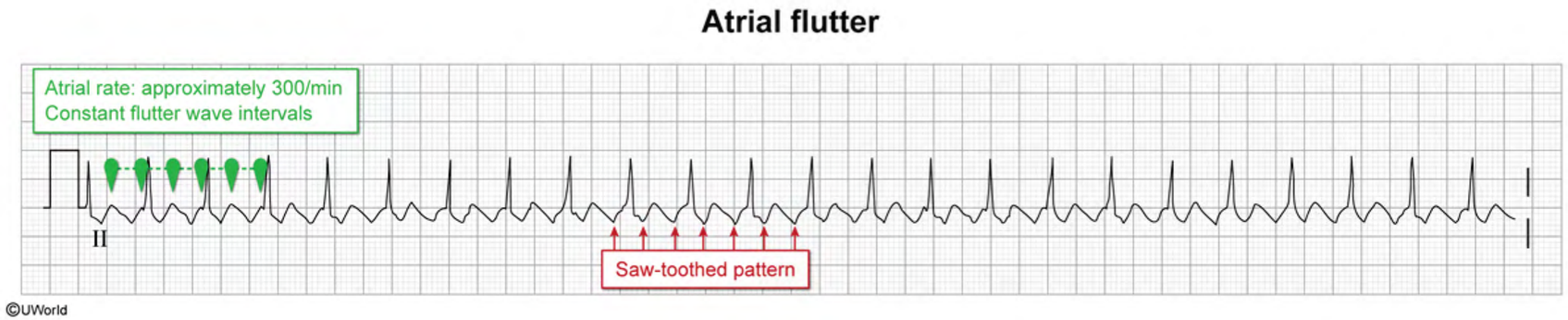

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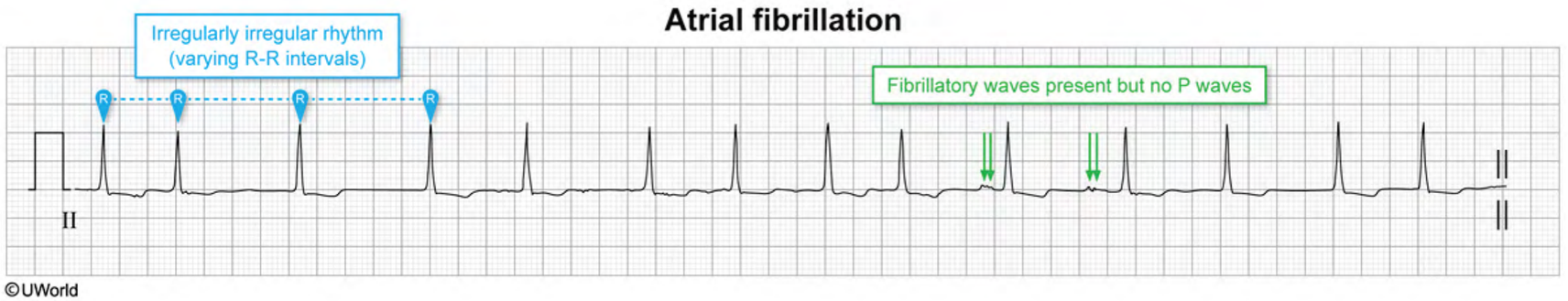

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A 68-year-old woman comes to the office due to a month-long history of worsening fatigue, shortness of breath, and dry cough. The patient has symptoms that worsen with exertion and when lying flat. She has had no chest pain, palpitations, or lightheadedness. The patient has a history of hypertension and breast cancer that is in remission after surgery and doxorubicin-based chemotherapy 15 years ago. She does not use tobacco, alcohol, or illicit drugs. Blood pressure is 110/62 mm Hg and pulse is 94/min. Physical examination shows crackles at the lung bases and bilateral pedal edema. Which of the following sets of findings are most likely present in this patient?

	Right Atrial Pressure	Left Ventricular End-Diastolic Pressure
○ A.	Decreased	Decreased
○ B.	Decreased	Increased
○ C.	Increased	Decreased
○ D.	Increased	Increased
○ E.	Increased	No change
○ F.	No change	Increased

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	Right Atrial Pressure	Left Ventricular End-Diastolic Pressure	
✗ A.	Decreased	Decreased	(5%)
B.	Decreased	Increased	(5%)
C.	Increased	Decreased	(16%)
✓ D.	Increased	Increased	(58%)
E.	Increased	No change	(7%)
F.	No change	Increased	(6%)

Incorrect
Correct answer
D

58%
Answered correctly

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Explanation

Left-sided heart failure

LV failure

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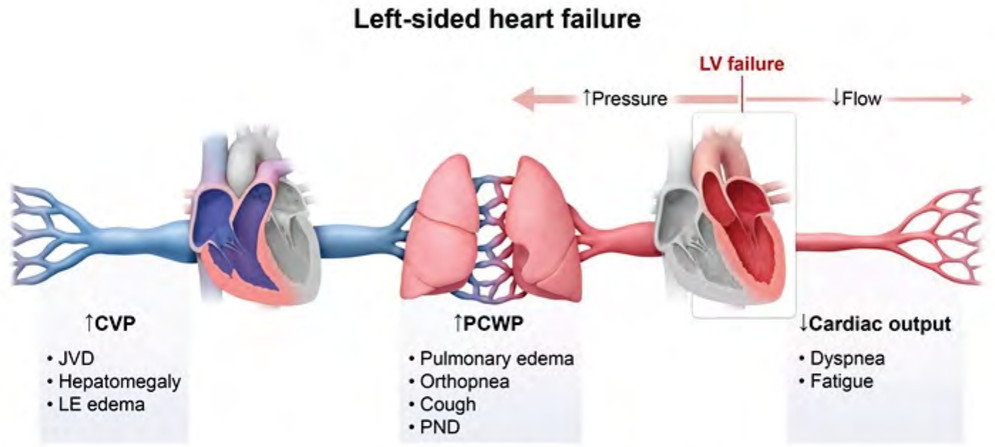

CVP = central venous pressure; **JVD** = jugular venous distension; **LE** = lower extremity; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **PND** = paroxysmal nocturnal dyspnea.

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LV failure

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CVP = central venous pressure; **JVD** = jugular venous distension; **LE** = lower extremity; **LV** = left ventricular; **PCWP** = pulmonary capillary wedge pressure; **PND** = paroxysmal nocturnal dyspnea.

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This patient with fatigue, dyspnea on exertion, orthopnea, cough, and lower extremity swelling is in **acute decompensated heart failure**. Anthracycline chemotherapeutic agents (eg, **doxorubicin**) are a common cause of dilated cardiomyopathy and can precipitate symptomatic heart failure that presents years after exposure.

Dilated cardiomyopathy results from a direct insult to cardiomyocytes that impairs their contractile function. There is an increase in left ventricular volume that is initially compensated for by the Frank-Starling mechanism and later by eccentric hypertrophy to maintain cardiac output. However, overwhelming wall stress eventually leads to marked impairment in myocardial contractile function, **reduced cardiac output**, and symptomatic decompensated heart failure. The **elevated left ventricular end-diastolic pressure** (LVEDP) is transmitted backward to the pulmonary veins and capillaries, leading to pulmonary edema (evidenced by crackles on lung auscultation).

Patients with **advanced heart failure** also develop **elevated right atrial pressure** (RAP). RAP is representative of central venous pressure and elevation is indicative of underlying volume overload and possible right-sided heart failure (which most commonly occurs secondary to left-sided heart failure due to backward transmission of elevated pressure overloading the right ventricle). In addition, since the underlying insult in dilated cardiomyopathy often directly affects the right ventricular myocardium, concomitant right ventricular failure is particularly likely in these individuals.

(Choice A) Decreased RAP and LVEDP can occur in the setting of intravascular volume depletion.

(Choices B and F) Increased LVEDP with normal or decreased RAP can occur early in the course of left-sided heart failure (eg, due to prolonged systemic hypertension). However, by the time overt signs of volume overload develop (eg, peripheral and pulmonary edema), RAP is invariably elevated.

(Choices C and E) Increased RAP with normal or decreased LVEDP suggests right-sided heart failure in the absence of left-sided heart failure, as can occur with pulmonary arterial hypertension or hypoxic lung disease (eg, interstitial lung disease). Right ventricular output is impaired, causing reduced blood flow through the lungs to the left atrium (low or normal left-sided pressures). In contrast, this patient's pulmonary crackles are indicative of

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Educational objective:
Patients with decompensated heart failure have elevated left ventricular end-diastolic pressure and decreased cardiac output that is most often primarily due to left ventricular dysfunction. Right atrial pressure (ie, central venous pressure) is also elevated in advanced heart failure due to volume overload; right-sided heart failure (most often occurring secondary to left-sided failure) can also contribute to elevated right atrial pressure.

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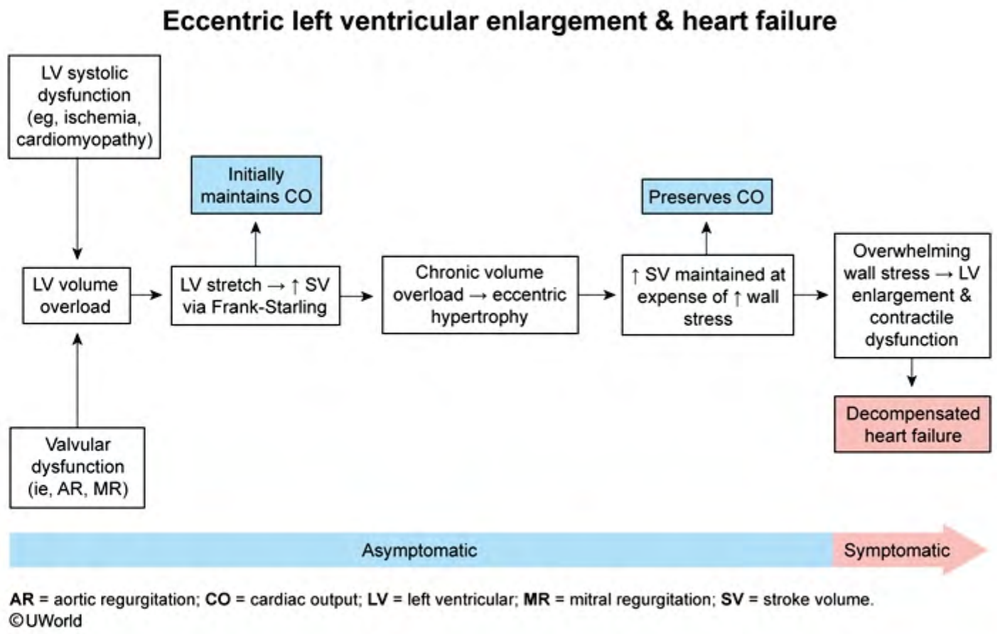

AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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Dilated cardiomyopathy results from a direct insult to cardiomyocytes that impairs their contractile function.

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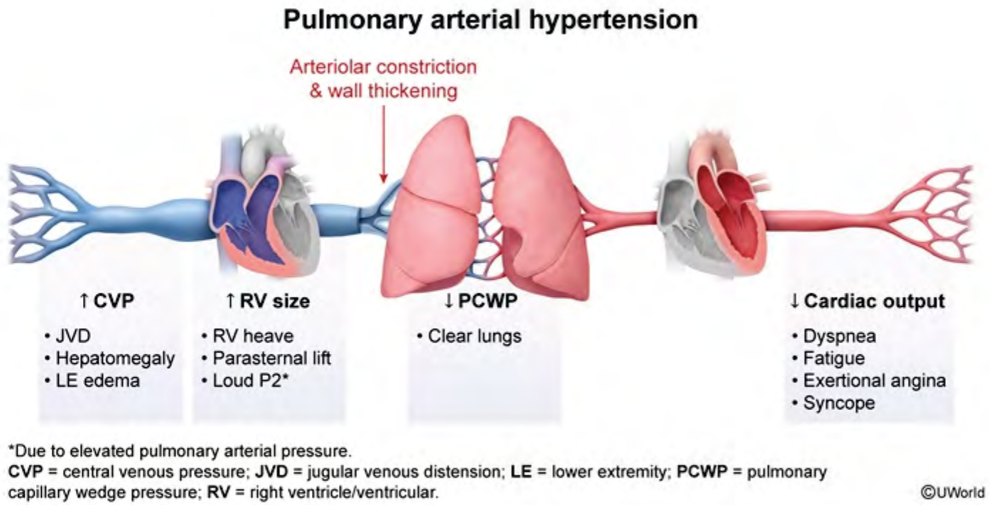

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often occurring secondary to left-sided failure) can also contribute to elevated right atrial pressure.

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A 46-year-old woman comes to the emergency department due to progressive dyspnea over the last 2 days. The patient's exercise tolerance has decreased dramatically and she had to sleep in a sitting position last night. She has no history of cardiovascular disease. The patient does not use tobacco or alcohol. Family history is unremarkable. Blood pressure is 110/65 mm Hg and pulse is 105/min and regular. The apical impulse is hyperdynamic. Cardiac auscultation reveals a diminished S1 and an apical holosystolic murmur radiating to the axilla. Diffuse pulmonary crackles are heard bilaterally. There is no peripheral edema. ECG shows sinus tachycardia but is otherwise unremarkable. Which of the following would most likely increase the ratio of forward flow volume to regurgitant flow volume in this patient?

- A. Decreasing heart rate
- B. Decreasing systemic vascular resistance
- C. Decreasing venous return to the left ventricle
- D. Increasing left ventricular contractility
- E. Increasing venous return to the left ventricle

Submit

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- ✗ A. Decreasing heart rate (4%)
- ✓ B. Decreasing systemic vascular resistance (67%)
- C. Decreasing venous return to the left ventricle (9%)
- D. Increasing left ventricular contractility (10%)
- E. Increasing venous return to the left ventricle (8%)

Incorrect
Correct answer
B

67%
Answered correctly

03 secs
Time Spent

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Version

Explanation

Effect of blood pressure reduction on mitral regurgitation

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A 46-year-old woman comes to the emergency department due to progressive dyspnea over the last 2 days. The patien
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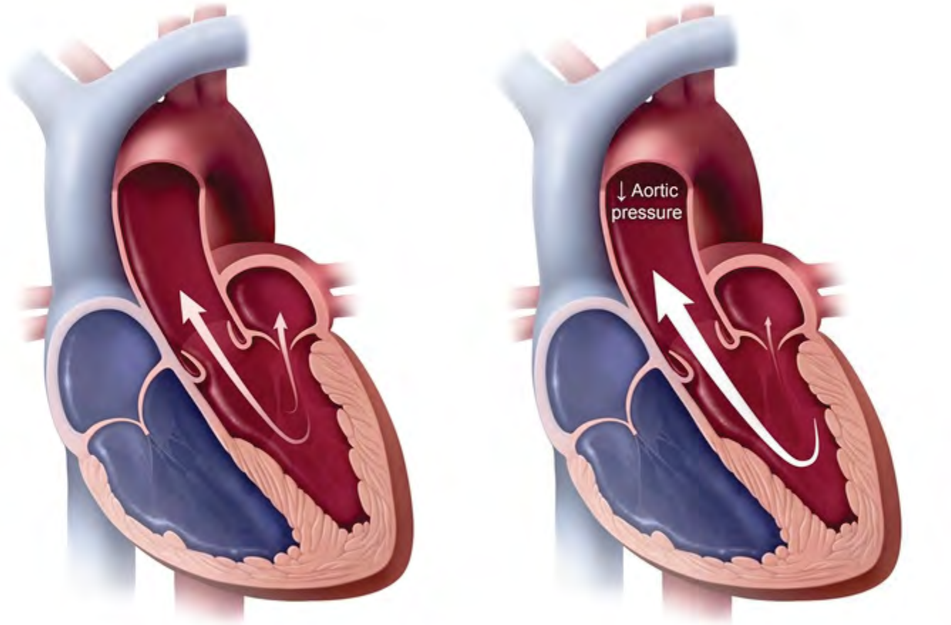
Effect of blood pressure reduction on mitral regurgitation



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This patient's dyspnea, orthopnea, and crackles on lung auscultation are consistent with decompensated left-sided heart failure; the **apical holosystolic murmur** radiating to the axilla suggests **mitral regurgitation** (MR) as a contributing factor. In patients with MR, some of the blood in the left ventricle is pumped forward through the aortic valve (forward stroke volume), and some is forced backward through the incompetent mitral valve (regurgitant stroke volume). The amount of blood flowing down each pathway is determined by the relative contribution of the resistance of each pathway to the total **left ventricular afterload**:

- Resistance to **forward flow** is primarily determined by the pressure in the aorta (systolic blood pressure)
- Resistance to **regurgitant flow** is determined by the mitral valve orifice size during systole and the degree of left atrial compliance. In chronic MR, the left atrium becomes more compliant and the lower left atrial pressures facilitate greater regurgitant flow.

Left atrial pressure remains relatively constant over the short term, but aortic pressure can vary significantly with changes in **systemic vascular resistance**. A reduction in systemic vascular resistance leads to reduced systemic blood pressure and an **increase in the ratio of forward to regurgitant blood flow**. Pharmacologic vasodilators (eg, nitroprusside) therefore help to increase forward cardiac output and reduce pulmonary congestion in patients with MR.

(Choices A and E) A decrease in heart rate leads to an increase in venous return to the left ventricle (preload) due to increased diastolic filling time. This causes an absolute increase in both forward and regurgitant blood flow (stroke volume increases in accordance with the Frank-Starling mechanism), but the ratio of forward to regurgitant flow is unlikely to significantly change.

(Choice C) A reduction in venous return to the left ventricle decreases stroke volume with absolute reductions in both forward and regurgitant flow, but is unlikely to significantly affect the ratio of forward to regurgitant flow.

(Choice D) An increase in left ventricular contractility increases stroke volume and leads to an increase in both forward and regurgitant blood flow. However again, as the relative resistance of each pathway is unchanged, the ratio of forward to regurgitant blood flow remains essentially the same.

Educational objective:

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valve (forward stroke volume), and some is forced backward through the incompetent mitral valve (regurgitant stroke volume). The amount of blood flowing down each pathway is determined by the relative contribution of the resistance of each pathway to the total **left ventricular afterload**:

- Resistance to **forward flow** is primarily determined by the pressure in the aorta (systolic blood pressure)
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Left atrial pressure remains relatively constant over the short term, but aortic pressure can vary significantly with changes in **systemic vascular resistance**. A reduction in systemic vascular resistance leads to reduced systemic blood pressure and an **increase in the ratio of forward to regurgitant blood flow**. Pharmacologic vasodilators (eg, nitroprusside) therefore help to increase forward cardiac output and reduce pulmonary congestion in patients with MR.

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Educational objective:
In patients with mitral regurgitation, left ventricular afterload is determined by the balance of resistance between forward flow (aortic pressure) and regurgitant flow (left atrial pressure). A reduction in systemic vascular resistance increases the ratio of forward to regurgitant blood flow and improves cardiac output.

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A 34-year-old woman comes to the office due to exertional dyspnea. The patient has a history of IgA nephropathy; she received hemodialysis for 2 years before undergoing kidney transplantation last month. Chest x-ray reveals cardiomegaly and pulmonary congestion. Further evaluation determines that her symptoms are likely due to persistence of the arteriovenous fistula that was used for hemodialysis. Which of the following physiologic changes are most likely present in this patient due to the fistula?

	Cardiac output	Systemic vascular resistance	Venous return
○ A.	↓	↑	↓
○ B.	↓	↑	↑
○ C.	↑	↓	↓
○ D.	↑	↓	↑
○ E.	↑	↑	↑

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		Cardiac output	Systemic vascular resistance	Venous return	
	A.	↓	↑	↓	(10%)
✗	B.	↓	↑	↑	(11%)
	C.	↑	↓	↓	(6%)
✓	D.	↑	↓	↑	(58%)
	E.	↑	↑	↑	(12%)

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Correct answer
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Answered correctly

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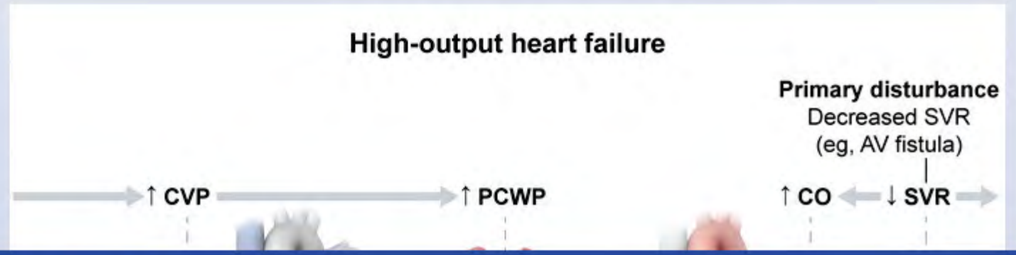
Explanation



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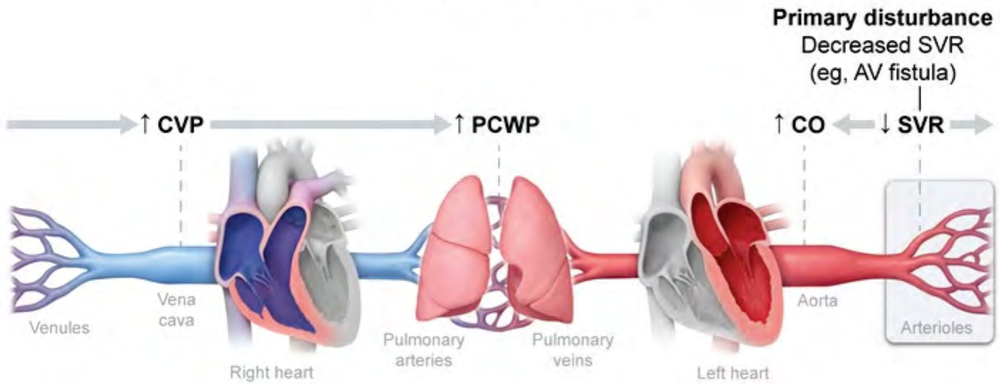
High-output heart failure



AV = arteriovenous; **CO** = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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AV = arteriovenous; **CO** = cardiac output; **CVP** = central venous pressure; **PCWP** = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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This patient with dyspnea and chest x-ray showing an enlarged heart and pulmonary edema likely has **high-output heart failure** due to excessive flow through her **arteriovenous fistula**. A surgically created arteriovenous fistula forms an enlarged vein that serves as an access point for hemodialysis (which requires a vessel with high blood flow rates); however, if the fistula becomes too large, it can lead to hemodynamic complications.

A large arteriovenous fistula allows a high proportion of blood flow to bypass the resistance of the systemic arterioles, causing markedly **decreased systemic vascular resistance** (SVR) (ie, reduced afterload). Blood passing through the low peripheral resistance returns to the right atrium quickly and easily, resulting in **increased venous return** (ie, increased preload). Both the reduced afterload and increased preload facilitate increased stroke volume (ie, **increased cardiac output**). A baroreceptor reflex-mediated increase in contractility and heart rate in response to hypotension may also contribute to increased cardiac output.

With excessively large fistulas, the left ventricle becomes unable to keep up with the increased venous return despite a persistent increase in cardiac output; left ventricular diastolic pressure gradually increases, and decompensated heart failure develops. Other common causes of high-output heart failure also involve decreased SVR as the initial disturbance.

(Choice A) Hypovolemia (eg, severe dehydration) involves a marked reduction in blood volume. Venous return is decreased, leading to reduced cardiac output and a compensatory increase in SVR.

(Choice B) Low-output heart failure (the typical cause of cardiogenic shock) involves left ventricular dysfunction as the initial disturbance, which leads to decreased cardiac output and a compensatory increase in SVR. Increased pressure is transmitted backward from the left ventricle, causing elevated central venous pressure (ie, increased venous return [preload]).

(Choice C) The pathophysiology of sepsis is similar to high-output heart failure in that reduced SVR is the initial disturbance and is followed by a compensatory increase in cardiac output. However, in sepsis the leaky vasculature reduces intravascular volume to decrease venous return, preventing volume overload of the left ventricle.

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(Choice E) Stimulation of both alpha-1 and beta-1 adrenergic receptors (eg, with norepinephrine) causes arteriolar constriction to increase SVR, venoconstriction to increase venous return, and increased heart rate and contractility to increase cardiac output.

Educational objective:
High-output heart failure results from markedly decreased systemic vascular resistance (eg, large arteriovenous fistula) that leads to increased venous return and increased cardiac output. Despite a sustained increase in cardiac output, the left ventricle is unable to keep up with the increased venous return, and decompensated heart failure develops.

References

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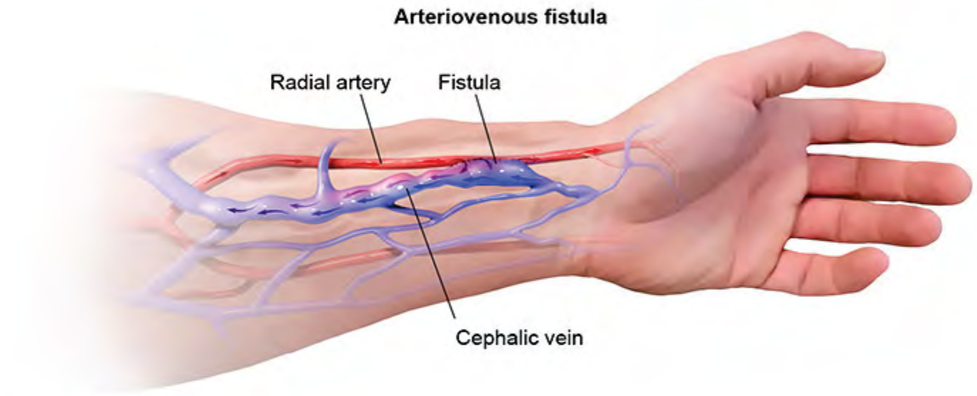

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Common mechanisms of high-output heart failure	
Increased quantity of peripheral vessels	• Morbid obesity • Paget disease
Bypass of systemic arteriolar resistance	• Acquired AV fistula (eg, trauma, HD) • Congenital AV malformation
Vasodilation due to unmet metabolic demand in tissues	• Hyperthyroidism • Severe anemia • Thiamine deficiency (wet beriberi)
AV = arteriovenous; **HD** = hemodialysis.	

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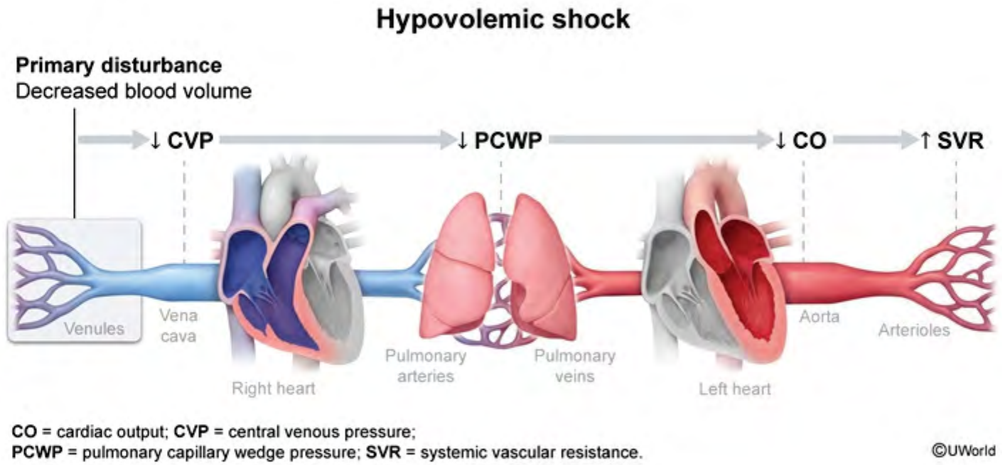

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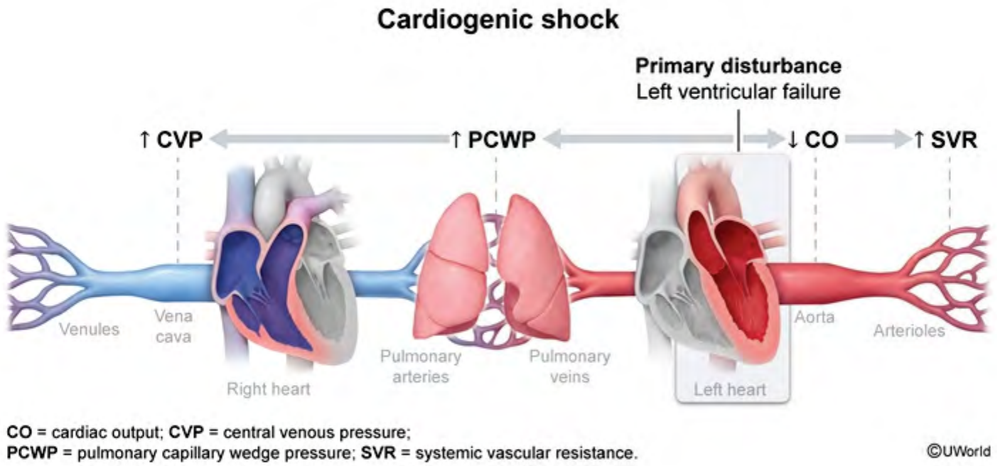

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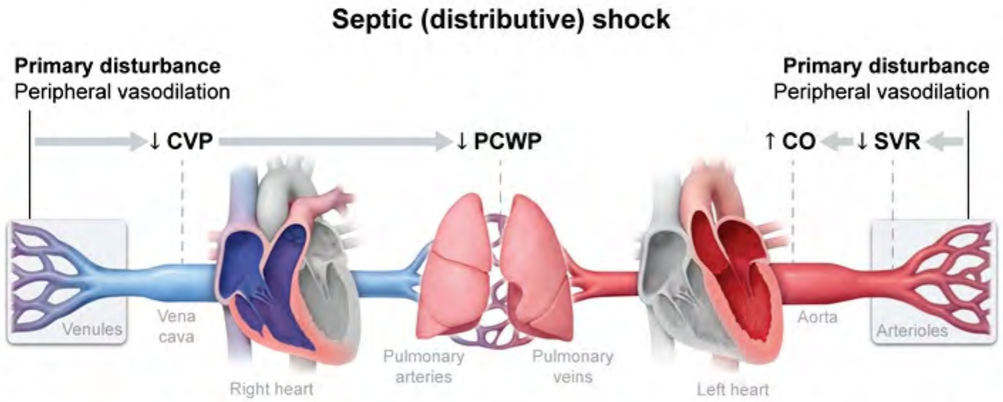

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A 49-year-old woman is brought to the emergency department with squeezing chest pain and profuse sweating for the last 2 hours. Medical history includes diet-controlled type 2 diabetes mellitus. ECG reveals ST-segment elevation in leads I, aVL, and V1-V4. The patient is immediately taken to the cardiac catheterization laboratory, where she is found to have complete occlusion of the left anterior descending coronary artery. The blockage is opened with percutaneous coronary intervention with stenting, but afterward she experiences recurrent and sustained episodes of ventricular arrhythmia. She is treated with an antiarrhythmic agent that preferentially binds to rapidly depolarizing and ischemic ventricular myocardial fibers and has minimal effect on normal ventricular myocardium. Which of the following agents was most likely used in this patient?

- A. Adenosine
- B. Digoxin
- C. Diltiazem
- D. Ibutilide
- E. Lidocaine
- F. Metoprolol
- G. Procainamide

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- ✗ A. Adenosine (9%)
- B. Digoxin (3%)
- C. Diltiazem (4%)
- D. Ibutilide (5%)
- ✓ E. Lidocaine (62%)
- F. Metoprolol (1%)
- G. Procainamide (13%)

Incorrect
Correct answer
E

62%
Answered correctly

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Explanation

This patient sustained an anterolateral ST elevation myocardial infarction (MI) with successful reperfusion following percutaneous coronary intervention. **Ventricular arrhythmias** (premature beats, ventricular tachycardia/fibrillation) are common in the first 24-48 hours after MI and may be treated with antiarrhythmic medications (eg, **amiodarone**, **lidocaine**).

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Class IB drugs like lidocaine are the weakest sodium channel blockers (**dissociate the fastest**) compared to other class I antiarrhythmics. They predominantly bind to sodium channels in the **inactivated state**, and dissociation from the channels occurs so rapidly that they have a negligible effect on QRS duration in normal cardiac tissues. Ischemic myocardium has higher than normal (less negative) resting membrane potential, which delays voltage-dependent recovery of sodium channels from the inactivated to the resting state. This allows increased binding of class IB agents; therefore, class IB antiarrhythmics are highly efficacious in inhibiting **ischemia-induced ventricular arrhythmias**.

(Choice A) Adenosine causes transient conduction delay through the atrioventricular (AV) node and is used in the acute treatment of paroxysmal supraventricular tachycardia; it is not indicated in ventricular arrhythmias.

(Choice B) Digoxin enhances vagal tone and leads to an increased effective refractory period and decreased conduction velocity through the AV node. These actions are useful in management of patients with supraventricular tachyarrhythmias.

(Choice C) Diltiazem is a class IV antiarrhythmic drug (calcium channel blocker) that slows the sinus rate, prolongs conduction through the AV node, and depresses myocardial contractility (ie, a negative inotropic effect). It is primarily used as a rate-control agent for the management of atrial tachyarrhythmias (eg, atrial flutter, atrial fibrillation).

(Choice D) Ibutilide is a class III antiarrhythmic drug (potassium channel blocker) and is occasionally used for acute termination of atrial flutter and fibrillation. It prolongs the QT interval and increases the risk of polymorphic ventricular tachycardia (torsades de pointes).

(Choice F) Metoprolol is a selective beta-1 receptor antagonist (class II antiarrhythmic) that acts as a negative inotropic and chronotropic agent (decreased myocardial contractility and heart rate). Beta blockers are frequently

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(Choice F) Metoprolol is a selective beta-1 receptor antagonist (class II antiarrhythmic) that acts as a negative inotropic and chronotropic agent (decreased myocardial contractility and heart rate). Beta blockers are frequently used in patients with acute or recent MI; they reduce the risk of ventricular arrhythmia by decreasing automaticity and reduce the size of ischemic myocardium by decreasing myocardial oxygen demand.

(Choice G) Class IA antiarrhythmic drugs (eg, procainamide, disopyramide) tend to bind to sodium channels in the open state and are more specific for suppressing arrhythmias arising from areas of normal automaticity (lidocaine is more specific to inactivated channels that are more predominant in ischemic myocardium). Class IA agents also have significant potassium channel-blocking activity, leading to prolongation of action potential and an increased risk of ventricular arrhythmias.

Educational objective:
Lidocaine is a class IB antiarrhythmic drug that tends to bind to inactivated sodium channels and rapidly dissociates. As a result, it is effective in suppressing ventricular tachyarrhythmias induced by rapidly depolarizing and ischemic myocardium.

References

- Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for

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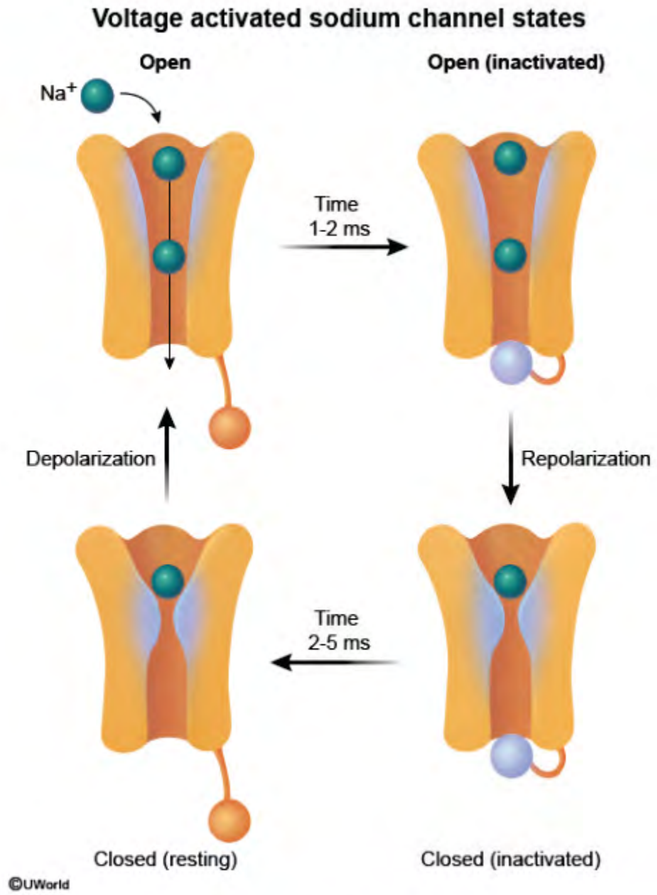

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A 44-year-old man with a history of intravenous drug use came to the emergency department due to fever and chills. Multiple blood culture sets were positive for *Staphylococcus aureus*, and the patient was diagnosed with infective endocarditis. He was successfully treated with a long course of antibiotics. Six months later, he returns to the clinic for a follow-up evaluation. The patient has no symptoms and reports good exercise tolerance. He is afebrile, blood pressure is 140/62 mm Hg, pulse is 82/min, and respirations are 16/min. Chest auscultation reveals a decrescendo diastolic murmur over the third intercostal space along the left sternal border. Echocardiogram shows severe aortic regurgitation, likely as a sequela to the prior infection. Which of the following changes is most responsible for maintaining cardiac output in the setting of this valvular abnormality?

- A. Concentric left ventricular hypertrophy
- B. Decrease in left ventricular preload
- C. Increase in aortic elasticity
- D. Increase in left ventricular afterload
- E. Increase in left ventricular stroke volume
- F. Sustained increase in heart rate

Submit

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- ✗ A. Concentric left ventricular hypertrophy (22%)
- B. Decrease in left ventricular preload (2%)
- C. Increase in aortic elasticity (1%)
- D. Increase in left ventricular afterload (4%)
- ✓ E. Increase in left ventricular stroke volume (56%)
- F. Sustained increase in heart rate (13%)

Incorrect
Correct answer
E

56%
Answered correctly

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Explanation

Pathophysiology of chronic aortic regurgitation

Chronic aortic regurgitation

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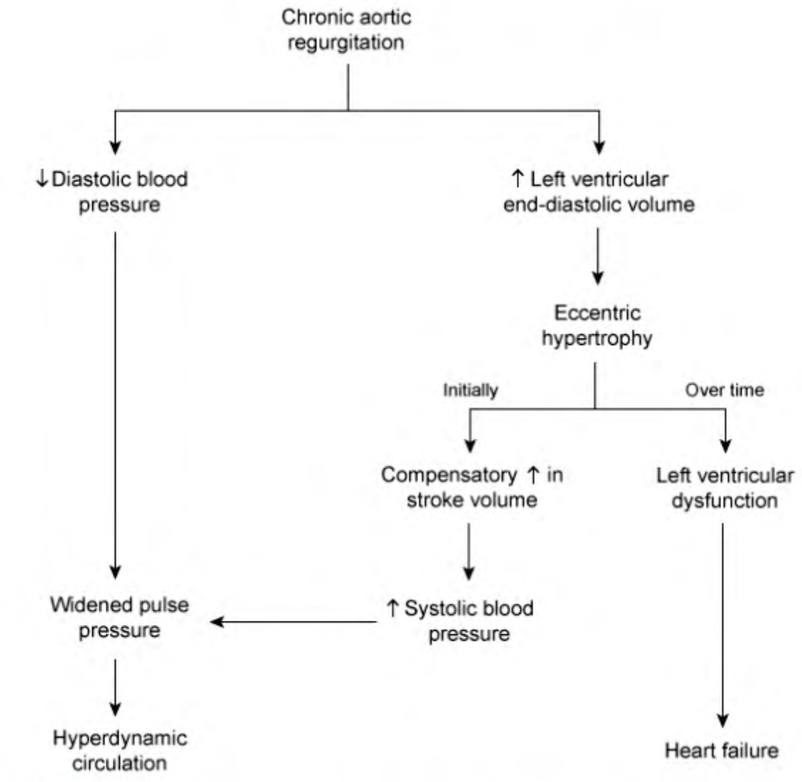
Pathophysiology of chronic aortic regurgitation



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Hyperdynamic circulation

Heart failure

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This patient has developed **chronic aortic regurgitation** (AR) as a consequence of infective endocarditis. Incomplete closure of the aortic valve during diastole allows a portion of left ventricular stroke volume to leak back into the left ventricle; in severe AR this regurgitant flow volume may be up to 50% of total forward stroke volume. Left ventricular dilation caused by the increase in left ventricular end-diastolic volume (LVEDV) (**volume overload**) triggers eccentric hypertrophy, which involves ventricular wall lengthening due to the addition of myocardial contractile fibrils organized in series. This compensatory response causes an **increase in stroke volume** that is able to maintain cardiac output, and allows for a relatively long asymptomatic period (eg, several years) in most patients with chronic AR.

However, the compensatory eccentric hypertrophy in chronic AR is overall **maladaptive**. Over time, progressive left ventricular dilation leads to overwhelming wall stress with decreased stroke volume and eventual left ventricular failure.

(Choice A) Concentric hypertrophy occurs due to conditions that create ventricular pressure overload (eg, hypertension, aortic stenosis); it involves ventricular wall thickening due to the addition of myocardial contractile fibrils in parallel. Although some concentric hypertrophy may occur in AR, eccentric hypertrophy due to volume overload is the predominant change.

(Choice B) In AR, left ventricular preload or LVEDV is increased, rather than decreased. This helps the ventricle maintain cardiac output via the Frank-Starling mechanism. Decreased preload would cause a reduction in cardiac output.

(Choice C) Increased aortic elasticity (ie, decreased compliance or increased aortic stiffening) occurs with aging and results in higher systolic blood pressure, widened pulse pressure, and reduced cardiac output.

(Choice D) In chronic AR, there is an effective increase in left ventricular afterload due to the enlarged chamber size (increases wall stress) and high peak systolic pressures. This increases left ventricular work, making it more difficult to maintain cardiac output and eventually leads to heart failure.

(Choice F) Because cardiac output is maintained by an increase in stroke volume, there is typically minimal

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(Choice B) In AR, left ventricular preload or LVEDV is increased, rather than decreased. This helps the ventricle maintain cardiac output via the Frank-Starling mechanism. Decreased preload would cause a reduction in cardiac output.

(Choice C) Increased aortic elasticity (ie, decreased compliance or increased aortic stiffening) occurs with aging and results in higher systolic blood pressure, widened pulse pressure, and reduced cardiac output.

(Choice D) In chronic AR, there is an effective increase in left ventricular afterload due to the enlarged chamber size (increases wall stress) and high peak systolic pressures. This increases left ventricular work, making it more difficult to maintain cardiac output and eventually leads to heart failure.

(Choice F) Because cardiac output is maintained by an increase in stroke volume, there is typically minimal increase in heart rate in chronic AR. However, in acute AR, eccentric hypertrophy does not have time to develop and the capacity to increase stroke volume is limited. The resulting decrease in cardiac output triggers a reflexive increase in heart rate, but this response is typically insufficient and most patients develop severe cardiogenic shock with pulmonary edema.

Educational objective:
In chronic aortic regurgitation, persistent left ventricular volume overload triggers eccentric hypertrophy, which causes a compensatory increase in stroke volume to maintain cardiac output. This compensatory mechanism allows for a relatively long asymptomatic period in most patients; however, left ventricular dysfunction eventually occurs, leading to heart failure.

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Widened pulse pressure ← ↑ Systolic blood pressure

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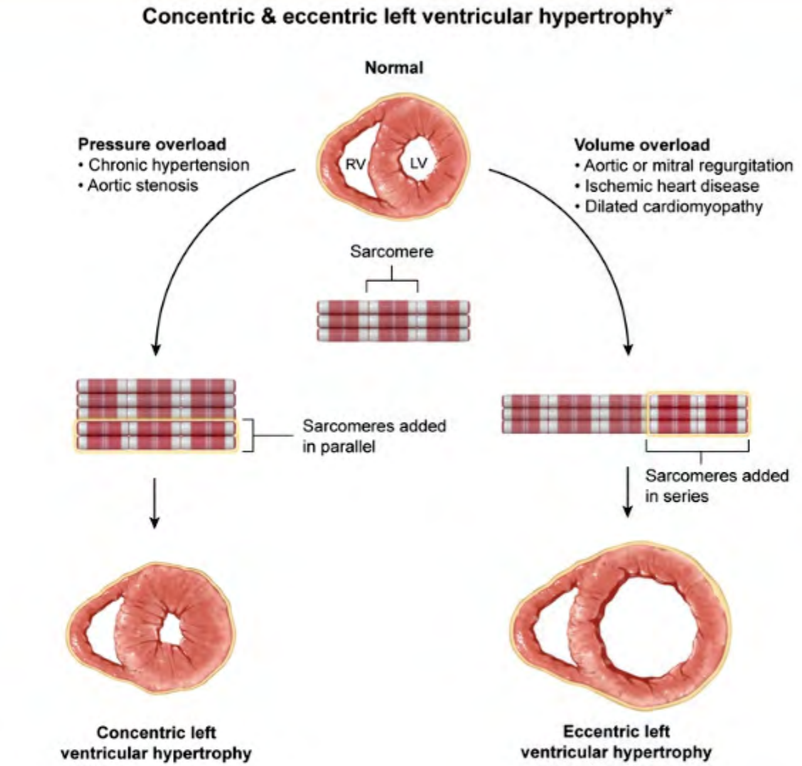

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(Choice C) Increased aortic elasticity (ie, decreased compliance or increased aortic stiffening) occurs with aging and results in higher systolic blood pressure, widened pulse pressure, and reduced cardiac output.

(Choice D) In chronic AR, there is an effective increase in left ventricular afterload due to the enlarged chamber

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A 57-year-old man is seen in the office after an episode of acute pancreatitis. Hospital evaluation found no evidence of gallstones. The patient does not consume alcohol, but he does have a history of severe hypertriglyceridemia. He was treated with a fibrate medication in the past but could not tolerate it due to liver toxicity. He has no history of diabetes mellitus or hypertension. On examination, the patient has no abdominal tenderness. Laboratory studies show normal hepatic and pancreatic enzyme levels, but the patient again has a severely elevated triglyceride level. The physician prescribes the appropriate medications and explains that the patient is likely to experience skin flushing and warmth after taking the pills. Which of the following is the primary agent mediating these side effects?

- A. Histamine
- B. Platelet-activating factor
- C. Prostaglandin
- D. Serotonin
- E. Substance P

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- ✗ A. Histamine (26%)
- B. Platelet-activating factor (1%)
- ✓ C. Prostaglandin (49%)
- D. Serotonin (17%)
- E. Substance P (5%)

Incorrect
Correct answer
C

49%
Answered correctly

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Explanation

Nicotinic acid, or **niacin**, has been used in the treatment of hyperlipidemia for almost 4 decades. It is effective in raising HDL cholesterol levels, as well as lowering triglycerides and LDL levels.

Niacin's main side effects are cutaneous **flushing**, warmth, and itching; these are primarily mediated by release of **prostaglandins** (particularly PGD_2 and PGE_2). **Aspirin**, which inhibits prostaglandin synthesis, can significantly reduce these side effects if given 30-60 minutes before niacin administration. The side effects are also reduced with slow-release preparations or if niacin is taken with meals. They are worst when niacin is first initiated and tend

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(Choice A) Vancomycin infusion reaction is mediated by histamine release due to non-IgE- mediated mast cell degranulation.

(Choice B) Platelet-activating factor is a powerful phospholipid activator produced by a number of cells, including neutrophils, basophils, platelets, and endothelial cells. It is a mediator of platelet aggregation.

(Choice D) Serotonin syndrome is a potential side effect of selective serotonin reuptake inhibitors. It is characterized by abnormal mental status, autonomic hyperactivity, and muscular rigidity with hyperreflexia.

(Choice E) Substance P is a polypeptide neurotransmitter involved in mediating pain signals in the peripheral and central nervous systems. Topical capsaicin causes release of substance P, resulting in burning pain in the area of application. Prolonged use causes depletion of substance P, and the pain diminishes with time.

Educational objective:
Niacin is used in the treatment of hyperlipidemia. It increases HDL levels and decreases LDL levels and triglycerides. Niacin causes cutaneous flushing, which is mediated by prostaglandins and can be diminished by pretreatment with aspirin.

References

- Aspirin reduces cutaneous flushing after administration of an optimized extended-release niacin formulation.
- Niacin use and cutaneous flushing: mechanisms and strategies for prevention.

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A 78-year-old woman is hospitalized due to acute myocardial infarction. The patient had been having intermittent chest pain for 3 days and came to the hospital when the pain became unremitting. She has type 2 diabetes mellitus, but her medical follow-up has been poor. On the third day of hospitalization, the patient has sudden-onset shortness of breath due to pulmonary edema. Echocardiography confirms severe mitral regurgitation. She is taken immediately to surgery, which reveals rupture of the posteromedial papillary muscle. This patient's finding typically suggests compromised blood flow through which of the following coronary arteries?

- A. Conus branch
- B. Diagonal branch
- C. Left anterior descending
- D. Obtuse marginal
- E. Posterior descending

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- ✗ A. Conus branch (1%)
- B. Diagonal branch (3%)
- C. Left anterior descending (25%)
- D. Obtuse marginal (3%)
- ✓ E. Posterior descending (67%)

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Correct answer
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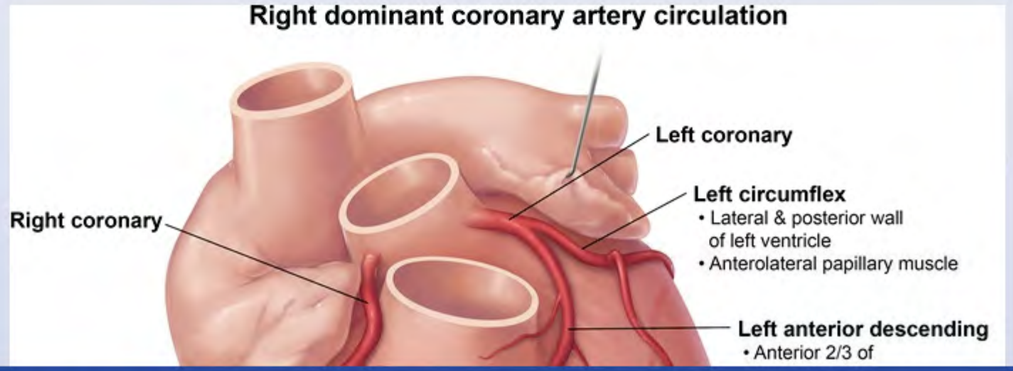
Explanation



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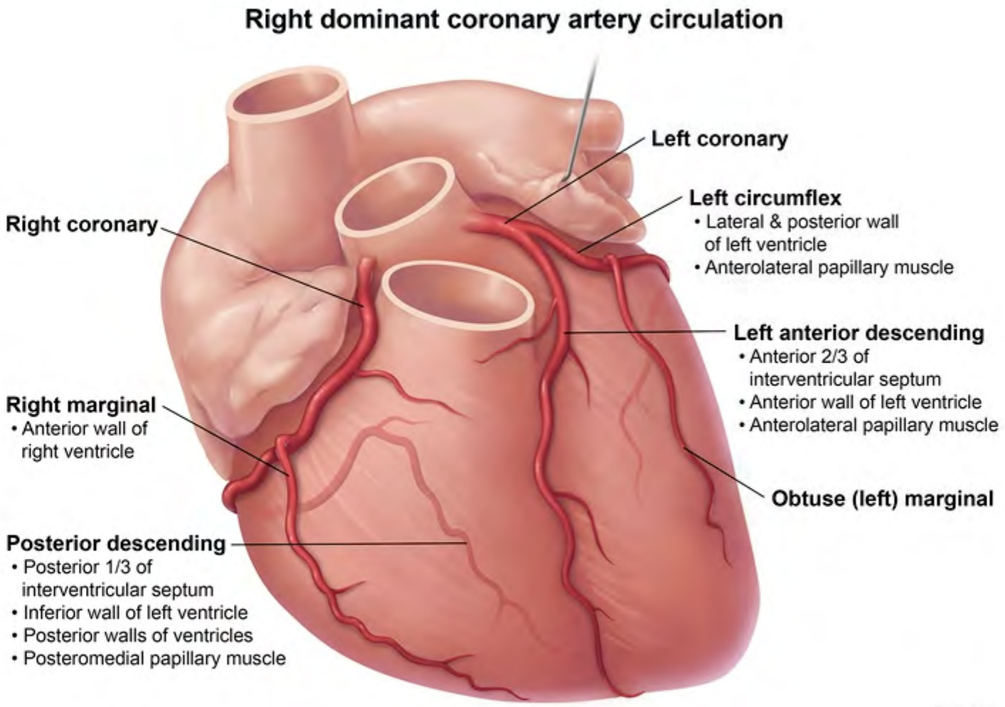

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Left anterior descending
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The mitral valve apparatus consists of the mitral valve annulus, anterior and posterior mitral leaflets, and chordae tendineae, which are tethered to the left ventricular wall via the anterolateral and posteromedial papillary muscles. Pathologic processes that disrupt any of these structures can lead to improper alignment of the mitral leaflets, resulting in **mitral regurgitation** and pulmonary edema.

The patient's mitral regurgitation is the result of **papillary muscle rupture**, a life-threatening mechanical complication typically occurring **3-5 days** following acute **myocardial infarction**. The anterolateral papillary muscle has a dual blood supply from the left anterior descending (LAD) and left circumflex arteries. In contrast, the **posteromedial** papillary muscle is supplied solely by the **posterior descending artery**, a branch of the right coronary artery (right dominant circulation) or left circumflex artery (left dominant circulation). Because blood flow to the posteromedial papillary muscle depends on a single artery, it is more likely to become ischemic and rupture following myocardial infarction.

(Choice A) The conus artery is an early branch of the proximal right coronary artery; it supplies the anterior area of the interventricular septum and conus of the pulmonary artery.

(Choice B) Diagonal arteries branch from the LAD artery and supply blood to the anterolateral walls of the left ventricle.

(Choice C) The LAD artery supplies the anterior two thirds of the interventricular septum, anterior wall of the left ventricle, and anterolateral papillary muscle. Abrupt occlusion of the LAD rarely results in ischemic rupture of the anterolateral papillary muscle as this muscle also receives blood supply from the left circumflex artery.

(Choice D) The obtuse (left) marginal artery is a branch of the left circumflex artery and supplies the lateral wall of the left ventricle.

Educational objective:
Papillary muscle rupture is a life-threatening complication that typically occurs 3-5 days after myocardial infarction and presents with acute mitral regurgitation and pulmonary edema. The posteromedial papillary muscle is supplied solely by the posterior descending artery, making it susceptible to ischemic rupture.

References

- A forgotten devil: rupture of mitral valve papillary muscle.

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interventricular septum
• Inferior wall of left ventricle
• Posterior walls of ventricles

Exhibit Display

Mechanical complications of acute myocardial infarction

Complication	Time course	Clinical findings
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR) • Hypotension/cardiogenic shock
Interventricular septum rupture	Acute or within 3-5 days	• Chest pain • New holosystolic murmur • Hypotension/cardiogenic shock • Step up in O_2 level from RA to RV
Free wall rupture**	Within 5 days or up to 2 weeks	• Chest pain • Distant heart sounds • Shock, rapid progression to cardiac arrest
Left ventricular aneurysm**	Up to several months	• Heart failure • Angina, ventricular arrhythmias

*Usually due to right coronary artery occlusion.
**Usually due to left anterior descending artery occlusion.
MR = mitral regurgitation; **RA** = right atrium; **RV** = right ventricle.

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Papillary muscle rupture is a life-threatening complication that typically occurs 3-5 days after myocardial infarction and presents with acute mitral regurgitation and pulmonary edema. The posteromedial papillary muscle is supplied

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A 32-year-old man comes to the emergency department with chest pain that started earlier in the day. The pain is midline and sharp and increases with deep breaths but decreases when the patient leans forward. He had a mild respiratory illness a week ago. Other medical history is unremarkable. The patient is a lifetime nonsmoker and has no family history of early-onset heart attack, sudden death, or cardiomyopathy. Blood pressure is 120/70 mm Hg and pulse is 110/min and regular. Which of the following physical examination findings is most expected in this patient?

- A. Friction rub
- B. Kussmaul sign
- C. Pericardial knock
- D. Pulsus paradoxus
- E. S3

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- ✓ A. Friction rub (81%)
- B. Kussmaul sign (5%)
- C. Pericardial knock (3%)
- D. Pulsus paradoxus (7%)
- E. S3 (1%)

Correct | 81% Answered correctly | 04 secs Time Spent | 2023 Version

Explanation

The chest pain of **acute pericarditis** is sharp and pleuritic and characteristically **decreases** when the patient sits and **leans forward**, which decreases pressure on the parietal pericardium. Fibrinous/serofibrinous pericarditis is the most common type of pericarditis, and a **pericardial friction rub** (described as high pitched, leathery, and scratchy) is the most specific physical finding. Fibrinous pericarditis may be caused by myocardial infarction, rheumatologic disease (eg, systemic lupus erythematosus), uremia, or viral infection. This patient's history of a recent upper respiratory illness suggests a **viral etiology**.

(Choice B) During inspiration, jugular venous pressure normally decreases as blood is pulled into the right side of the heart. Kussmaul sign is a paradoxical increase in jugular venous pressure on inspiration. It occurs because of

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The chest pain of **acute pericarditis** is sharp and pleuritic and characteristically **decreases** when the patient sits and **leans forward**, which decreases pressure on the parietal pericardium. Fibrinous/serofibrinous pericarditis is the most common type of pericarditis, and a **pericardial friction rub** (described as high pitched, leathery, and scratchy) is the most specific physical finding. Fibrinous pericarditis may be caused by myocardial infarction, rheumatologic disease (eg, systemic lupus erythematosus), uremia, or viral infection. This patient's history of a recent upper respiratory illness suggests a **viral etiology**.

(Choice B) During inspiration, jugular venous pressure normally decreases as blood is pulled into the right side of the heart. Kussmaul sign is a paradoxical increase in jugular venous pressure on inspiration. It occurs because of impaired right-sided diastolic filling in conditions such as constrictive pericarditis, restrictive cardiomyopathy, and tricuspid stenosis. Although constrictive (chronic) pericarditis can result from prolonged pericardial inflammation (over months to years), this patient's presentation is consistent with acute pericarditis, which is not associated with impaired right-sided diastolic filling.

(Choice C) A pericardial knock is a brief, high-frequency, precordial sound heard in early diastole (shortly after S2) in patients with constrictive pericarditis. It occurs earlier than S3 and may be confused with the opening snap of mitral stenosis.

(Choice D) Pulsus paradoxus is a drop in systolic blood pressure >10 mm Hg during inspiration. It represents an exaggeration of the normal reduction in systolic blood pressure during inspiration. It can be seen in cardiac tamponade, constrictive pericarditis, and severe asthma or chronic obstructive pulmonary disease.

(Choice E) An S3 typically results from reverberation of the ventricular walls during rapid filling of an overfilled ventricle. It is usually associated with heart failure as occurs with dilated cardiomyopathy, severe mitral regurgitation, or severe aortic regurgitation. A right-sided S3 can occur with right-sided heart failure.

Educational objective:
Acute-onset, sharp, and pleuritic chest pain that decreases with leaning forward is characteristic of acute pericarditis. Fibrinous/serofibrinous pericarditis is the most common form of pericarditis and a pericardial friction rub is the most specific physical finding. Viral pericarditis is often preceded by an upper respiratory infection.

References

- Acute pericarditis

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References

- Acute pericarditis

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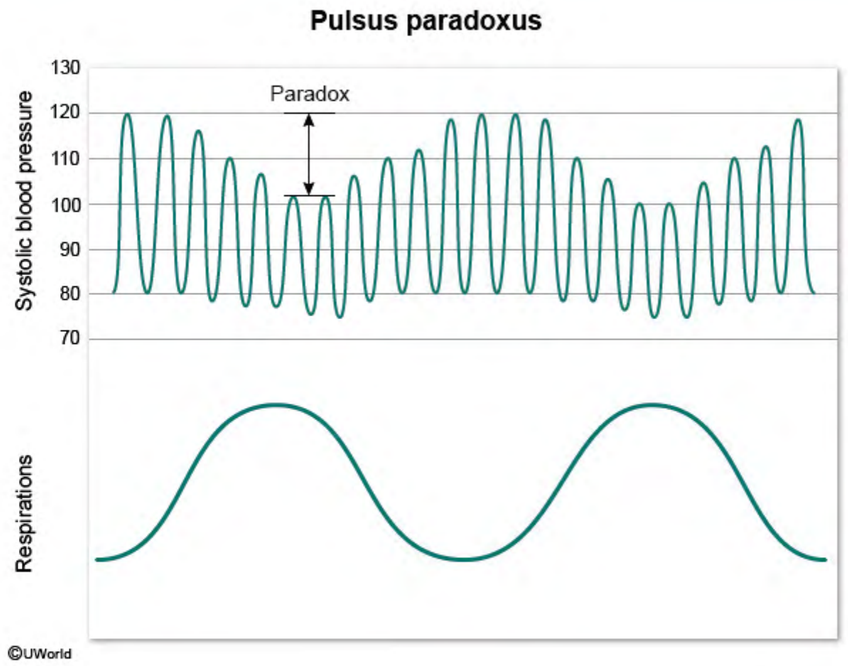

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A 4-year-old boy is brought to the office for routine evaluation of a heart murmur. The patient has no fatigue, shortness of breath, or exercise intolerance. He has no other medical conditions and has had no surgeries. Vital signs are within normal limits. On examination, the patient has no cyanosis or clubbing of the fingers. There is a harsh, 3/6 holosystolic murmur best heard at the left lower sternal border. There are no rubs or gallops. Pulses are 2+ in the upper and lower extremities. Compared to an unaffected child, which of the following changes in oxygen saturation would be expected in this patient?

	Right atrium	Right ventricle	Left atrium	Left ventricle	Systemic circulation
○ A.	Decreased	Increased	Decreased	Decreased	Decreased
○ B.	Increased	Increased	Decreased	Decreased	Decreased
○ C.	Increased	Increased	Normal	Normal	Normal
○ D.	Normal	Increased	Normal	Normal	Normal
○ E.	Normal	Normal	Normal	Decreased	Decreased

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	Right atrium	Right ventricle	Left atrium	Left ventricle	Systemic circulation	
✗ A.	Decreased	Increased	Decreased	Decreased	Decreased	(6%)
B.	Increased	Increased	Decreased	Decreased	Decreased	(12%)
C.	Increased	Increased	Normal	Normal	Normal	(12%)
✓ D.	Normal	Increased	Normal	Normal	Normal	(58%)
E.	Normal	Normal	Normal	Decreased	Decreased	(9%)

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Correct answer
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Answered correctly

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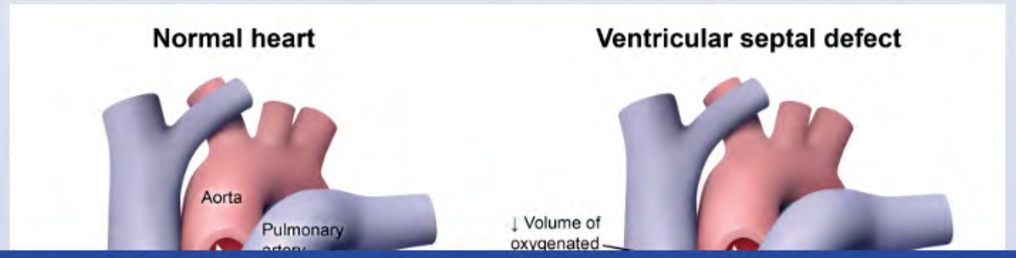
Explanation



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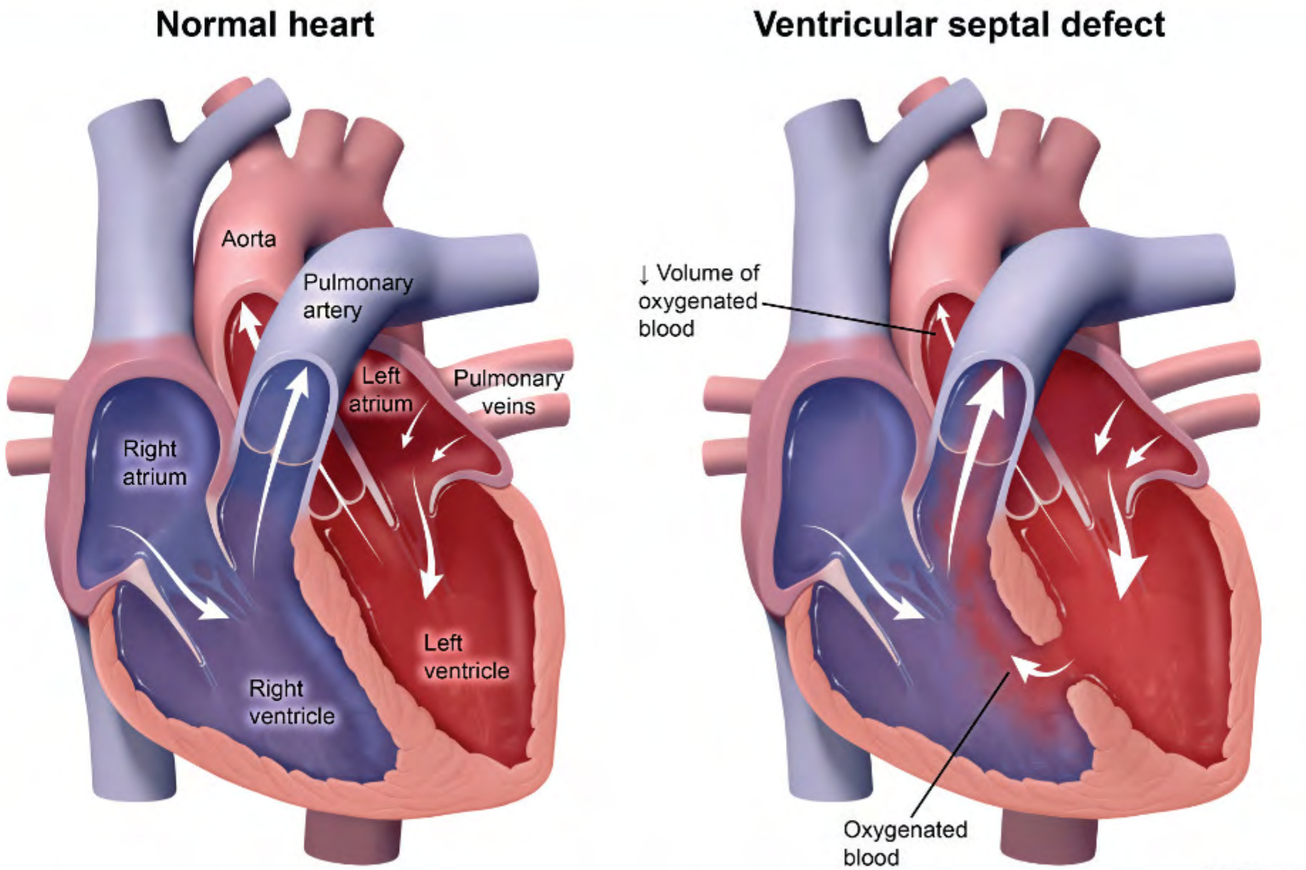

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This patient with a harsh, **holosystolic murmur** along the **lower left sternal border** most likely has a **ventricular septal defect** (VSD), an abnormal connection between the right ventricle (RV) and the left ventricle (LV). Deoxygenated blood is normally delivered to the right atrium (RA) from the systemic circulation, and oxygenated blood is delivered to the left atrium (LA) via the pulmonary circulation. Because of increased pressure on the left side of the heart compared to the right, a VSD allows for **left-to-right shunting** of blood.

The delivery of **oxygenated blood to the RV** via a VSD results in **increased RV oxygen saturation** (SaO_2) compared to that of a patient with an anatomically normal heart. The SaO_2 in the other cardiac chambers and systemic circulation remains unchanged, and patients are often **asymptomatic** (eg, no shortness of breath, cyanosis, or clubbing).

(Choice A) In tricuspid atresia (absent communication between the RA and the RV), an atrial septal defect (ASD) is always present, allowing deoxygenated blood to flow from the RA to the LA. This decreases SaO_2 in the LA, LV, and systemic circulation, causing neonatal cyanosis. The little blood that reaches the lungs usually does so through a VSD, causing a relative increase in RV SaO_2.

(Choice B) The pattern of increased right-sided and decreased left-sided SaO_2 is seen in total anomalous pulmonary venous return, a condition in which the pulmonary veins (ie, oxygenated blood) drain into the RA rather than the LA.

(Choice C) An ASD typically causes left-to-right shunting of oxygenated blood between the atria, increasing SaO_2 in both the RA and RV. In contrast to a VSD, an ASD typically causes a systolic ejection murmur at the left upper sternal border (pulmonic flow murmur).

(Choice E) Over time, increased pulmonary blood flow from left-to-right shunting via a VSD can reverse to right-to-left shunting (ie, Eisenmenger syndrome). SaO_2 on the right side will be normal, whereas that in the LV and systemic circulation is decreased because of mixing with deoxygenated blood from the RV. However, Eisenmenger syndrome takes years to develop and typically presents with signs of right heart failure and cyanosis in adolescents/adults.

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side of the heart compared to the right, a VSD allows for **left-to-right shunting** of blood.

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Educational objective:
A ventricular septal defect, classically characterized by a harsh, holosystolic murmur at the left lower sternal border, causes left-to-right shunting of oxygenated blood from the left ventricle to the right ventricle. Therefore, right ventricular oxygen saturation is increased compared to normal.

References

- Ventricular septal defect.

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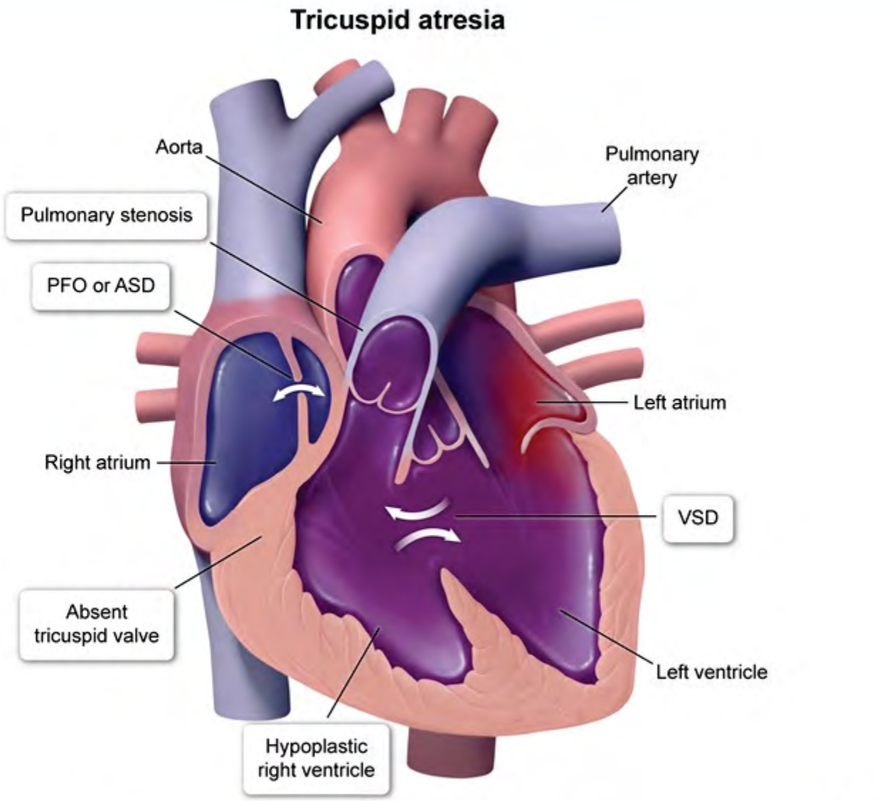

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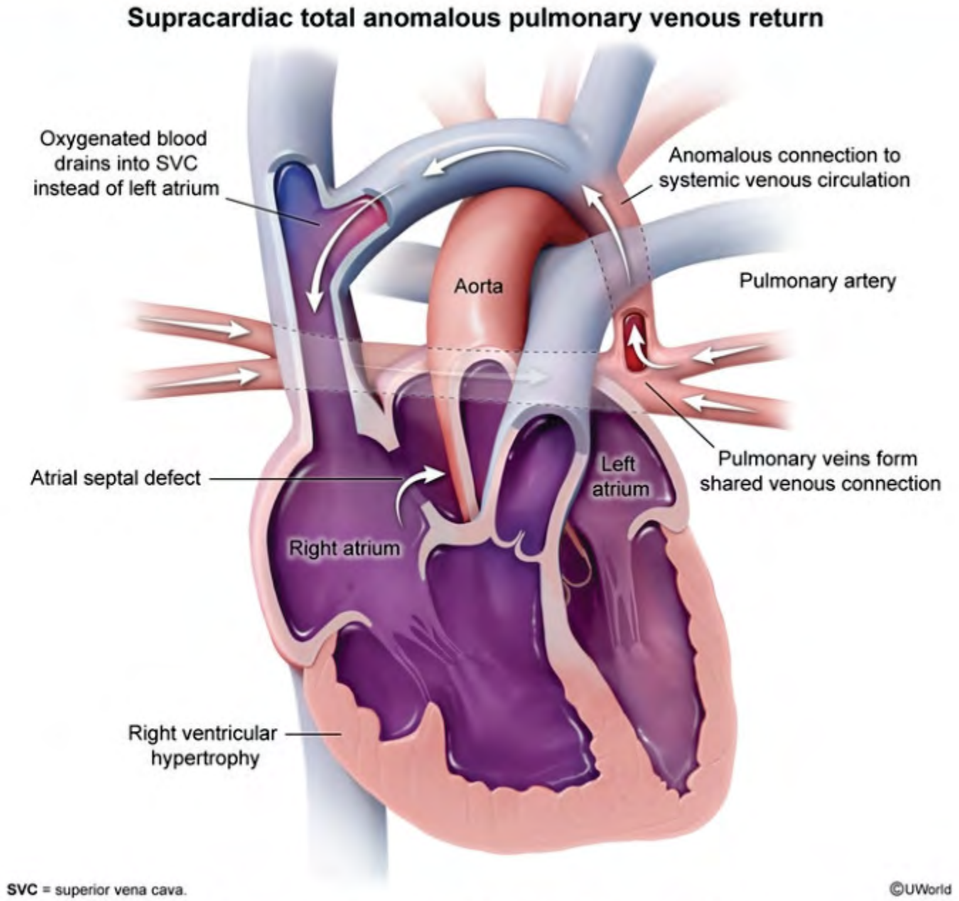

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Oxygenated blood

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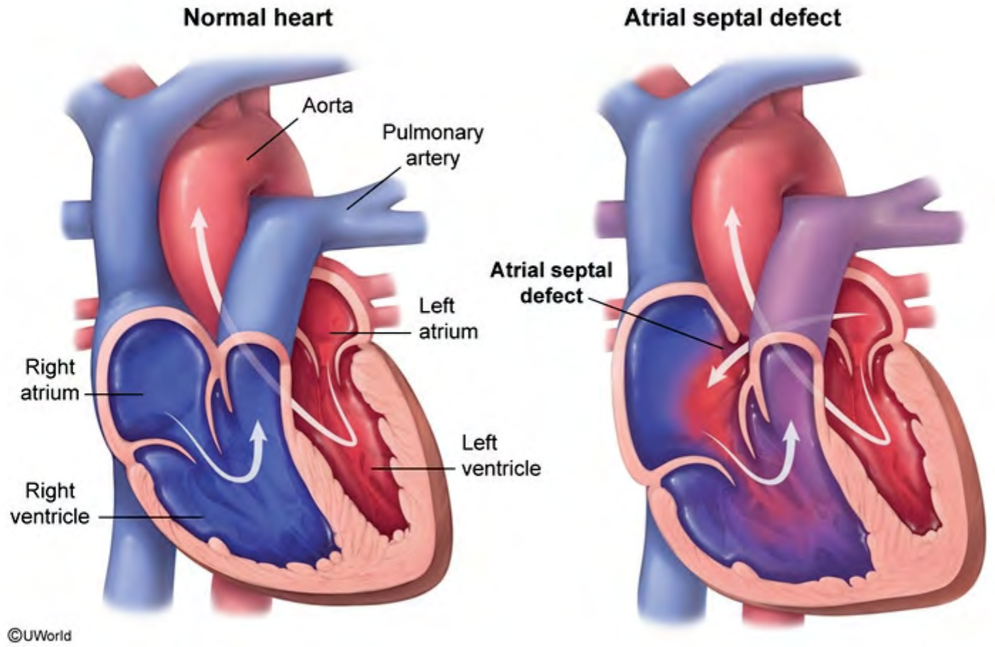

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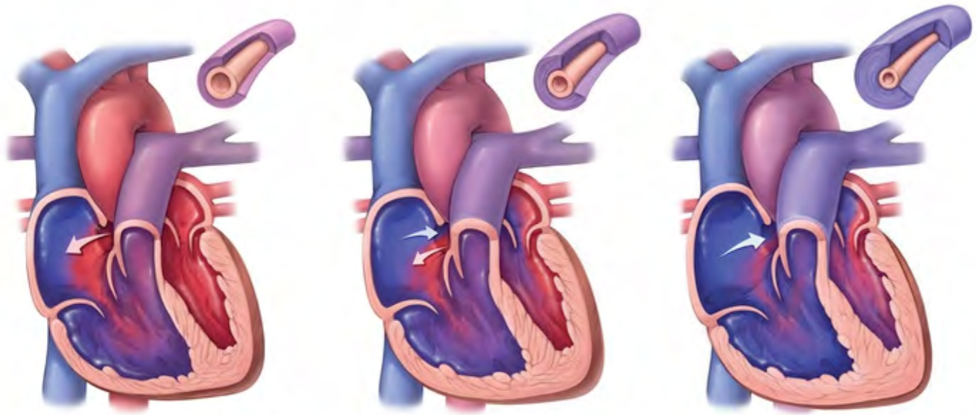
Eisenmenger syndrome



Congenital defect (eg, ASD) allows left-to-right shunt

Over time, ↑ PVR leads to bidirectional flow

PVR continues to increase with eventual continuous right-to-left shunt (Eisenmenger syndrome)

ASD = atrial septal defect; **PVR** = pulmonary vascular resistance.

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A 47-year-old man with known coronary artery disease comes to the emergency department with chest tightness, sweating, and palpitations. His symptoms began abruptly 2 hours ago while he was at work. Six months ago, the patient underwent percutaneous angioplasty of the right coronary artery. Past medical history also includes anxiety and panic attacks. Blood pressure is 180/90 mm Hg and pulse is 110/min with a regular rhythm. An esmolol infusion produces rapid symptom relief and heart rate slowing. Which of the following portions of the ECG will be affected most significantly by the medication?

- A. A
- B. B
- C. C
- D. D
- E. E

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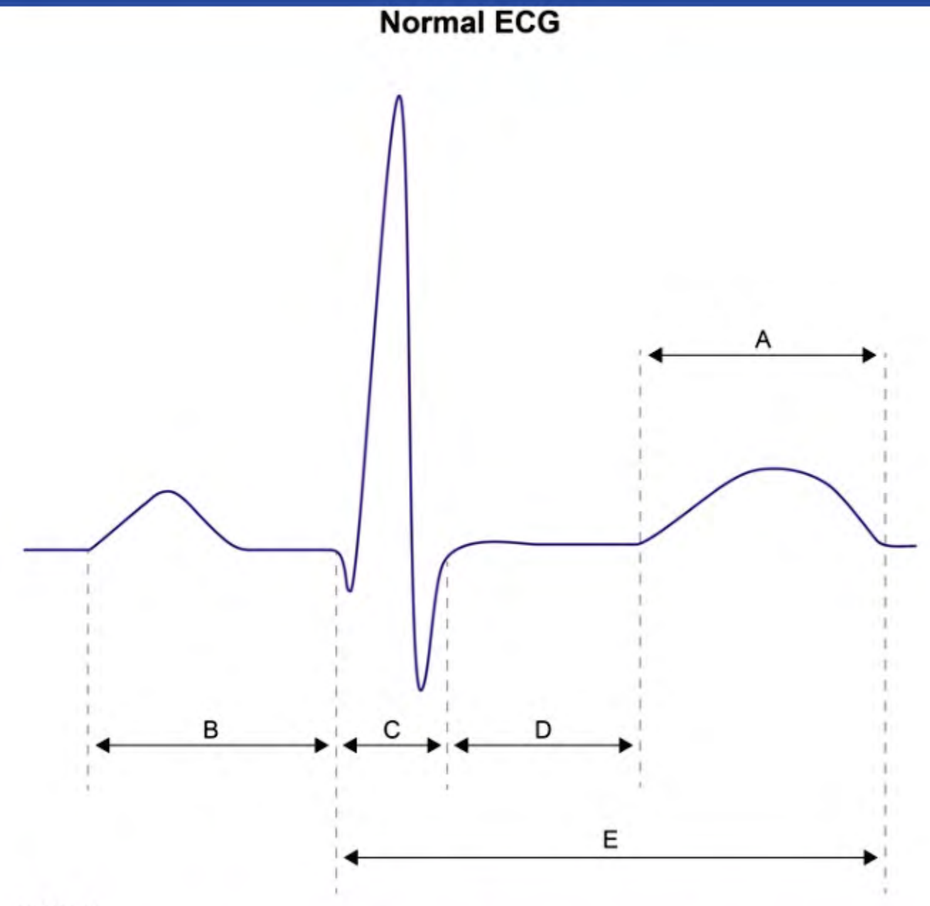

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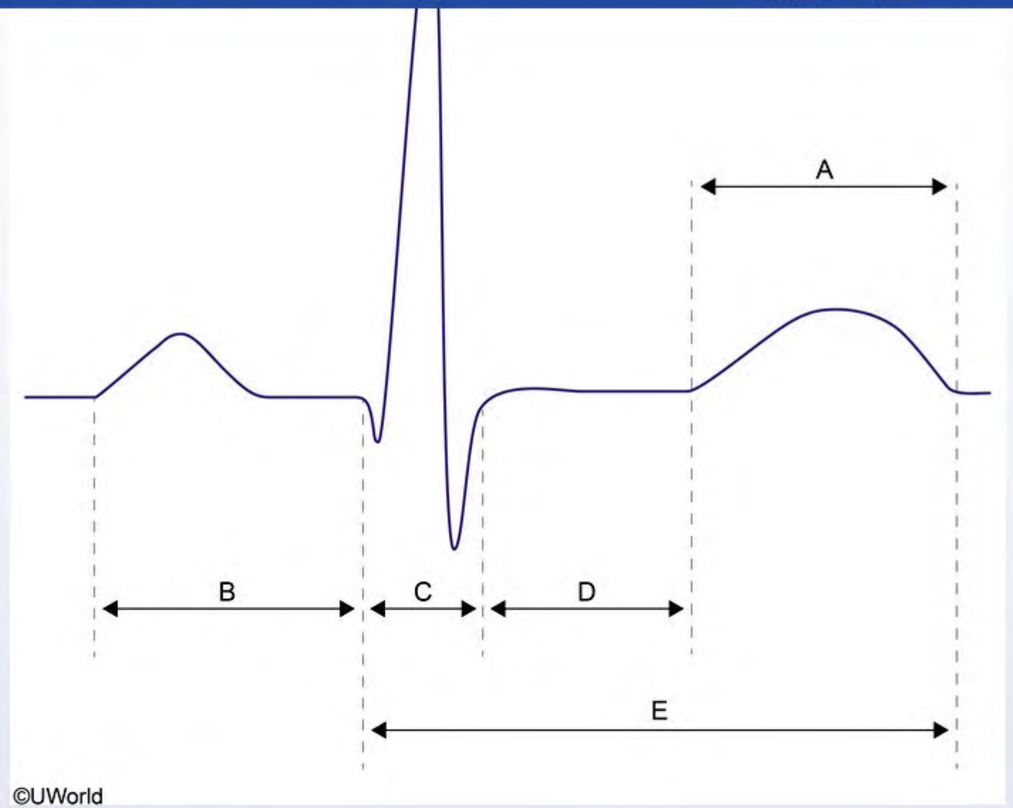

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- A. A
- B. B
- C. C
- D. D
- E. E

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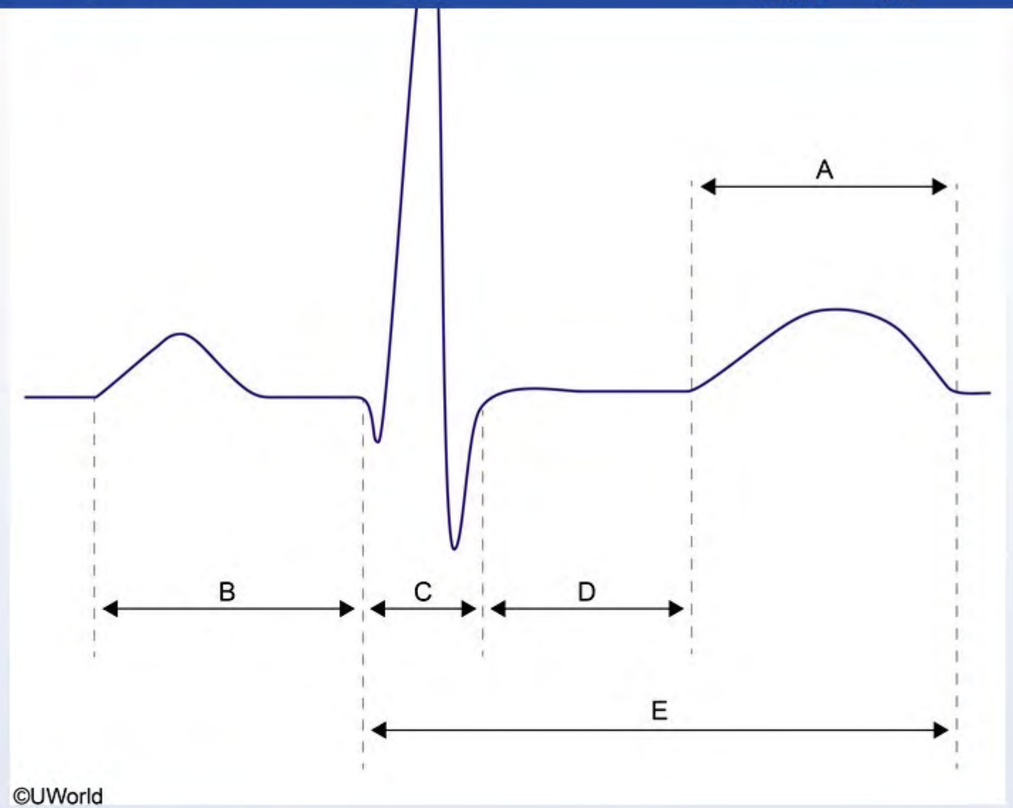

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- A. A (4%)
- ✓ B. B (63%)
- ✗ C. C (15%)
- D. D (4%)
- E. E (12%)

Incorrect
Correct answer
B

63%
Answered correctly

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Correct answer
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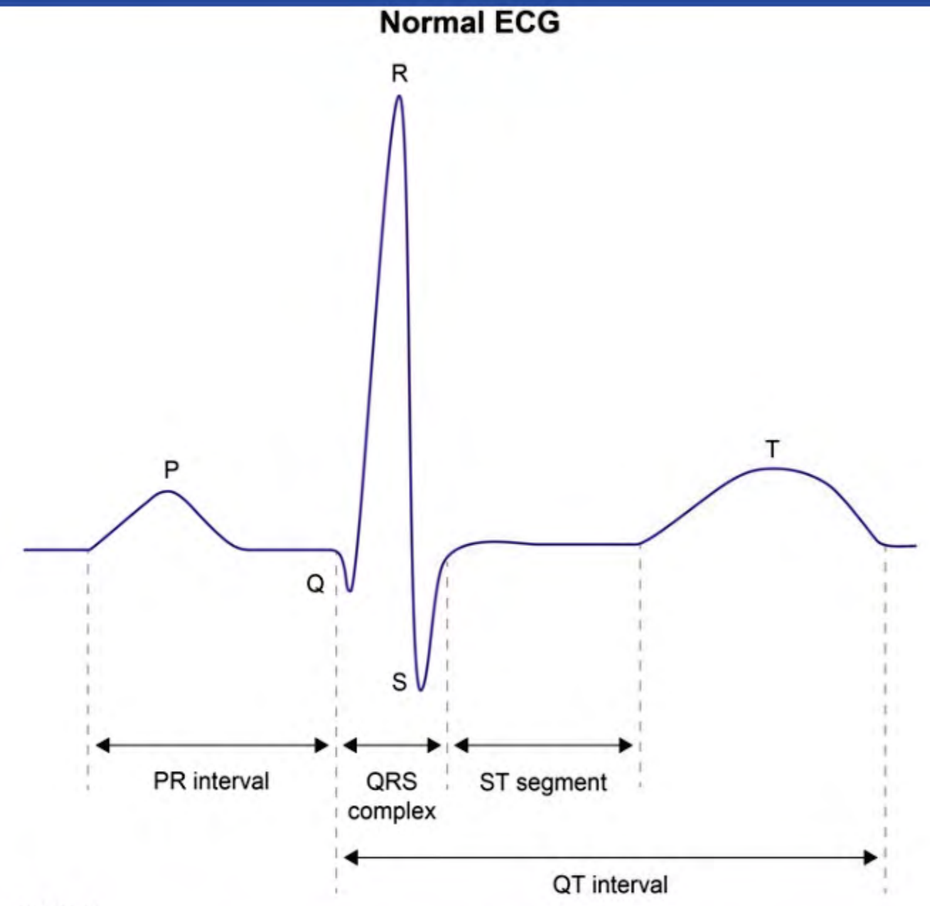

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QT interval

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On an ECG, the P wave represents atrial depolarization and the QRS complex represents ventricular depolarization. The **PR interval** is the period from the beginning of atrial depolarization (**start of P wave**) to the beginning of ventricular depolarization (**start of QRS complex**). The longer the PR interval, the longer it takes for an electrical stimulus to travel from the SA node to the ventricles via the AV node, bundle of His, and fascicular branches. Drugs that slow AV nodal conduction, such as **beta blockers**, will **prolong** the PR interval. Beta blockers (except sotalol, which also has class III properties) do not have any specific effects on QRS or QT interval durations.

(Choice A) The T wave represents ventricular repolarization. It is not affected by beta-adrenergic blockers. Myocardial ischemia can cause T wave inversion.

(Choice C) The QRS complex represents ventricular depolarization. It is not affected by beta-adrenergic blockers. A widened QRS complex can occur in the setting of ventricular automaticity, left or right bundle branch blocks, or ventricular pacemakers.

(Choice D) The ST-segment is a period of electrocardiographic inactivity that extends between ventricular depolarization and repolarization. It is not affected by beta-adrenergic blockers. ST-segment elevation or depression is usually a sign of myocardial ischemia.

(Choice E) The QT interval represents the time needed for ventricular depolarization and repolarization (ventricular electrical systole). Long QT syndrome can be congenital or acquired. Antiarrhythmics such as quinidine and sotalol are most frequently associated with acquired long QT. However, most beta blockers have a minimal effect on the resting QT interval.

Educational objective:
Beta blockers decrease AV nodal conduction, leading to an increased AV nodal refractory period. This correlates to PR interval prolongation on an ECG.

Pharmacology	Cardiovascular System	Antiarrhythmic drugs
Subject	System	Topic

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A 25-year-old man comes to the emergency department due to chest heaviness and palpitations for the last 3 hours. Over the past year, he has had similar episodes that resolved with deep breathing, but none lasted as long as this one. The patient has no chronic medical conditions and takes no medications. He does not use tobacco, alcohol, caffeine, or illicit drugs. Blood pressure is 100/70 mm Hg, and pulse is 160/min and regular. The neck is supple, the thyroid is normal, and there is no lymphadenopathy. The lungs are clear to auscultation. ECG is shown in the exhibit. Which of the following is the most likely cause of this patient's symptoms?

- A. Abnormal conduction in areas of ischemic ventricular scarring
- B. Abnormal conduction through the atrioventricular node
- C. Atrial remodeling with ectopic foci in the pulmonary vein ostia
- D. Degeneration of the sinoatrial node
- E. Delayed repolarization of ventricular cardiomyocytes

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A 25-year-old man comes to the emergency department due to chest heaviness and palpitations for the last 3 hours. as this alcoho supple shown

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- A. Abnormal conduction in areas of ischemic ventricular scarring (5%)
- ✓ B. Abnormal conduction through the atrioventricular node (60%)
- ✗ C. Atrial remodeling with ectopic foci in the pulmonary vein ostia (19%)
- D. Degeneration of the sinoatrial node (7%)
- E. Delayed repolarization of ventricular cardiomyocytes (6%)

Incorrect
Correct answer
B

60%
Answered correctly

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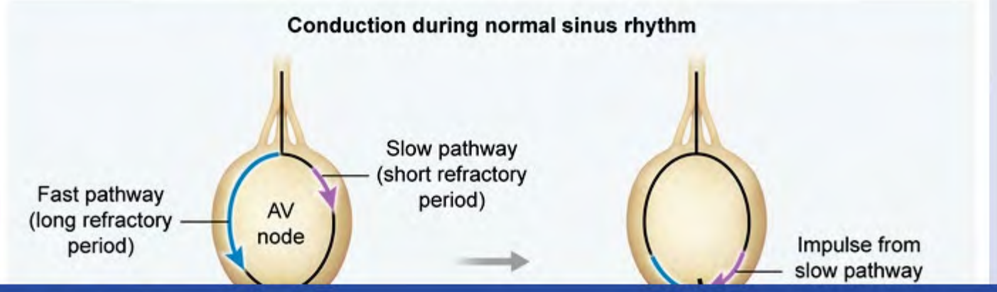
Explanation

Atrioventricular nodal reentrant tachycardia (AVNRT)



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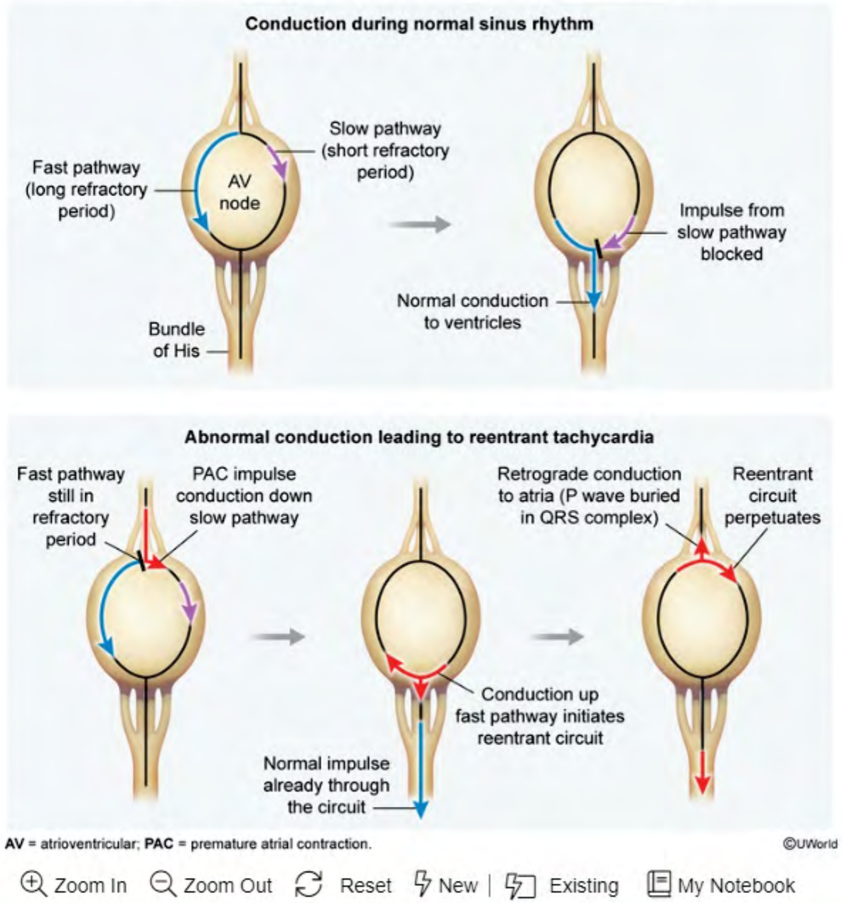
× C. Atrial remodeling with ectopic foci in the pulmonary vein ostia (19%)

Exhibit Display

Atrioventricular nodal reentrant tachycardia (AVNRT)



AV = atrioventricular; **PAC** = premature atrial contraction.

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AV = atrioventricular; PAC = premature atrial contraction. ©UWorld

This patient's ECG reveals a **narrow QRS complex tachycardia** with a **regular rhythm** and nonvisible P waves. These findings are consistent with **paroxysmal supraventricular tachycardia** (PSVT), an episodic cardiac arrhythmia that originates at or above the atrioventricular (AV) node. The most common type of PSVT is AV nodal reentrant tachycardia (AVNRT), which usually affects **young patients** (eg, age <40) with an otherwise normal heart.

Patients with AVNRT have **2 distinct** AV nodal conduction pathways: a **fast pathway** with a long refractory period and a **slow pathway** with a short refractory period. During normal conduction, an impulse from the atria travels down both the fast and slow pathways, and the impulse from the slow pathway is blocked by the long refractory period of the fast pathway, allowing organized conduction of one impulse to the ventricles. On occasion, an impulse from the atria (eg, premature atrial contraction) occurs while the fast pathway is still refractory, traveling down only the slow pathway. If the fast pathway is no longer refractory by the time the impulse reaches the bottom of the slow pathway, the impulse may travel back up the fast pathway, creating a **reentrant circuit** with rapid conduction of impulses to the ventricles.

On ECG, retrograde conduction up the fast pathway creates a **retrograde P wave**; however, because this conduction is usually simultaneous with anterograde conduction to the ventricles, the **P wave is often buried within the QRS complex** and is not visible.

(Choice A) Abnormal conduction in areas of ischemic ventricular scarring is a common cause of monomorphic ventricular tachycardia. ECG shows a regular rhythm, but with wide QRS complexes because the arrhythmia originates below the AV node.

(Choice C) Atrial remodeling with ectopic foci in the pulmonary vein ostia is the typical cause of atrial fibrillation. ECG shows an irregular rhythm and no P waves (P waves are truly absent because no organized atrial depolarization occurs). The QRS complexes are narrow.

(Choice D) Degeneration of the sinoatrial node can lead to sick sinus syndrome, usually in patients age >65. ECG can show sinus bradycardia with sinus pauses and sinus arrest. Patients may sometimes develop episodes of sinus tachycardia, which can appear similar to PSVT but with normal P waves present.

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down both the fast and slow pathways, and the impulse from the slow pathway is blocked by the long refractory period of the fast pathway, allowing organized conduction of one impulse to the ventricles. On occasion, an impulse from the atria (eg, premature atrial contraction) occurs while the fast pathway is still refractory, traveling down only the slow pathway. If the fast pathway is no longer refractory by the time the impulse reaches the bottom of the slow pathway, the impulse may travel back up the fast pathway, creating a **reentrant circuit** with rapid conduction of impulses to the ventricles.

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(Choice E) Delayed repolarization of ventricular cardiomyocytes can lead to torsade de pointes, a form of polymorphic ventricular tachycardia. ECG shows wide QRS complexes that oscillate in height and position.

Educational objective:
Atrioventricular nodal reentrant tachycardia is the most common type of paroxysmal supraventricular tachycardia, resulting from abnormal conduction through 2 distinct atrioventricular nodal pathways. ECG typically demonstrates a narrow QRS complex tachycardia with a regular rhythm and buried (not visible) P waves.

Pathophysiology	Cardiovascular System	Supraventricular tachycardia
Subject	System	Topic

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Conduction up

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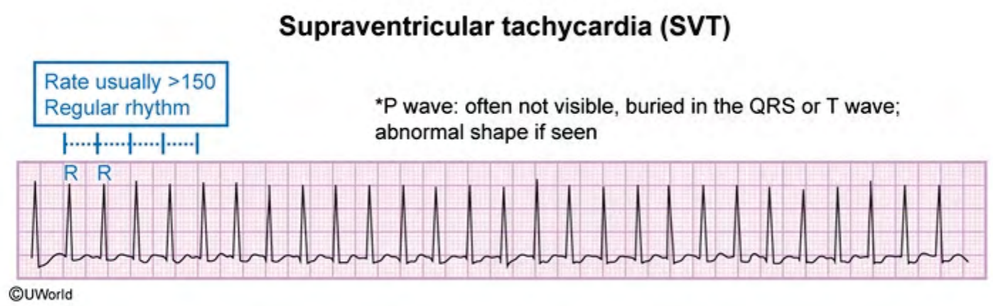

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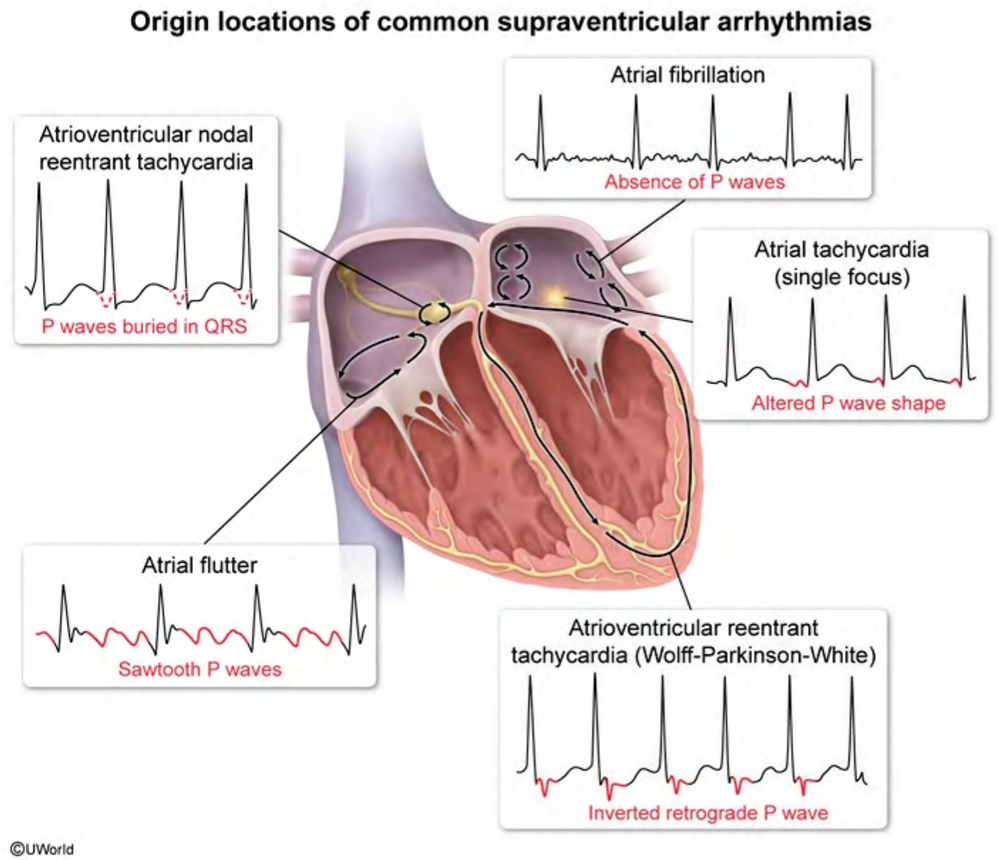

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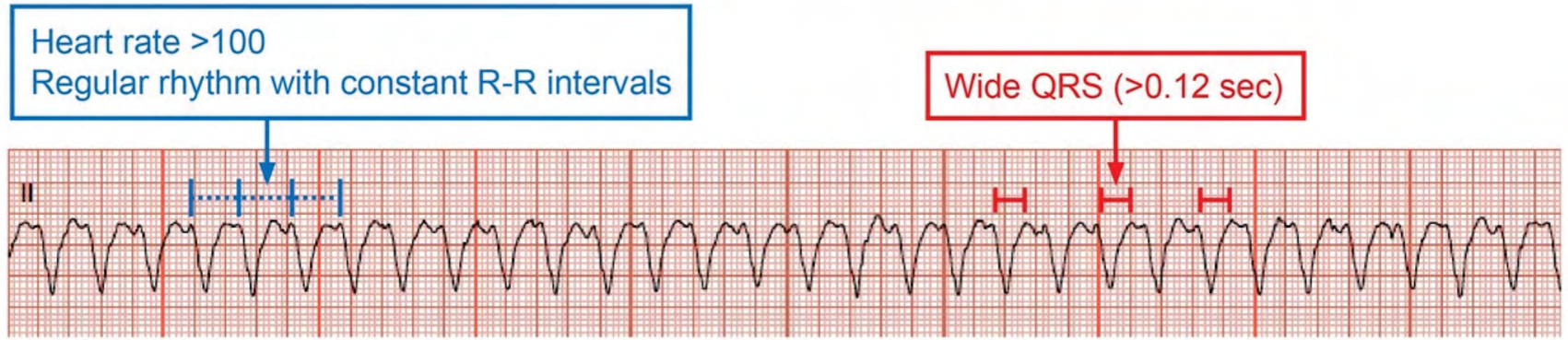
Ventricular tachycardia (VT)



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These findings are consistent with **paroxysmal supraventricular tachycardia** (PSVT), an episodic cardiac arrhyth
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(Choice E) Delayed repolarization of ventricular cardiomyocytes can lead to torsade de pointes, a form of

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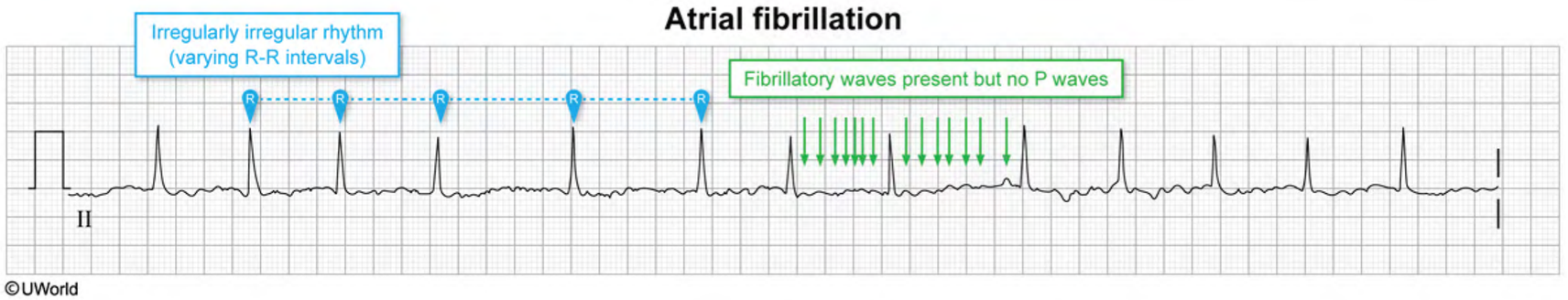

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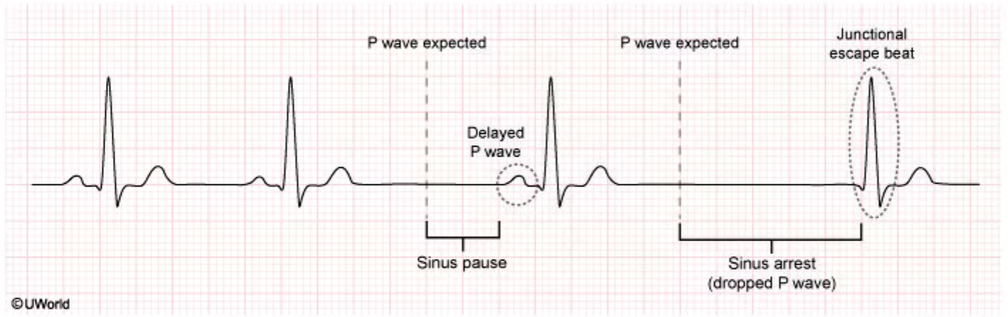

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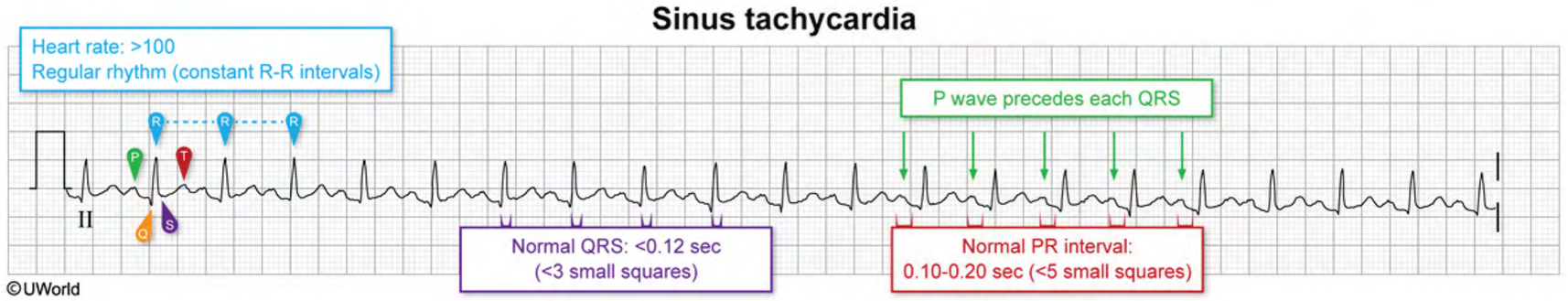

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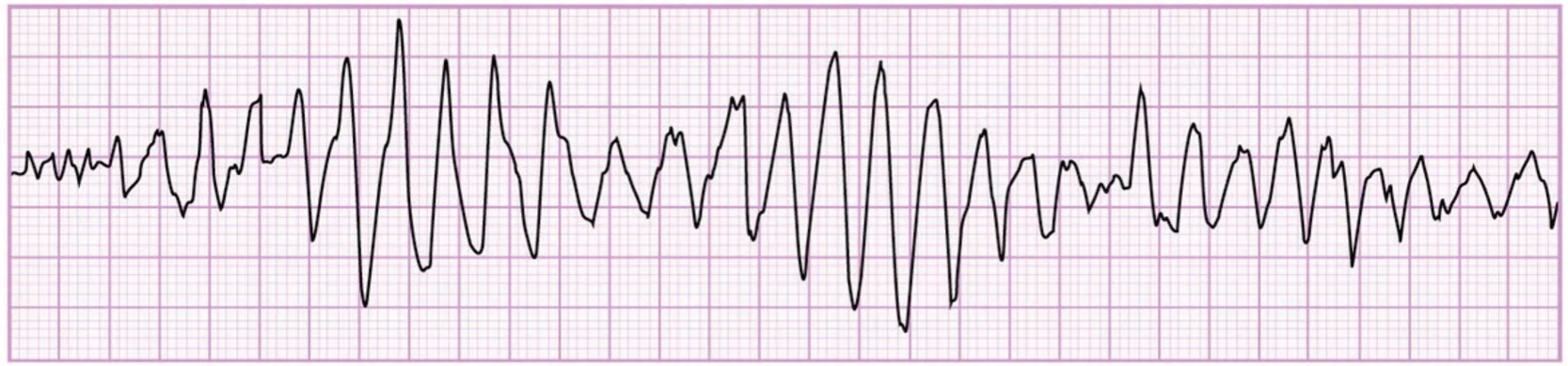
Torsade de pointes



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A 36-year-old woman is brought to the emergency department with sudden-onset right side weakness and speech difficulty. During the last 3 weeks, the patient has experienced progressive fatigue, malaise, and low-grade fevers. Despite the symptoms, she did not seek medical attention and did not take any medications. She had a dental extraction 5 weeks ago, which was uncomplicated. The patient has never previously been significantly ill or hospitalized. She works as a receptionist at a legal firm and has never traveled outside the United States. She does not use tobacco, alcohol, or illicit drugs. The patient is admitted to the hospital, but despite adequate resuscitative measures, she dies 2 hours later. Gross autopsy shows large, friable irregular masses attached to the atrial surface of a valve. Which of the following underlying conditions most likely predisposed this patient to developing these cardiac changes?

- A. Atrial septal defect
- B. Coronary artery disease
- C. Hypertrophic cardiomyopathy
- D. Mitral annular calcification
- E. Regurgitant mitral valve prolapse

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- ✗ A. Atrial septal defect (9%)
- B. Coronary artery disease (0%)
- C. Hypertrophic cardiomyopathy (0%)
- D. Mitral annular calcification (29%)
- ✓ E. Regurgitant mitral valve prolapse (58%)

Incorrect
Correct answer
E

58%
Answered correctly

03 secs
Time Spent

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Explanation

This patient had **subacute infective endocarditis (IE)** complicated by an **embolic stroke**. The large, friable irregular masses seen on autopsy are most likely vegetations and are the most probable source of this patient's left middle cerebral artery embolus (eg, right-sided weakness, speech difficulty).

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Explanation

This patient had **subacute infective endocarditis (IE)** complicated by an **embolic stroke**. The large, friable irregular masses seen on autopsy are most likely vegetations and are the most probable source of this patient's left middle cerebral artery embolus (eg, right-sided weakness, speech difficulty).

The mitral valve is the valve most frequently affected by IE. **Mitral valve prolapse (MVP)** is the most common underlying valvular disease predisposing to the development of IE in **developed countries**, particularly when it is associated with coexistent **mitral regurgitation**. Microscopic deposits of platelets and fibrin occur spontaneously in individuals with valvular disease secondary to endocardial injury from turbulent blood flow. These deposits then become colonized by microorganisms (eg, viridans streptococci) during episodes of bacteremia (eg, following dental extraction).

In **developing countries**, the most common acquired heart disease leading to IE development is **rheumatic heart disease** (RHD). RHD is a late (10-20 years) complication of acute rheumatic fever (eg, migratory arthritis, carditis/valvulitis, erythema marginatum). RHD was historically considered a frequent predisposing factor for IE; however, MVP, valvular sclerosis, and mechanical valves are now more common causes in developed nations due to the recognition and treatment of group A streptococcal pharyngitis.

(Choice A) Congenital heart lesions that predispose to increased risk of IE include bicuspid aortic valve, ventricular septal defects, patent ductus arteriosus, and unrepaired tetralogy of Fallot. Isolated atrial septal defects are not commonly associated with an increased risk of IE, likely due to the low-pressure differential between the atria and the absence of high-velocity intracardiac flow jets that can damage the endocardium.

(Choice B) Coronary heart disease can cause ischemic papillary muscle dysfunction with resultant mitral regurgitation. However, the clinical presentation and autopsy findings in this patient are most consistent with MVP and IE.

(Choice C) IE is a rare complication of hypertrophic cardiomyopathy and is almost always seen in patients with

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become colonized by microorganisms (eg, viridans streptococci) during episodes of bacteremia (eg, following dental extraction).

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(Choice B) Coronary heart disease can cause ischemic papillary muscle dysfunction with resultant mitral regurgitation. However, the clinical presentation and autopsy findings in this patient are most consistent with MVP and IE.

(Choice C) IE is a rare complication of hypertrophic cardiomyopathy and is almost always seen in patients with outflow obstruction. Hypertrophic cardiomyopathy is characterized by asymmetric ventricular hypertrophy leading to reduced left ventricular cavity size.

(Choice D) Mitral annular calcification refers to degeneration and progressive calcium deposition involving the fibrous annulus of the mitral valve. Mitral annular calcification is usually seen in older adults (age >60), and the annulus is not a common location for IE.

Educational objective:
Mitral valve prolapse with regurgitation is the most common predisposing condition for native valve infective endocarditis (IE) in developed nations. Rheumatic heart disease remains a frequent cause of IE in developing nations.

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The mitral valve is the valve most frequently affected by IE. **Mitral valve prolapse (MVP)** is the most common underlying valvular disease predisposing to the development of IE in **developed countries**, particularly when it is associ
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Educational objective:

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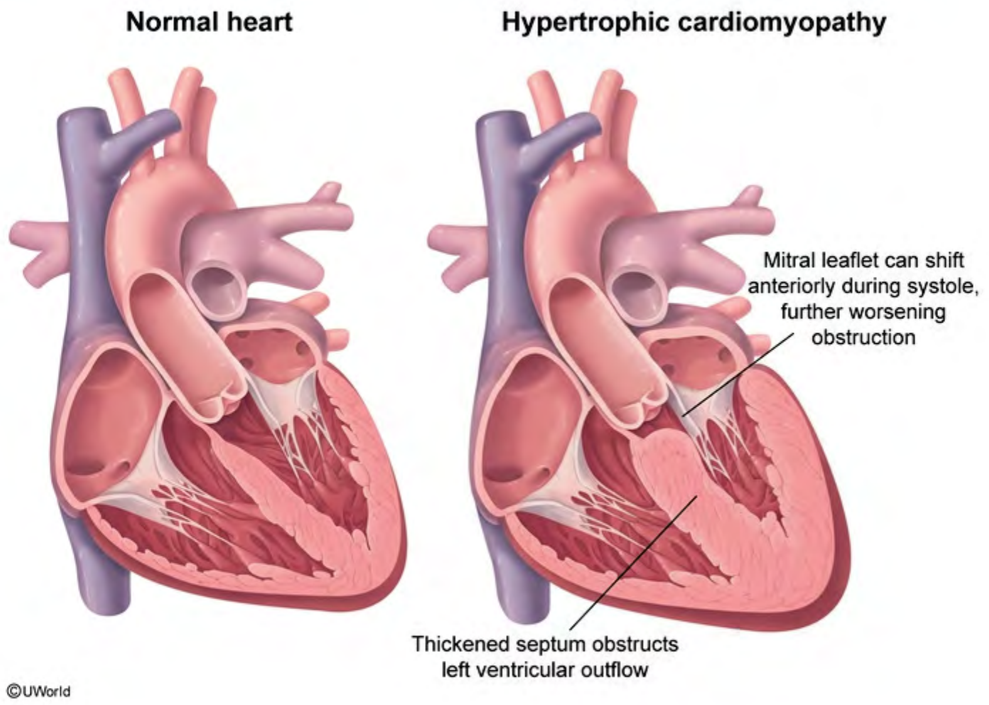

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Atherosclerotic lesions of the coronary arteries can limit blood flow to the myocardial regions supplied by the affected vessels. In some patients, certain medications can cause a redistribution of blood flow away from the ischemic areas, exacerbating existing myocardial ischemia. Which of the following drug effects is most likely to produce this phenomenon?

- A. Coronary arteriolar dilation
- B. Decreased myocardial contractility
- C. Epicardial vessel dilation
- D. Negative chronotropic effect
- E. Systemic venous dilation

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Atherosclerotic lesions of the coronary arteries can limit blood flow to the myocardial regions supplied by the affected vessels. In some patients, certain medications can cause a redistribution of blood flow away from the ischemic areas, exacerbating existing myocardial ischemia. Which of the following drug effects is most likely to produce this phenomenon?

- ✓ A. Coronary arteriolar dilation (61%)
- B. Decreased myocardial contractility (4%)
- C. Epicardial vessel dilation (11%)
- D. Negative chronotropic effect (4%)
- E. Systemic venous dilation (18%)

Correct | 61% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Hemodynamically significant atherosclerotic lesions (>70% occlusion) reduce the amount of blood supplied by a coronary vessel to the corresponding myocardial territory. In response, the myocardium releases locally acting, endogenous substances (eg, adenosine, nitric oxide) that cause **dilation of coronary arterioles**, leading to a downstream reduction in vascular resistance and recruitment of additional blood flow to that region. Arterioles that supply areas of significantly **ischemic myocardium** can become **maximally dilated** at rest, limiting their capacity to further increase blood flow.

Pharmacologic stress agents (eg, adenosine, dipyridamole) are used during myocardial perfusion imaging to simulate exercise-induced coronary arteriole dilation and assist in identifying areas of ischemic myocardium. Most

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Pharmacologic stress agents (eg, adenosine, dipyridamole) are used during myocardial perfusion imaging to simulate exercise-induced coronary arteriole dilation and assist in identifying areas of ischemic myocardium. Most of the time, administration of these drugs will result in a significant **increase in blood flow to nonischemic myocardium** and no change in blood flow to ischemic myocardium (because additional blood flow is recruited proximally).

In some patients with extensive coronary artery disease, the recruitment of additional upstream blood flow is limited due to proximal atherosclerotic disease (reduced coronary flow reserve). In such a setting, these drugs can cause an absolute **decrease in blood flow to ischemic myocardium**, as most of the limited blood supply is redirected toward the newly vasodilated areas of nonischemic myocardium. This redistribution of blood flow leading to worsening ischemia is referred to as **coronary steal**.

(Choices B, D, and E) Decreased myocardial contractility, negative chronotropy, and systemic venous dilation (leading to reduced preload) all decrease myocardial oxygen demand through a reduction in cardiac work. These mechanisms act to improve myocardial ischemia globally and will not result in a redistribution of blood flow away from areas of ischemic myocardium.

(Choice C) The epicardial vessels are the large coronary arteries of the heart (eg, right coronary, left circumflex). Although dilation of these large arteries plays a role in increasing myocardial blood flow, coronary blood flow regulation is mostly mediated by the arterioles. Dilation of large coronary arteries would improve cardiac blood flow and would not trigger coronary steal.

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Educational objective:
Increases in resting blood flow to ischemic myocardium are primarily mediated by locally-acting substances (eg, adenosine, nitric oxide) that trigger coronary arteriolar vasodilation. Pharmacologic arteriolar vasodilators (eg, adenosine, dipyridamole) mimic the vasodilation that occurs with exercise and may cause redistribution of blood flow from ischemic to nonischemic areas of myocardium (ie, coronary steal).

Pathophysiology Cardiovascular System Coronary blood flow

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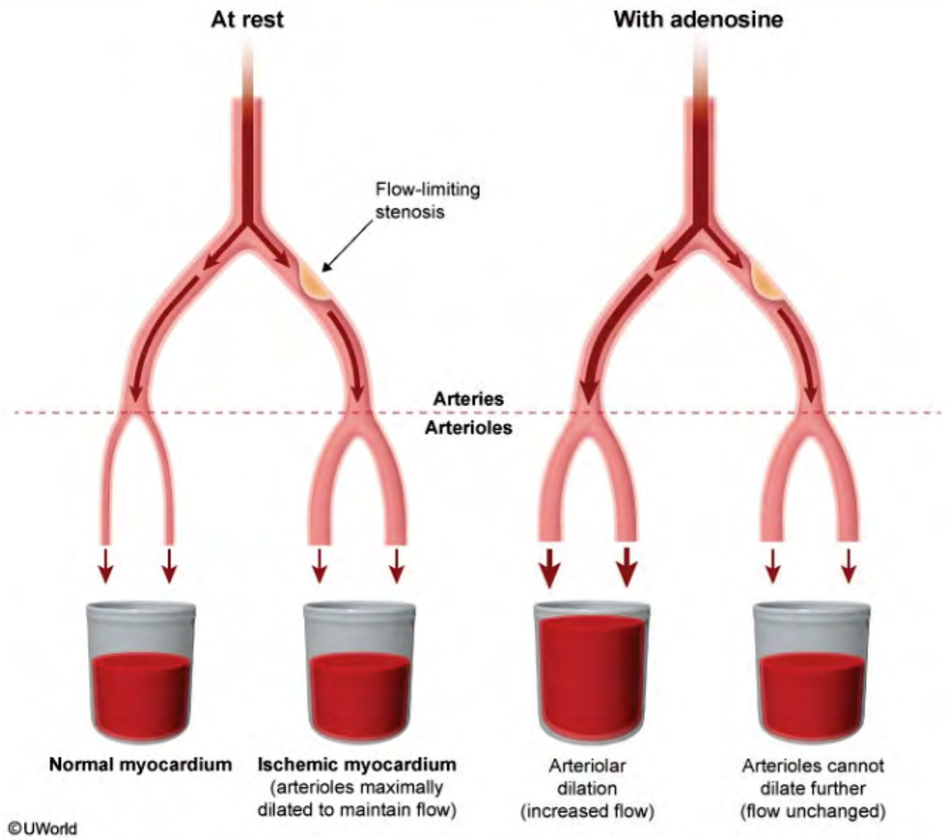

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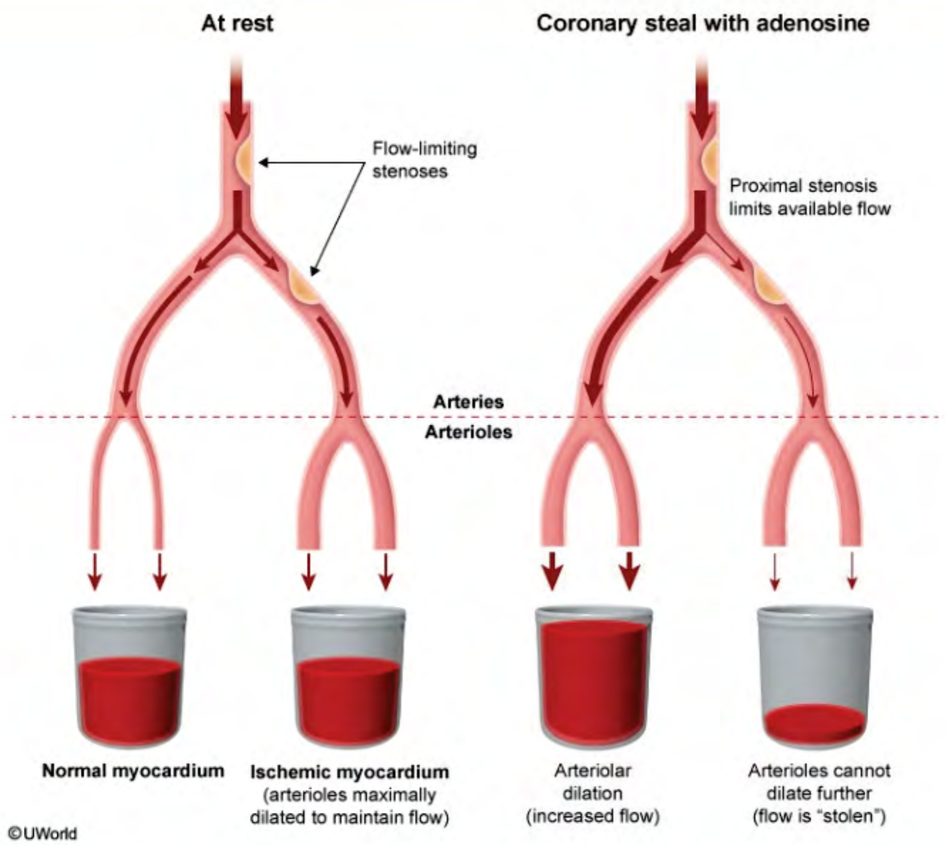

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A 30-year-old man is brought to the emergency department due to left-sided weakness. His left arm was sluggish after awakening, and he had difficulty walking to the bathroom. The patient has no known medical conditions but has a 2-month history of positional dyspnea. Vital signs are within normal limits. On examination, strength of the left upper and left lower extremity is reduced, and touch/temperature sensation is similarly diminished. Cardiovascular examination reveals an intermittent murmur at the apex without carotid bruits. Echocardiography shows a mobile, 3-cm left atrial mass obstructing the mitral opening during diastole. Histologic examination of this mass would most likely show which of the following findings?

- A. Amorphous extracellular matrix
- B. Malignant epithelial cells with necrotic debris
- C. Malignant vascular spindle cells
- D. Organized thrombus
- E. Well-organized, mature adipocytes

Submit

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- ✓ A. Amorphous extracellular matrix (74%)
- ✗ B. Malignant epithelial cells with necrotic debris (5%)
- C. Malignant vascular spindle cells (5%)
- D. Organized thrombus (12%)
- E. Well-organized, mature adipocytes (2%)

Incorrect
Correct answer
A

74%
Answered correctly

03 secs
Time Spent

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Explanation

Cardiac myxoma

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A 30-year-old man is brought to the emergency department due to left-sided weakness. His left arm was sluggish after a
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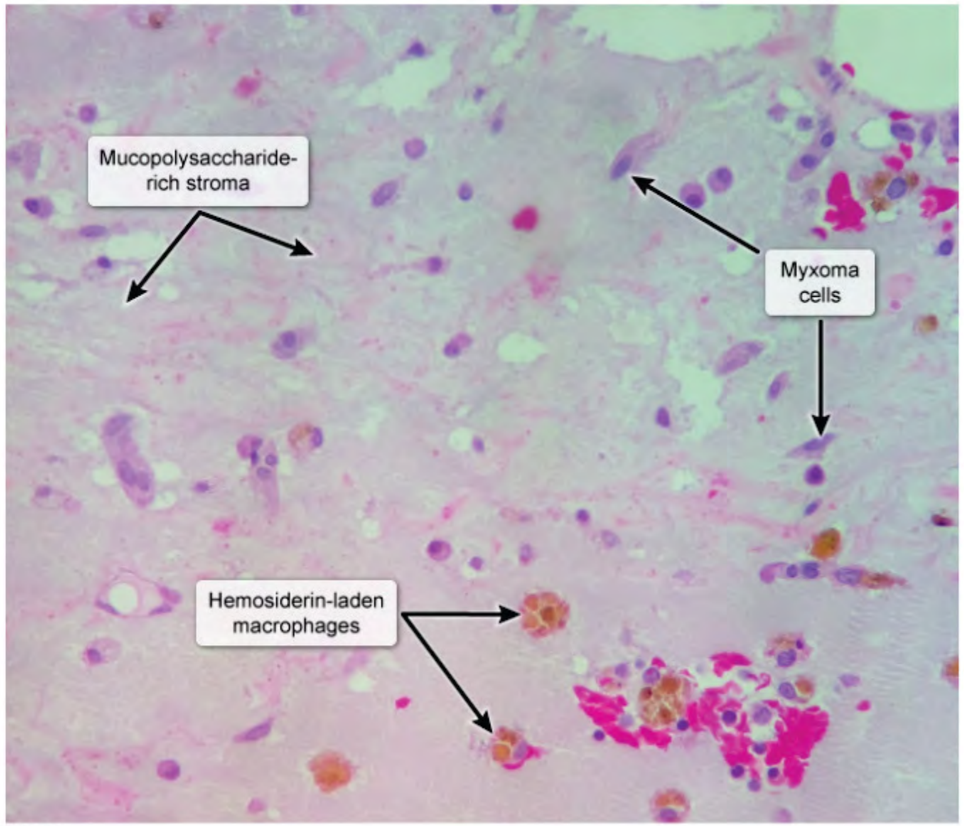
Cardiac myxoma



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This patient's left atrial mass on echocardiography is consistent with an **atrial myxoma**; this benign tumor is the most common primary cardiac neoplasm. Approximately 80% originate in the **left atrium**, and they can present with **systemic embolization** (eg, stroke, as in this patient) from tumor fragments passing into the systemic circulation. The tumors may also lead to constitutional symptoms (eg, fatigue, weight loss, low-grade fever) resulting from cytokine release, and they may cause transient mitral valve obstruction, leading to symptoms that can **mimic mitral valve stenosis** (eg, dyspnea, hemoptysis).

Physical examination often reveals an intermittent or positional **mid-diastolic murmur** ("tumor plop") that results from the motion of the tumor mass obstructing the mitral valve orifice. Histopathologic examination of a myxoma reveals **amorphous extracellular matrix** with scattered stellate or globular myxoma cells within abundant mucopolysaccharide (myxoid) ground substance containing chondroitin sulfate and hyaluronic acid. Because of their high vascularity, these tumors often demonstrate areas of hemorrhage accompanied by brown, hemosiderin-laden macrophages.

(Choices B and C) Primary malignant cardiac tumors are quite rare; those that occur are usually either sarcomas or lymphomas. Malignant vascular spindle cells suggest primary cardiac angiosarcoma, which usually arises in the right atrium and is exceedingly rare. Malignant epithelial cells with necrotic debris suggest secondary involvement of the heart from a metastatic malignancy (eg, melanoma, lung cancer, breast cancer). Metastatic spread to the heart usually involves the pericardium and rarely creates a mobile intracardiac mass.

(Choice D) An organized thrombus can form in the left atrium or left atrial appendage in patients with atrial flutter or fibrillation and may cause stroke due to systemic embolization. However, thrombi are typically adherent to the cardiac endothelium; a mobile mass in the left atrium is more characteristic of a myxoma.

(Choice E) Lipomas are benign tumors composed of well-organized, mature adipocytes. They are the second most common type of primary cardiac neoplasm; however, unlike myxomas, lipomas rarely embolize.

Educational objective:
Myxomas are the most common primary cardiac neoplasm, and approximately 80% originate in the left atrium. Patients may have systemic embolization (eg, stroke) or symptomatic mitral valve obstruction that may be worse

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circulation. The tumors may also lead to constitutional symptoms (eg, fatigue, weight loss, low-grade fever) resulting from cytokine release, and they may cause transient mitral valve obstruction, leading to symptoms that can **mimic mitral valve stenosis** (eg, dyspnea, hemoptysis).

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Educational objective:
Myxomas are the most common primary cardiac neoplasm, and approximately 80% originate in the left atrium. Patients may have systemic embolization (eg, stroke) or symptomatic mitral valve obstruction that may be worse with certain body positions. Histopathologic examination reveals amorphous extracellular matrix with scattered stellate or globular myxoma cells within abundant mucopolysaccharide ground substance.

References

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A 56-year-old man comes to the clinic for a routine checkup. Medical history includes hypertension, type 2 diabetes mellitus, hyperlipidemia, and mild intermittent asthma. The patient currently takes no medications and has not seen a physician in 7 years. He reports feeling well. Blood pressure is 152/101 mm Hg and pulse is 87/min. Waist circumference is 110 cm (43 in). Laboratory results are as follows:

Low-density lipoprotein	161 mg/dL
Fasting blood glucose	201 mg/dL
Hemoglobin A1c	7.4%

Which of the following vascular beds is most likely to carry the highest atherosclerotic burden in this patient?

- A. Coronary arteries
- B. Internal thoracic arteries
- C. Mesenteric arteries
- D. Popliteal arteries
- E. Renal arteries

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Low-density lipoprotein	161 mg/dL
Fasting blood glucose	201 mg/dL
Hemoglobin A1c	7.4%

Which of the following vascular beds is most likely to carry the highest atherosclerotic burden in this patient?

- ✓ A. Coronary arteries (61%)
- B. Internal thoracic arteries (2%)
- C. Mesenteric arteries (6%)
- D. Popliteal arteries (6%)
- E. Renal arteries (22%)

Correct

61% Answered correctly

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Explanation

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E. Renal arteries (22%)

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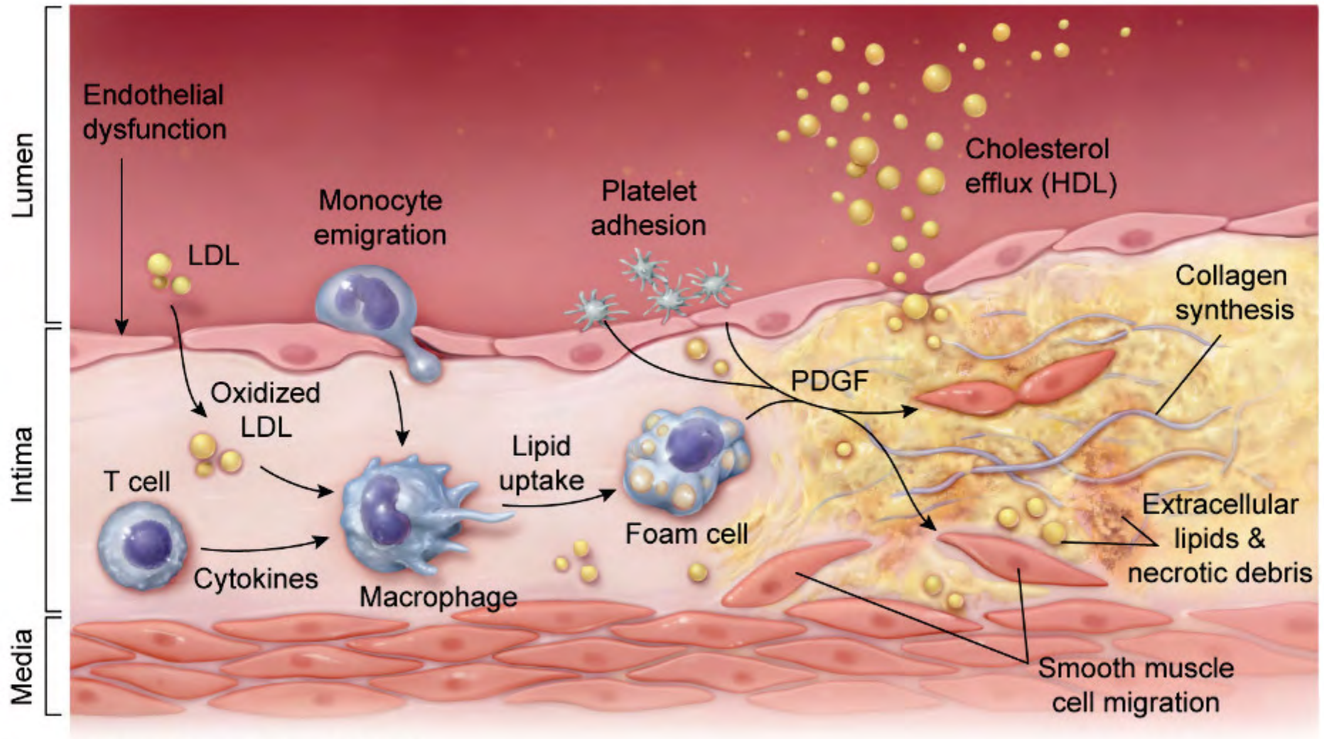
Formation of atheroma



PDGF = platelet-derived growth factor.

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This patient has multiple risk factors (eg, hypertension, diabetes mellitus, hyperlipidemia) that contribute to atherosclerosis. Atherosclerosis is a pathophysiologic process that begins in childhood with the development of focal intimal thickening due to accumulation of lipid-laden macrophages and extracellular matrix ("fatty streaks"). With advancing age, fatty streaks transition into atherosclerotic plaques, such as **fibrous cap atheromas** and fibrous plaques. More advanced lesions develop a lipid-rich necrotic core and areas of calcification.

Atherosclerosis is a chronic disease of the arterial walls and involves all major vascular beds. The most susceptible vascular regions involve bends and branch points that encourage **turbulent blood flow**, which disrupts vascular wall integrity and leads to endothelial cell dysfunction. Turbulent blood flow also leads to prolonged endothelial contact with cholesterol particles.

The hemodynamics of the **lower abdominal aorta** and the **coronary arteries** make these vascular beds the most susceptible to atherosclerosis of all the major vascular beds in the body. In fact, atherosclerotic lesions (eg, intimal thickening, fatty streaks) occur in these vessels as early as the second decade of life.

(Choice B) The internal thoracic arteries (previously known as the internal mammary arteries) are relatively resistant to developing atherosclerosis and are often used as conduits in patients undergoing coronary artery bypass grafting.

(Choices C and E) The mesenteric and renal arteries are susceptible to developing atherosclerosis; however, the burden of disease tends to be less severe than in the coronary circulation.

(Choice D) Along with the carotid arteries, the popliteal arteries are among the most susceptible to atherosclerosis in the body. However, these arteries are not as susceptible to atherosclerosis as the aorta and coronary arteries.

Educational objective:
Atherosclerosis is a pathophysiologic process involving endothelial cell dysfunction, and it develops most rapidly in areas with bends and branch points that encourage turbulent blood flow. The lower abdominal aorta and coronary arteries are the vascular beds most susceptible to atherosclerosis; they tend to develop atherosclerosis earliest in life and have the highest overall atherosclerotic burden.

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A 56-year-old man is brought to the emergency department due to sudden-onset, severe chest pain. Blood pressure is 88/56 mm Hg and pulse is 83/min. On physical examination, the patient is in moderate distress. The jugular veins are distended. No murmurs or extra heart sounds are heard on cardiac auscultation. The lungs are clear to auscultation. ECG shows evidence of an ST-segment elevation myocardial infarction. Bedside echocardiography reveals contractile dysfunction of a large portion of the right ventricle and normal function of the entire left ventricle consistent with myocardial infarction involving only the right ventricle. Percutaneous coronary intervention with coronary angiography is performed and shows thrombotic occlusion affecting one of the major coronary arteries. No other significant atherosclerotic lesions are identified. Which of the following most likely explains the findings in this patient?

	Occluded artery	Coronary artery dominance
○ A.	Left anterior descending	Left dominant
○ B.	Left anterior descending	Right dominant
○ C.	Left circumflex	Left dominant
○ D.	Left circumflex	Right dominant
○ E.	Right coronary	Left dominant
○ F.	Right coronary	Right dominant

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		Occluded artery	Coronary artery dominance	
✗	A.	Left anterior descending	Left dominant	(2%)
	B.	Left anterior descending	Right dominant	(7%)
	C.	Left circumflex	Left dominant	(2%)
	D.	Left circumflex	Right dominant	(2%)
✓	E.	Right coronary	Left dominant	(46%)
	F.	Right coronary	Right dominant	(39%)

Incorrect
Correct answer
E

46%
Answered correctly

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Explanation

Coronary artery dominance

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A. Left anterior descending — Left dominant — (2%)

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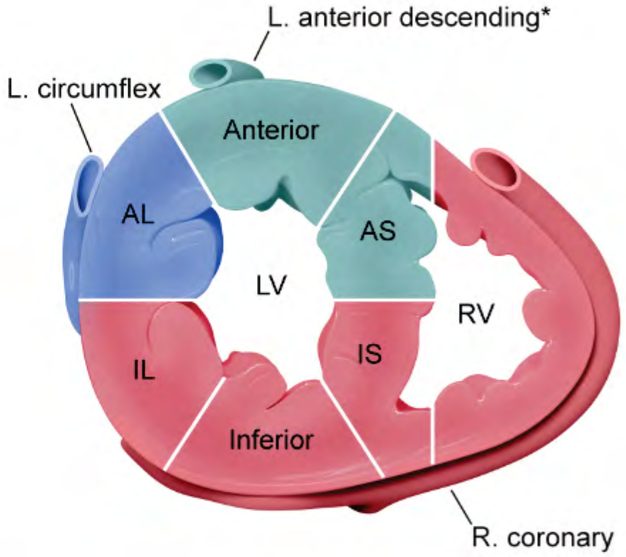
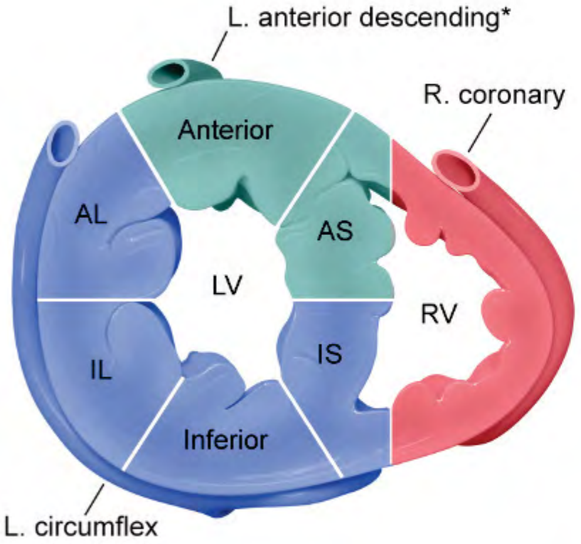
Coronary artery dominance

Right dominant circulation
~85% of patients



Left dominant circulation
~10% of patients



*The anterior wall is substantially larger than the inferior wall in 3 dimensions, such that the left anterior descending artery supplies ~50% of the total left ventricular myocardium.

AL = anterolateral; **AS** = anteroseptal; **IL** = inferolateral; **IS** = inferoseptal; **LV** = left ventricle; **RV** = right ventricle.

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R. coronary L. circumflex

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AL = anterolateral; **AS** = anteroseptal; **IL** = inferolateral; **IS** = inferoseptal; **LV** = left ventricle; **RV** = right ventricle. ©UWorld

The coronary circulation is composed of 3 major epicardial vessels.

- The **left anterior descending** artery (LAD) arises from the left main artery and courses down the anterior heart, supplying the anterior left ventricular (LV) wall and septum.
- The **left circumflex** artery (LCx) arises from the left main artery and courses around the left side of the heart to supply the lateral LV wall. Occasionally, the LCx extends all the way around the heart to supply the inferior LV wall (left-dominance).
- The **right coronary** artery (RCA) comes directly off the aorta and courses around the right side of the heart to supply the right ventricle (RV). In most patients, the RCA extends past the RV to also supply the inferior LV wall (right-dominance).

Blood supply to the **inferior LV wall** depends on coronary artery **dominance**. Right coronary dominance is present in approximately 85% of patients and involves the RCA coursing past the RV to give off the posterior descending artery (PDA) and supply the inferior LV wall. Left coronary dominance is present in approximately 10% of patients and involves the LCx extending around the left side of the heart to give off the PDA and supply the inferior LV wall. Approximately 5% of patients have codominant circulation.

In virtually all patients, the RV is fully supplied by the RCA, and in patients with left-dominant circulation, none of the inferior LV wall is supplied by the RCA. Therefore, **isolated RV myocardial infarction** (MI) can occur due to **RCA occlusion** in a patient with **left-dominant circulation**. Given the low prevalence of left-dominant circulation, isolated RV MI is an uncommon event (approximately 3% of MIs).

(Choices A, B, C, and D) The LAD and LCx do not supply the RV and are not involved in RV MI.

(Choice F) With right-dominant circulation, the RCA supplies the inferior LV wall. Therefore, RCA occlusion would cause MI involving the RV and the inferior LV wall, or the inferior LV wall alone if the occlusion is distal to the RV.

Educational objective:
Blood supply to the inferior left ventricular (LV) wall is determined by coronary dominance. Most patients have right-dominant circulation, in which the inferior LV wall is supplied by the right coronary artery. Approximately 10% of patients have left-dominant circulation, in which the inferior LV wall is supplied by the left circumflex artery. The

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References

Cardiac dominance

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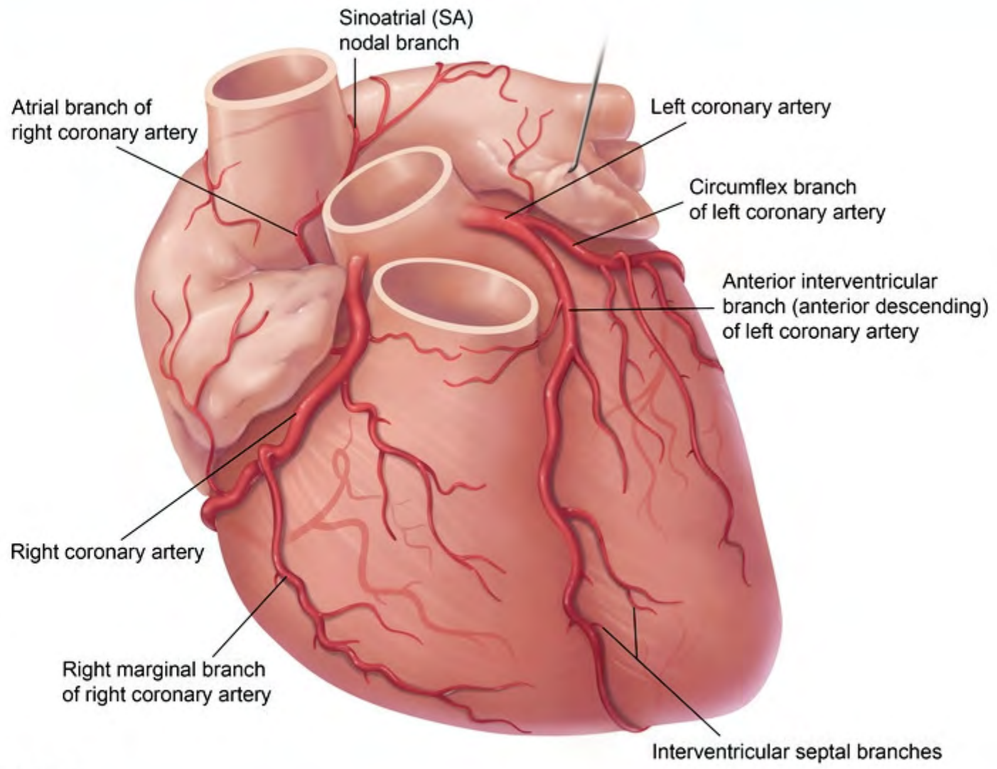
Coronary arteries: anterior view



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A 32-year-old man comes to the office due to progressive dyspnea, dizziness, and chest discomfort. He has no prior medical problems but had a mild respiratory illness 2 weeks earlier that resolved spontaneously. His father has a history of myocardial infarction. Echocardiography shows pericardial fluid accumulation with late diastolic collapse of the right atrium. Which of the following physical examination findings is most likely to be seen in this patient?

- A. Beat-to-beat variation in pulse amplitude
- B. Drop in pulse amplitude during inspiration
- C. Pulse with 2 distinct peaks
- D. Rapidly rising pulse with high amplitude
- E. Slow-rising low-amplitude pulse

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- A. Beat-to-beat variation in pulse amplitude (17%)
- ✓ B. Drop in pulse amplitude during inspiration (61%)
- C. Pulse with 2 distinct peaks (7%)
- D. Rapidly rising pulse with high amplitude (3%)
- E. Slow-rising low-amplitude pulse (10%)

Correct | 61% Answered correctly | 07 secs Time Spent | 2023 Version

Explanation

This patient has a pericardial effusion with associated **cardiac tamponade**. Echocardiography showing pericardial fluid accumulation with **late diastolic collapse of the right atrium** (due to excessive pericardial fluid compressing the right atrium) is diagnostic.

Pulsus paradoxus is an important physical finding in cardiac tamponade and refers to an exaggerated drop in systolic blood pressure (>10 mm Hg) during inspiration. Fluid accumulation in the pericardium limits the ability of the right ventricular free wall to expand outward; during inspiration, the increase in venous return is instead accommodated by bowing of the interventricular septum toward the left ventricle (LV), decreasing LV end-diastolic volume and stroke volume. The result is **decreased systolic pulse pressure during inspiration**.

Less common causes of pulsus paradoxus include severe asthma and chronic obstructive pulmonary disease (due

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This patient has a pericardial effusion with associated **cardiac tamponade**. Echocardiography showing pericardial fluid accumulation with **late diastolic collapse of the right atrium** (due to excessive pericardial fluid compressing the right atrium) is diagnostic.

Pulsus paradoxus is an important physical finding in cardiac tamponade and refers to an exaggerated drop in systolic blood pressure (>10 mm Hg) during inspiration. Fluid accumulation in the pericardium limits the ability of the right ventricular free wall to expand outward; during inspiration, the increase in venous return is instead accommodated by bowing of the interventricular septum toward the left ventricle (LV), decreasing LV end-diastolic volume and stroke volume. The result is **decreased systolic pulse pressure during inspiration**.

Less common causes of pulsus paradoxus include severe asthma and chronic obstructive pulmonary disease (due to excessive swings in intrathoracic pressure with breathing), as well as the restriction of right ventricular filling that occurs with constrictive pericarditis.

(Choice A) Pulsus alternans refers to beat-to-beat variation in pulse amplitude (ie, systolic blood pressure); it is most commonly seen in patients with severe LV systolic dysfunction (possibly related to the effect of the Frank-Starling mechanism on the failing ventricle) and is not expected with cardiac tamponade. Electrical alternans is sometimes seen with cardiac tamponade and refers to a beat-to-beat variation in the QRS complex amplitude on ECG (ie, variation in electrical signal rather than pulse) due to the swinging motion of the heart in the pericardial fluid.

(Choice C) A dicrotic pulse has 2 distinct peaks (one during systole and the other during diastole) due to an accentuated diastolic dicrotic wave after the dicrotic notch. This finding typically occurs in patients with severe systolic dysfunction and high systemic vascular resistance.

(Choice D) A hyperkinetic pulse has a rapidly rising amplitude due to the rapid ejection of a large stroke volume against a decreased afterload. This can occur with aortic regurgitation and high-output heart failure (eg, thyrotoxicosis, arteriovenous fistula).

(Choice E) Fixed LV outflow tract obstruction (eg, valvular aortic stenosis) can cause pulsus parvus et tardus, or a

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(Choice E) Fixed LV outflow tract obstruction (eg, valvular aortic stenosis) can cause pulsus parvus et tardus, or a slow-rising, low-amplitude pulse resulting from diminished stroke volume (pulsus parvus) and prolonged LV ejection time (pulsus tardus).

Educational objective:
Pulsus paradoxus refers to an exaggerated drop (>10 mm Hg) in systolic blood pressure during inspiration. It is most commonly seen in patients with cardiac tamponade but can also occur in severe asthma, chronic obstructive pulmonary disease, or constrictive pericarditis.

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pulmonary disease, or constrictive pericarditis.

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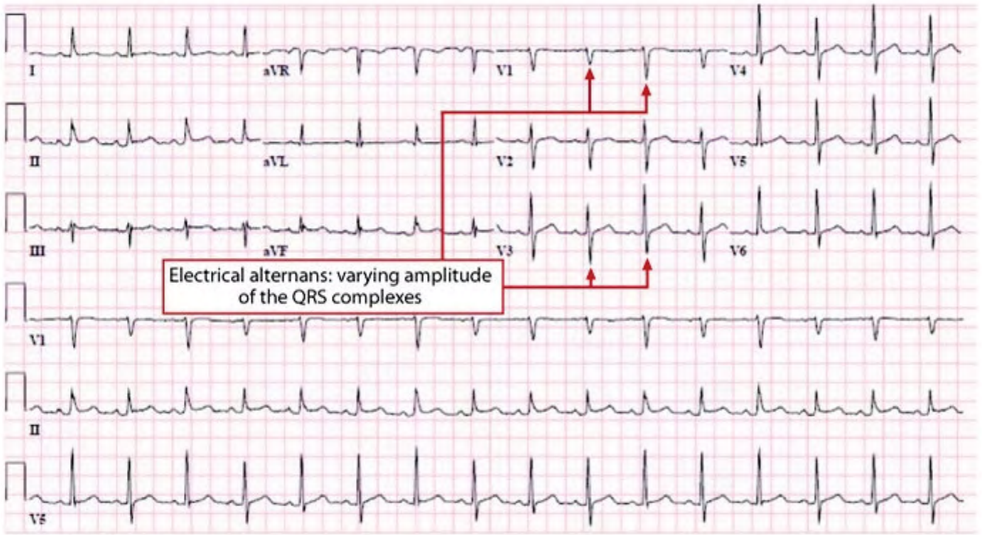

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A 34-year-old man comes to the clinic for evaluation of a heart murmur. The patient has no chest pain, palpitations, dizziness, or shortness of breath at rest. He also exercises regularly without any abnormal symptoms. Cardiac auscultation in the second right intercostal space reveals an ejection click along with a soft systolic murmur radiating to the neck. A normal volume and pressure tracing of the left ventricle is shown in the image below. Which of the following letters corresponds to the opening of the valve most likely affected in this patient?

- A. A
- B. B
- C. C
- D. D
- E. E

Submit

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A 34-year-old man comes to the clinic for evaluation of a heart murmur. The patient has no chest pain,
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patien

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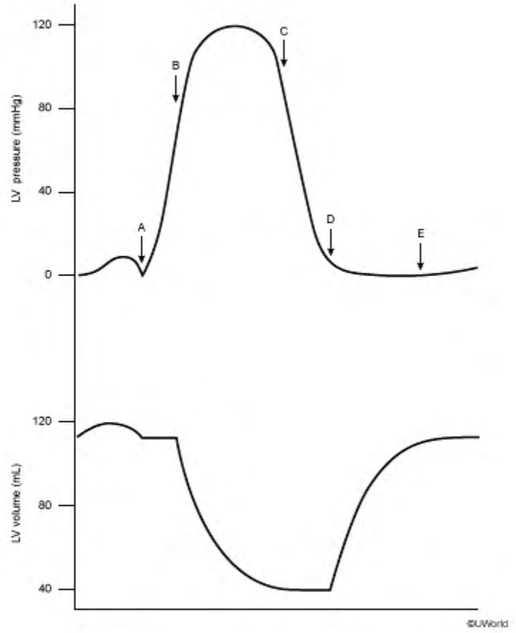

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E. E

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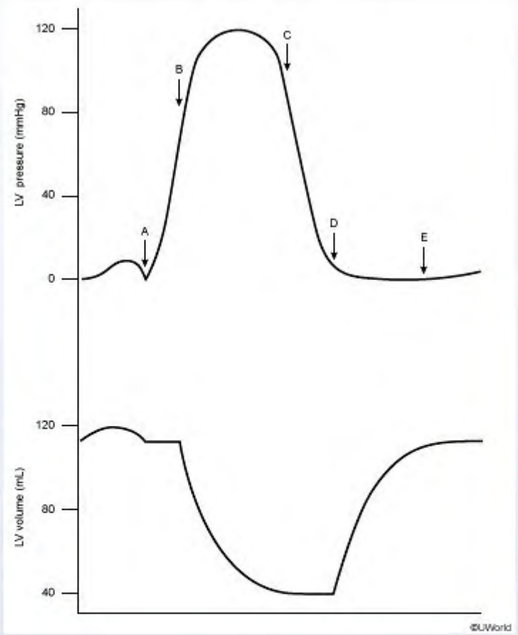

- A. A
- B. B
- C. C
- D. D
- E. E

Submit

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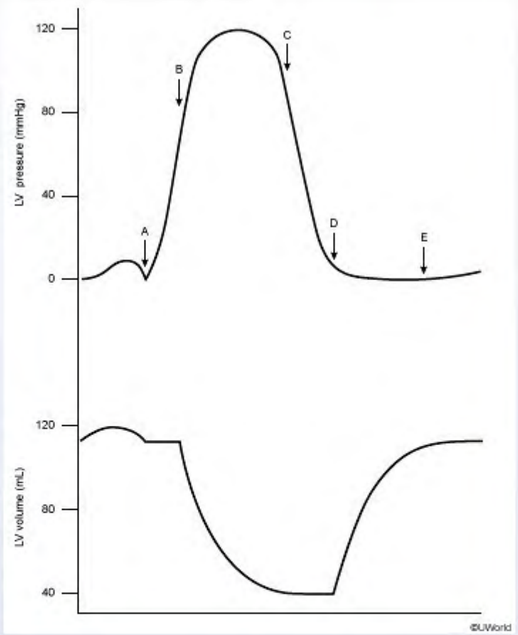

- A. A (7%)
- ✓ B. B (77%)
- ✗ C. C (8%)
- D. D (5%)
- E. E (0%)

Incorrect
Correct answer
B

77%
Answered correctly

13 secs
Time Spent

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D. D (5%)

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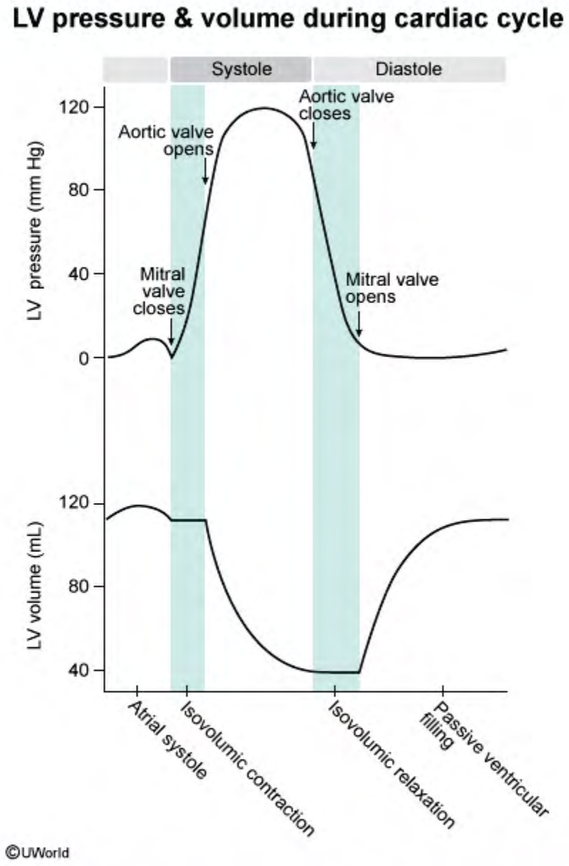

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This young, asymptomatic patient with a soft systolic ejection murmur at the right second intercostal space most likely has a **bicuspid aortic valve**.

The graph above depicts the normal left ventricular (LV) pressure and volume curves during the cardiac cycle. Ventricular pressure and volume curves allow one to identify the phases of the cardiac cycle and to determine the exact time of opening and closure of the cardiac valves. **Aortic valve opening** occurs when the LV pressure exceeds the central aortic pressure at the **end of isovolumetric contraction**. Subsequently, LV pressure continues to rise with forward blood flow into the aorta, until the pressure in the left ventricle falls below the aortic pressure leading to aortic valve closure.

(Choice A) The initial part of the LV pressure and volume curves corresponds to atrial systole, which results in a slight increase in LV volume and pressure. This is immediately followed by mitral valve closure and the beginning of isovolumetric contraction, where the LV pressure rises rapidly while LV volume remains constant.

(Choice C) This point corresponds to aortic valve closure, where LV pressure falls below the central aortic pressure. This also marks the beginning of isovolumetric relaxation, where the LV pressure rapidly falls while LV volume remains relatively constant.

(Choice D) Left atrial pressure gradually increases from passive filling from the pulmonary veins and exceeds LV pressure at the end of isovolumetric relaxation, leading to the opening of the mitral valves.

(Choice E) This point corresponds to passive LV filling with blood flow from the left atrium, which results in a gradual rise in LV volume and pressure.

Educational objective:
Ventricular pressure and volume curves allow one to identify the phases of the cardiac cycle and to determine the exact time of opening and closure of the cardiac valves. The aortic valve opens when left ventricular pressure exceeds the central aortic pressure at the end of isovolumetric contraction.

References

- The cardiac cycle and the physiological basis of left ventricular contraction, ejection, relaxation, and filling.

Physiology	Cardiovascular System	Cardiac physiology
Subject	System	Topic

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Educational objective:
Ventricular pressure and volume curves allow one to identify the phases of the cardiac cycle and to determine the exact time of opening and closure of the cardiac valves. The aortic valve opens when left ventricular pressure exceeds the central aortic pressure at the end of isovolumetric contraction.

References

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A 58-year-old man comes to the office due to progressive fatigue, dyspnea on exertion, and orthopnea. Medical history is significant for hypertension, hyperlipidemia, and a myocardial infarction 2 years ago. The patient says he has not taken his medications in several months because he was "feeling well." Blood pressure is 160/95 mm Hg and pulse is 94/min and regular. Physical examination shows bilateral lung crackles and lower extremity edema. The point of maximal impulse is displaced toward the axilla, and an S3 is heard. Serum creatinine is 0.9 mg/dL and serum potassium is 4.2 mEq/L. After this patient's condition has stabilized, long-term use of which of the following medications will most likely reduce mortality?

- A. Amlodipine
- B. Digoxin
- C. Diltiazem
- D. Furosemide
- E. Hydrochlorothiazide
- F. Lisinopril
- G. Triamterene

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- ✗ A. Amlodipine (2%)
- B. Digoxin (3%)
- C. Diltiazem (2%)
- D. Furosemide (8%)
- E. Hydrochlorothiazide (5%)
- ✓ F. Lisinopril (73%)
- G. Triamterene (3%)

Incorrect
Correct answer
F

73%
Answered correctly

03 secs
Time Spent

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Explanation

Pharmacotherapy for chronic heart failure with reduced ejection fraction	
Agent	**Mortality benefit**
Angiotensin receptor-neprilysin inhibitor (eg, sacubitril-valsartan)	

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Pharmacotherapy for chronic heart failure with reduced ejection fraction	
Agent	**Mortality benefit**
Angiotensin receptor-neprilysin inhibitor (eg, sacubitril-valsartan) OR **ACE inhibitor** (eg, lisinopril) OR **Angiotensin II receptor blocker** (eg, losartan)	Yes
Beta blocker (eg, metoprolol, carvedilol)	Yes
Mineralocorticoid receptor antagonist (eg, spironolactone, eplerenone)	Yes
Sodium-glucose cotransporter-2 inhibitor (eg, dapagliflozin, empagliflozin)	Yes
Diuretic (eg, furosemide, metolazone)	No*
Digoxin	No**
*Improves symptoms & reduces hospitalization. **Reduces hospitalization.	

This patient with a previous myocardial infarction has most likely developed chronic **heart failure with reduced ejection fraction** (HFrEF). Appropriate pharmacologic management is important in such patients because it has been shown to reduce mortality. Much of this **mortality benefit** is likely related to a **reduction** in deleterious

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Neurohormonal pathways involving norepinephrine, angiotensin II, and aldosterone all have a demonstrated role in cardiac remodeling. In particular, elevated angiotensin II and aldosterone levels increase pathologic cardiomyocyte hypertrophy and stimulate collagen deposition by fibroblasts (cardiac fibrosis). Accordingly, **ACE inhibitors** (eg, lisinopril), **angiotensin-receptor blockers** (eg, losartan), and **mineralocorticoid receptor antagonists** (eg, spironolactone, epleronone) reduce mortality in patients with HFrEF via a reduction in angiotensin II–mediated (and downstream aldosterone-mediated) cardiac remodeling.

Beta blockers (eg, metoprolol, carvedilol) also reduce mortality in patients with chronic HFrEF, likely through similar inhibition of neurohormonal mechanisms. Potentiation of the natriuretic peptide neurohormonal pathway with **angiotensin receptor-neprilysin inhibitor** therapy (eg, sacubitril-valsartan) has also demonstrated mortality benefit in these patients.

(Choice A) Blood pressure control is important for patients with heart failure because it slows the progression of coronary artery disease and may reduce cardiac remodeling that is triggered by increased left ventricular pressure load. However, aside from the benefit of blood pressure reduction, dihydropyridine calcium channel blockers (eg, amlodipine) provide no additional mortality benefit in patients with HFrEF.

(Choice B) Digoxin is used as an adjunctive therapy in patients with HFrEF. It improves symptoms, thereby reducing the rate of hospitalization, but does not improve mortality.

(Choice C) Nondihydropyridine calcium channel blockers (eg, verapamil, diltiazem) can depress cardiac contractility and are usually contraindicated in patients with HFrEF.

(Choices D and E) Loop (eg, furosemide) and thiazide (eg, metolazone) diuretics reduce cardiac preload and are useful for symptomatic management of patients with HFrEF; however, these drugs do not provide a clear mortality benefit.

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(Choice G) Although potassium-sparing diuretics that function via blockade of mineralocorticoid receptors have shown mortality benefit for HFrEF, those that act via blockade of the epithelial sodium channel (ENaC) (eg, triamterene, amiloride) have minimal neurohormonal effects and no demonstrated benefit.

Educational objective:
Neurohormones (eg, norepinephrine, angiotensin II, and aldosterone) play a large role in the deleterious cardiac remodeling that occurs in heart failure with reduced ejection fraction. ACE inhibitors, angiotensin-receptor blockers, mineralocorticoid receptor antagonists, beta blockers, and angiotensin receptor-neprilysin inhibitors reduce mortality in these patients by reducing neurohormonal-mediated cardiac remodeling.

References

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A 54-year-old man comes to the office due to new-onset muscle cramps. The patient has a history of hypertension and was started on hydrochlorothiazide 4 weeks ago. Blood pressure is 138/86 mm Hg and pulse is 78/min. Examination shows no abnormalities. Based on his most recent laboratory results, triamterene is added to the current therapy. Which of the following best describes the mechanism of action of this medication?

- A. Blocking renal tubular epithelial sodium channels
- B. Decreased aldosterone production
- C. Increased intestinal absorption of potassium
- D. Inhibiting aldosterone receptor activity in renal collecting duct
- E. Inhibiting Na+/K+ ATPase activity in skeletal muscle

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- ✓ A. Blocking renal tubular epithelial sodium channels (61%)
- B. Decreased aldosterone production (2%)
- C. Increased intestinal absorption of potassium (7%)
- D. Inhibiting aldosterone receptor activity in renal collecting duct (25%)
- E. Inhibiting Na+/K+ ATPase activity in skeletal muscle (2%)

Correct

61% Answered correctly

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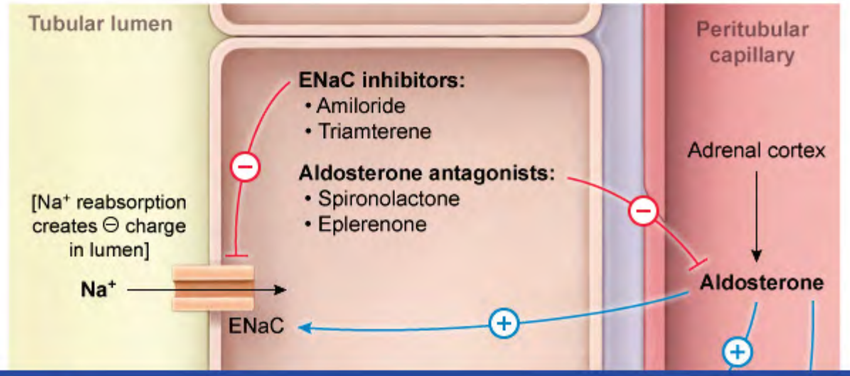
Explanation

Action of aldosterone in the collecting duct of the nephron



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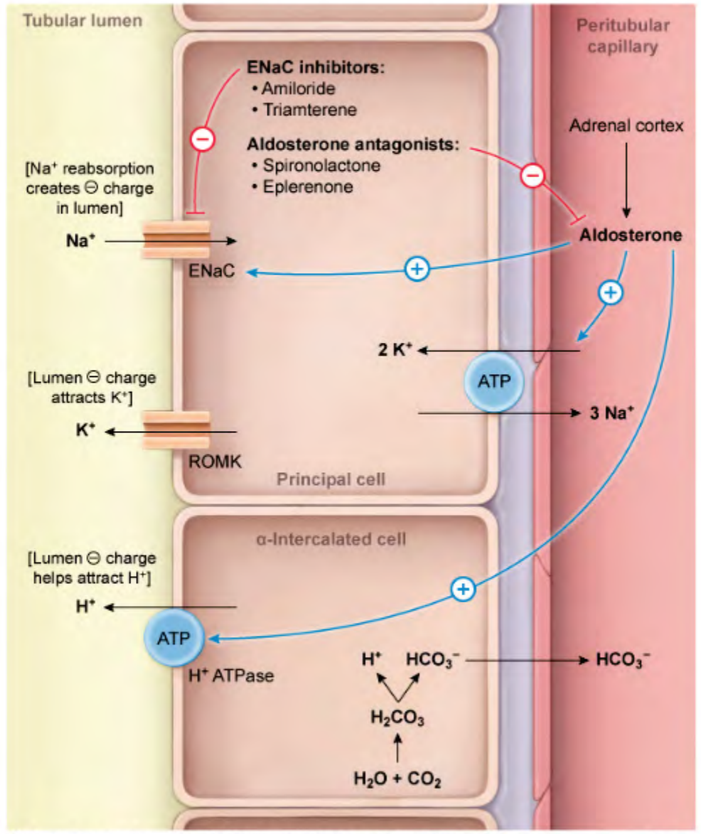

ENaC = epithelial sodium channel; **ROMK** = renal outer medullary potassium channel. ©UWorld

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ENaC = epithelial sodium channel; **ROMK** = renal outer medullary potassium channel. ©UWorld

Thiazide diuretics lower blood pressure by inhibiting Na^+/Cl^- cotransporters in the distal convoluted tubules, thereby decreasing reabsorption of Na^+ and Cl^-. Both thiazides and loop **diuretics induce volume loss** with subsequent activation of the renin-angiotensin-aldosterone system. Aldosterone increases renal sodium resorption in exchange for potassium by upregulating the synthesis and activity of the following:

- Epithelial sodium channels: Found on the apical membrane of principal cells, it absorbs Na+ from the tubular lumen
- Na+/K+-ATPase pump: Located on the basal membrane of principal cells, it extrudes Na+ into the interstitial space in exchange for potassium

Because of these effects, **increased aldosterone** levels commonly result in **hypokalemia**, which can lead to muscle cramps, weakness, and (when severe) cardiac arrhythmias.

Potassium-sparing diuretics may be added to **prevent hypokalemia**. These weak diuretic medications act in the distal nephron, where they reduce potassium excretion via the following mechanisms:

- **Triamterene** and **amiloride** directly **inhibit the epithelial sodium channel**, preventing sodium from entering principal cells. This reduces the electrochemical gradient (ie, negative luminal charge) that helps drive potassium secretion.
- Aldosterone antagonists (ie, spironolactone, eplerenone) directly inhibit the mineralocorticoid receptor, resulting in decreased formation and activity of epithelial sodium channels and Na+/K+-ATPase pumps **(Choice D)**.

(Choice B) ACE inhibitors (eg, lisinopril) and angiotensin II blockers (eg, losartan) promote potassium retention by inhibiting the effects of angiotensin II, thereby reducing aldosterone production.

(Choice C) Potassium supplements increase total potassium absorption by the gastrointestinal tract. They are another type of medication commonly prescribed to treat hypokalemia in patients on loop or thiazide diuretics.

(Choice E) Digoxin, an antiarrhythmic medication, inhibits the Na+-K+-ATPase in cardiac and skeletal muscles and can also cause hyperkalemia. However, it is never prescribed to prevent hypokalemia.

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Educational objective:
Potassium-sparing diuretics are often added to loop or thiazide diuretics to prevent hypokalemia. Amiloride and triamterene directly inhibit the epithelial sodium channel, preventing sodium from entering the principal cell, which reduces the electrochemical gradient (ie, negative luminal charge) that helps drive potassium secretion.

Pharmacology	Cardiovascular System	Hypokalemia
Subject	System	Topic

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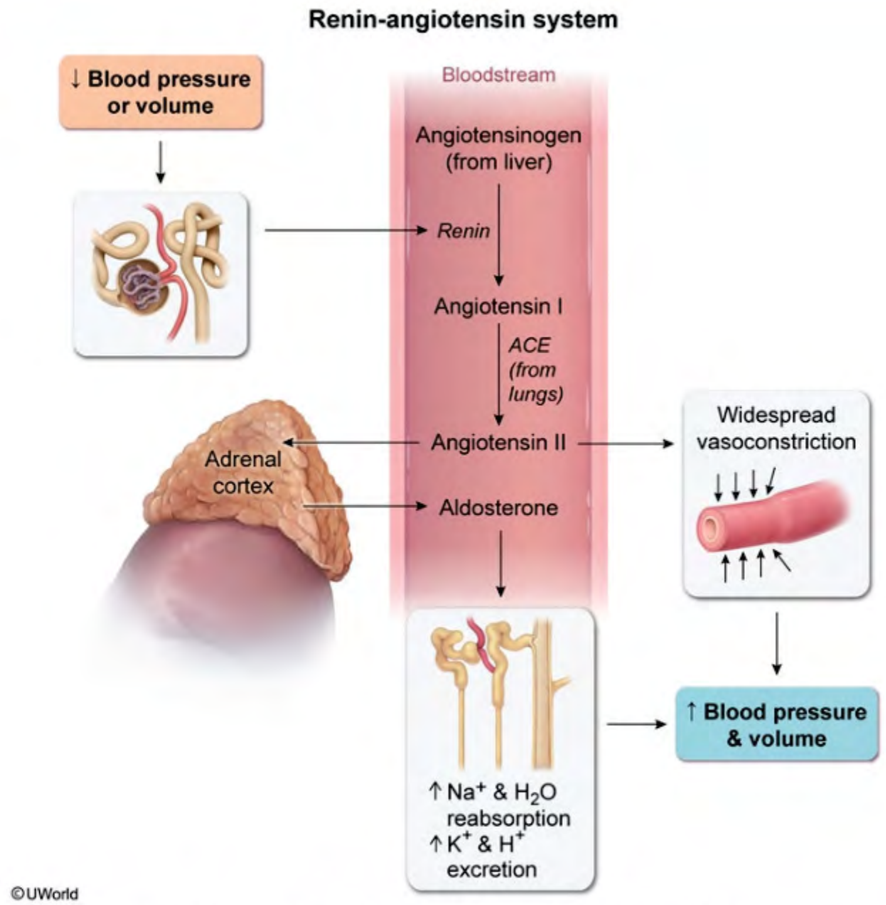

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(Choice C) Potassium supplements increase total potassium absorption by the gastrointestinal tract. They are

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A 56-year-old woman with unknown medical history is brought to the emergency department in an unresponsive state. Paramedics were called for sudden-onset, left-sided weakness, and when they arrived at the patient's house she was having a generalized tonic-clonic seizure. Antiseizure medications were given and the patient was subsequently intubated. Evaluation shows a comatose woman with left hemiplegia. Neuroimaging reveals a large infarction in the right middle cerebral artery territory with cerebral edema. Despite aggressive interventions, the patient dies from brain herniation. Autopsy reveals multiple small, nondestructive masses attached to the edges of the mitral valve leaflets. Microscopy shows that these masses are composed of platelet-rich thrombi, but no organisms are present. Which of the following is most closely associated with this patient's heart valve findings?

- A. Advanced malignancy
- B. Chronic liver failure
- C. Dilated cardiomyopathy
- D. Intravenous drug use
- E. Large-vessel vasculitis

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- ✓ A. Advanced malignancy (37%)
- ✗ B. Chronic liver failure (6%)
- C. Dilated cardiomyopathy (22%)
- D. Intravenous drug use (10%)
- E. Large-vessel vasculitis (23%)

Incorrect
Correct answer
A

37%
Answered correctly

03 secs
Time Spent

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Version

Explanation

The autopsy finding of **sterile platelet-rich thrombi** attached to the mitral valve leaflets is characteristic of **nonbacterial thrombotic endocarditis** (NBTE) (marantic endocarditis). The pathogenesis of NBTE is thought to begin with valvular endothelial injury caused by circulating inflammatory cytokines, which triggers platelet deposition in the presence of an underlying **hypercoagulable state**. NBTE is most commonly associated with **advanced malignancy** (especially mucinous adenocarcinoma) and systemic lupus erythematosus (Libman-Sacks endocarditis); less commonly, it can occur with antiphospholipid syndrome, disseminated intravascular coagulation, and extensive burns

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Explanation

The autopsy finding of **sterile platelet-rich thrombi** attached to the mitral valve leaflets is characteristic of **nonbacterial thrombotic endocarditis** (NBTE) (marantic endocarditis). The pathogenesis of NBTE is thought to begin with valvular endothelial injury caused by circulating inflammatory cytokines, which triggers platelet deposition in the presence of an underlying **hypercoagulable state**. NBTE is most commonly associated with **advanced malignancy** (especially mucinous adenocarcinoma) and systemic lupus erythematosus (Libman-Sacks endocarditis); less commonly, it can occur with antiphospholipid syndrome, disseminated intravascular coagulation, and extensive burns.

Histologically, NBTE vegetations consist of bland thrombus with strands of fibrin, immune complexes, and mononuclear cells (white thrombus). The vegetations typically affect the left-sided heart valves (mitral or aortic) and are often asymptomatic; however, **systemic embolization** (eg, stroke, acute limb ischemia) can occur and is the most common presentation of NBTE.

(Choice B) Chronic liver failure (ie, cirrhosis) can be complicated by portal vein thrombosis; however, it is not associated with NBTE.

(Choice C) Dilated cardiomyopathy is characterized by ventricular enlargement and reduced systolic function. It can occasionally lead to left ventricular mural thrombus with risk of systemic embolization, but it is not associated with NBTE.

(Choice D) Intravenous drug use increases the risk of bacterial endocarditis, not NBTE. Injected particulate matter (eg, dirt) is thought to cause microdamage to the right-sided heart valves before being filtered out by the lungs, facilitating bacterial attachment to primarily the tricuspid valve.

(Choice E) Temporal (giant cell) arteritis, a large-vessel vasculitis that typically presents with headaches and fever, is associated with an increased risk of venous thromboembolic disease. Although associated NBTE has been reported, it is quite rare.

Educational objective:
Nonbacterial thrombotic endocarditis is characterized by valvular deposition of sterile platelet-rich thrombi. It likely results from valvular damage due to inflammatory cytokines in the setting of an underlying hypercoagulable state

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nonbacterial thrombotic endocarditis (NBTE) (marantic endocarditis). The pathogenesis of NBTE is thought to begin with valvular endothelial injury caused by circulating inflammatory cytokines, which triggers platelet deposition in the presence of an underlying **hypercoagulable state**. NBTE is most commonly associated with **advanced malignancy** (especially mucinous adenocarcinoma) and systemic lupus erythematosus (Libman-Sacks endocarditis); less commonly, it can occur with antiphospholipid syndrome, disseminated intravascular coagulation, and extensive burns.

Histologically, NBTE vegetations consist of bland thrombus with strands of fibrin, immune complexes, and mononuclear cells (white thrombus). The vegetations typically affect the left-sided heart valves (mitral or aortic) and are often asymptomatic; however, **systemic embolization** (eg, stroke, acute limb ischemia) can occur and is the most common presentation of NBTE.

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(Choice E) Temporal (giant cell) arteritis, a large-vessel vasculitis that typically presents with headaches and fever, is associated with an increased risk of venous thromboembolic disease. Although associated NBTE has been reported, it is quite rare.

Educational objective:
Nonbacterial thrombotic endocarditis is characterized by valvular deposition of sterile platelet-rich thrombi. It likely results from valvular damage due to inflammatory cytokines in the setting of an underlying hypercoagulable state, and it is most commonly seen with advanced malignancy (especially mucinous adenocarcinoma) or systemic lupus erythematosus.

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A 32-year-old man is evaluated in the emergency department due to fever, night sweats, and chills over the last several days. The patient has been using intravenous drugs recently as he is "stressed out." He has otherwise been in good health with no medical problems. Temperature is 38.3 C (101 F), blood pressure is 120/80 mm Hg, and pulse is 105/min and regular. Further evaluation reveals aortic valve endocarditis with an intracardiac abscess and small fistula formation between the aortic root and right ventricle. Doppler ultrasound interrogation of the fistula will most likely reveal which of the following blood flow patterns?

- A. Flow from the aortic root to the right ventricle continuously
- B. Flow from the aortic root to the right ventricle only in diastole
- C. Flow from the aortic root to the right ventricle only in systole
- D. Flow from the right ventricle to the aortic root continuously
- E. Flow from the right ventricle to the aortic root only in diastole
- F. Flow from the right ventricle to the aortic root only in systole

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- ✓ A. Flow from the aortic root to the right ventricle continuously (41%)
- ✗ B. Flow from the aortic root to the right ventricle only in diastole (15%)
- C. Flow from the aortic root to the right ventricle only in systole (28%)
- D. Flow from the right ventricle to the aortic root continuously (4%)
- E. Flow from the right ventricle to the aortic root only in diastole (3%)
- F. Flow from the right ventricle to the aortic root only in systole (6%)

Incorrect
Correct answer
A

41%
Answered correctly

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Explanation

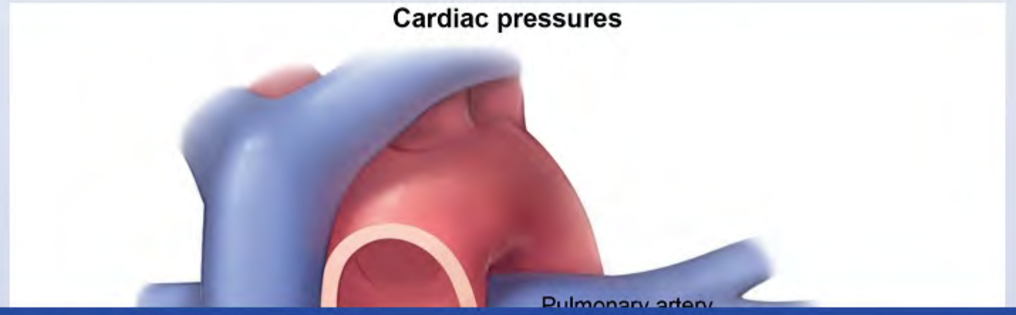

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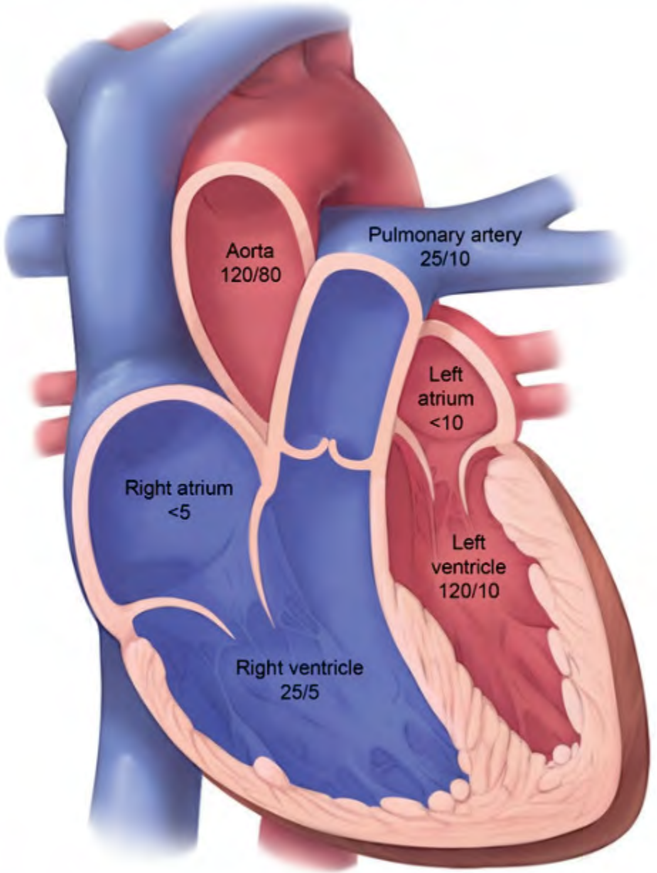
Exhibit Display

Cardiac pressures



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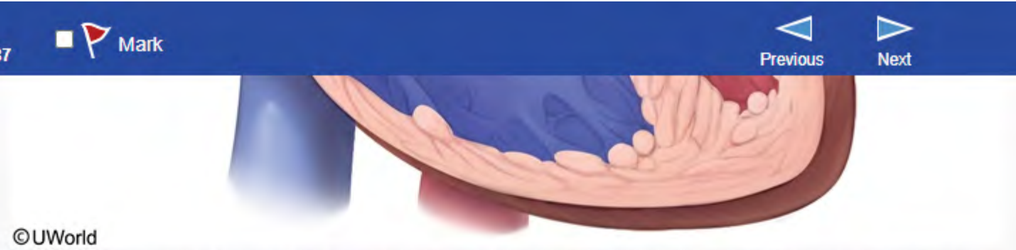

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This patient with aortic valve endocarditis has developed an intracardiac fistula between the aortic root and right ventricle. **Aortocavitary fistulas** are an uncommon complication of **bacterial endocarditis** caused by extension of the infection from the valve to the adjacent myocardium.

Echocardiography with Doppler analysis can be used to detect and quantify shunts in patients with intracardiac fistulas. During the normal cardiac cycle, central aortic pressure (eg, 120/80 mm Hg) is higher than right ventricular pressure (eg, 25/5 mm Hg) during systole and diastole. Consequently, in patients with aortocavitary fistula, Doppler interrogation will most likely demonstrate **continuous blood flow** from the **higher-pressure aortic root** to the lower-pressure **right ventricle** (left-to-right cardiac shunt) **(Choices B and C)**. This can lead to a continuous murmur heard on cardiac auscultation.

(Choices D, E, and F) Pressure in the aortic root is always greater than in the right ventricle throughout systole and diastole; therefore, blood flow from the right ventricle to the aortic root would not be expected in this patient.

Educational objective:
During the normal cardiac cycle, central aortic pressure is higher than right ventricular pressure during systole and diastole. Consequently, an intracardiac fistula between the aortic root and right ventricle will most likely demonstrate a left-to-right cardiac shunt as blood continuously flows from the aortic root (high pressure) to the right ventricle (low pressure).

References

- Aorto-cavitary fistulous tract formation in infective endocarditis: clinical and echocardiographic features of 76 cases and risk factors for mortality.

Physiology	Cardiovascular System	Endocarditis
Subject	System	Topic

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An 85-year-old man is transferred to the hospital from a nursing home due to fever and confusion. On arrival, the patient is admitted directly to the intensive care unit with a presumptive diagnosis of septic shock. Antibiotic therapy is initiated. The patient is unable to provide any history, but his caretakers state that he has had nonspecific symptoms, including fever, for the past few days. The patient has a history of cardiovascular disease, diverticulitis, and dementia. Blood pressure is 60/40 mm Hg despite aggressive intravenous hydration. Intravenous norepinephrine is administered in response to the patient's hypotension. Which of the following cellular changes is most likely to occur in direct response to the medication?

- A. cAMP decrease in bronchial smooth muscle cells
- B. cAMP increase in cardiac muscle cells
- C. cAMP increase in vascular smooth muscle cells
- D. DAG decrease in vascular smooth muscle cells
- E. IP_3 increase in cardiac muscle cells

Submit

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- A. cAMP decrease in bronchial smooth muscle cells (2%)
- ✓ B. cAMP increase in cardiac muscle cells (38%)
- C. cAMP increase in vascular smooth muscle cells (35%)
- D. DAG decrease in vascular smooth muscle cells (9%)
- E. IP_3 increase in cardiac muscle cells (13%)

Correct

38% Answered correctly

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Explanation

Receptor stimulated	Change in second messenger	Primary effects
Alpha-1	↑ IP_3	• Peripheral vasoconstriction • Urethral constriction • Pupillary dilation
Alpha-2	↓ cAMP	• CNS sympatholytic • ↓ insulin release & intestinal motility

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Receptor stimulated	Change in second messenger	Primary effects
Alpha-1	↑ IP_3	• Peripheral vasoconstriction • Urethral constriction • Pupillary dilation
Alpha-2	↓ cAMP	• CNS sympatholytic • ↓ insulin release & intestinal motility
Beta-1	↑ cAMP	• ↑ Cardiac contractility & heart rate • ↑ Renin release by JG cells of kidney
Beta-2	↑ cAMP	• Peripheral vasodilation • Bronchodilation
Muscarinic-2	↓ cAMP	• ↓ Cardiac contractility & heart rate
Muscarinic-3	↑ IP_3	• Bronchoconstriction • ↑ insulin release & intestinal motility • Bladder contraction • Pupillary constriction • Peripheral vasodilation*

*Via release of nitric oxide.
cAMP = cyclic AMP; **IP_3** = inositol 1,4,5-triphosphate; **JG** = juxtaglomerular.

Stimulation of adrenoreceptors leads to a change in concentration of a second messenger (eg, cAMP), which mediates the effects on the target organ. **Norepinephrine**, an adrenergic agonist, is the preferred pharmacologic treatment for **septic shock** as it predominately stimulates a1, a2, and B1 receptors, with little influence on B2 receptors. This leads to the following clinical effects:

1. Stimulation of **α1 adrenoreceptors** leads to **vasoconstriction** in the skin and viscera, resulting in increased systolic and diastolic blood pressure with decreased renal and hepatic blood flow. α1-mediated

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1. Stimulation of **α1 adrenoreceptors** leads to **vasoconstriction** in the skin and viscera, resulting in increased systolic and diastolic blood pressure with decreased renal and hepatic blood flow. α1-mediated vasoconstriction occurs via the IP_3-second messenger pathway.
2. Stimulation of **β1 adrenoreceptors** within the heart increases **cAMP** concentrations through G_s-mediated receptor coupling, which leads to increased **cardiac contractility**, **conduction**, and **heart rate**. However, the direct effect of norepinephrine on heart rate is counteracted by an indirect, baroreceptor-mediated reflex bradycardia that occurs following the increase in peripheral resistance. The combined result of these effects is often an unchanged or even decreased heart rate.
3. Stimulation of α2 adrenoreceptors causes a decrease in cAMP in pancreatic β cells and in the intestines, resulting in decreased insulin secretion and reduced intestinal motility. Central α2 adrenoreceptors are not stimulated by intravenous administration of norepinephrine as the molecule does not cross the blood-brain barrier.

(Choice A) Stimulation of β2 adrenoreceptors in bronchial smooth muscle cells leads to increased, rather than decreased, cAMP synthesis, resulting in bronchodilation. Regardless, norepinephrine does not significantly stimulate β2 adrenoreceptors.

(Choices C and D) Norepinephrine-mediated stimulation of α1 adrenoreceptors increases, rather than decreases, IP_3 (increased calcium release) and DAG concentrations in vascular smooth muscle cells. cAMP is increased after β2 adrenoreceptors are stimulated; however, because norepinephrine does not significantly stimulate β2 adrenoreceptors, cAMP concentrations in vascular smooth muscle are not increased.

(Choice E) A significant elevation in IP_3 concentration does not occur in cardiac muscle cells following

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2. Stimulation of **β1 adrenoreceptors** within the heart increases **cAMP** concentrations through G_s-mediated receptor coupling, which leads to increased **cardiac contractility**, **conduction**, and **heart rate**. However, the direct effect of norepinephrine on heart rate is counteracted by an indirect, baroreceptor-mediated reflex bradycardia that occurs following the increase in peripheral resistance. The combined result of these effects is often an unchanged or even decreased heart rate.

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(Choice E) A significant elevation in IP_3 concentration does not occur in cardiac muscle cells following norepinephrine administration. The cardiac effects of norepinephrine are primarily mediated by β1 adrenoreceptors that are linked to a cAMP, rather than IP_3, signal transduction pathway.

Educational objective:
Norepinephrine stimulates cardiac β1 adrenoreceptors, which increases cAMP concentration within cardiac myocytes and leads to increased contractility, conduction, and heart rate. Peripheral vasoconstriction occurs via stimulation of α1 adrenoreceptors in vascular smooth muscle cells and activation of an IP_3 signaling pathway.

References

- Early administration of norepinephrine increases cardiac preload and cardiac output in septic patients with life-threatening hypotension

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Muscarinic-3	↑ IP_3	• Bladder contraction

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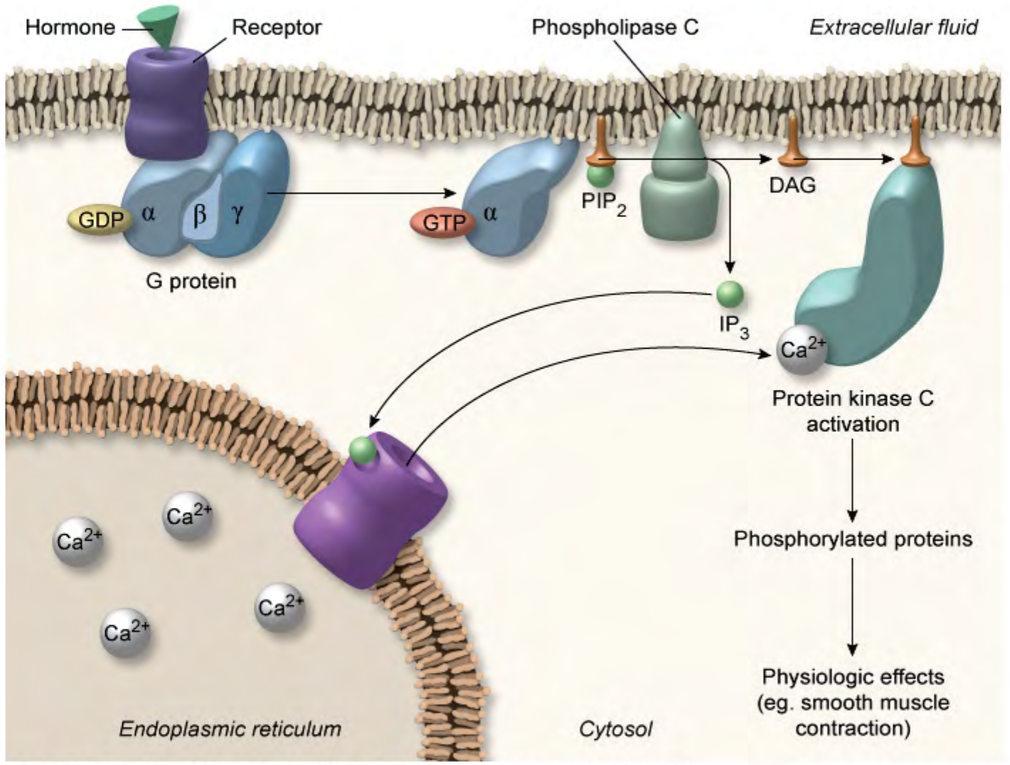
Phosphatidylinositol 2nd messenger system



DAG = diacylglycerol; **GDP** = guanosine 5′-diphosphate; **GTP** = guanosine 5′-triphosphate; **IP_3** = inositol 1,4,5-trisphosphate; **PIP_2** = phosphatidylinositol 4,5-bisphosphate.

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Muscarinic-3	↑ IP_3	• Bladder contraction

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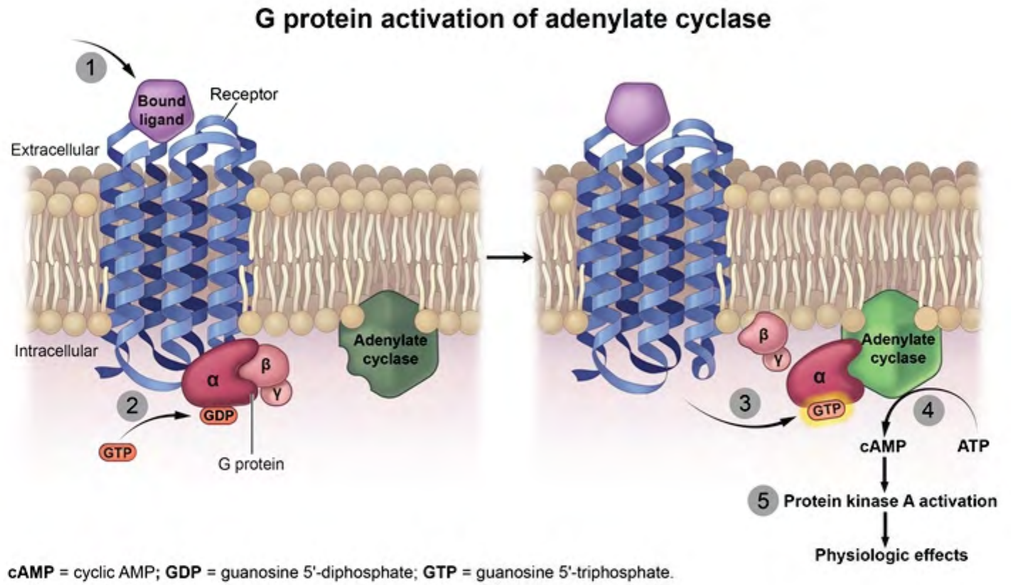

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A 58-year-old man comes to the emergency room due to headache. Medical history is significant for long-standing hypertension and medication nonadherence. Blood pressure on arrival is 231/135 mm Hg. Twenty minutes after intravenous hydralazine is administered, blood pressure is 145/95 mm Hg. Over the following 24 hours, serum creatinine increases from 1.1 to 1.6 mg/dL. Urine sediment demonstrates abundant granular casts. Which of the following processes best explains this patient's acute kidney injury?

- A. Alteration of the blood pressure-flow relationship in renal arterioles
- B. Drug-induced glomerular injury due to immune complex formation
- C. Drug-induced renal interstitial inflammation
- D. Drug-induced renal tubular injury due to crystal deposition
- E. Prerenal azotemia due to a global reduction in cardiac output

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- ✓ A. Alteration of the blood pressure-flow relationship in renal arterioles (44%)
- ✗ B. Drug-induced glomerular injury due to immune complex formation (10%)
- C. Drug-induced renal interstitial inflammation (11%)
- D. Drug-induced renal tubular injury due to crystal deposition (12%)
- E. Prerenal azotemia due to a global reduction in cardiac output (21%)

Incorrect
Correct answer
A

44%
Answered correctly

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Time Spent

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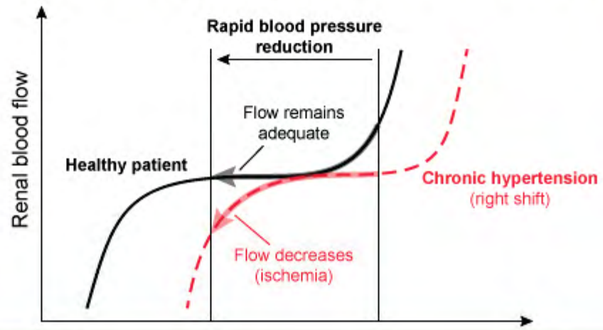
Explanation

Autoregulation of blood flow in chronic hypertension



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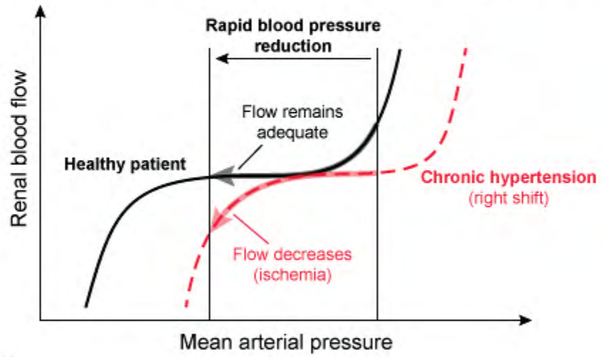
Autoregulation of blood flow in chronic hypertension



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(ischemia)

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This patient with chronic hypertension has hypertensive urgency. He received hydralazine, a fast-acting antihypertensive agent. After abrupt normalization of blood pressure (BP), he developed acute tubular necrosis (ATN) (granular casts). This sequence of events is consistent with **normotensive ischemia** (ie, inadequate renal perfusion despite a normal BP).

Blood flow autoregulation allows organs to receive the same perfusion (flow) across a wide range of pressure. In the kidneys, blood flow autoregulation involves 2 key mechanisms:

- **Myogenic response:** Afferent glomerular arterioles reflexively constrict when they sense greater stretch forces (high BP).
- **Tubuloglomerular feedback:** High arterial pressure causes hyperfiltration (↑ glomerular filtration rate), increasing sodium and chloride delivery to the macula densa, which secretes vasoactive mediators (eg, adenosine) to constrict the *afferent* arterioles

The autoregulation response protects organs from dangerous, persistent BP elevations. In **chronic hypertension**, there is **baseline constriction** of afferent glomerular arterioles. This causes the entire renal autoregulation curve to be **shifted to the right**: a hypertensive patient's kidneys **receive less blood flow at any given BP** relative to those of a healthy patient without hypertension. Quickly lowering the BP to normal (eg, 145/95 mm Hg) causes a steep drop in blood flow, leading to ischemic ATN; as a result, BP should be lowered gently, targeting above-normal values (eg, 25% reduction over several hours).

(Choice B) Drug-induced lupus, a rare adverse effect of hydralazine, causes acute kidney injury (AKI) due to immune complex–mediated glomerulonephritis. However, an active urine sediment (eg, red cells, white cells, casts) and other systemic signs of lupus (eg, rash, pleuritis) would be expected.

(Choice C) Drug-induced allergic interstitial nephritis is an eosinophilic AKI usually triggered by beta-lactam antibiotics and nonsteroidal anti-inflammatory drugs. It occurs after continual exposure (eg, several days), and eosinophiluria is usually present.

(Choice D) Some drugs (eg, acyclovir, indinavir) can precipitate in the urine, causing AKI due to crystalluria. Urine sediment may show tubular necrosis (eg, granular casts), but crystals are usually also seen.

(Choice E) Decreased cardiac output can cause prerenal AKI, which may evolve to ischemic ATN. However,

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increasing sodium and chloride delivery to the macula densa, which secretes vasoactive mediators (eg, adenosine) to constrict the *afferent* arterioles

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(Choice E) Decreased cardiac output can cause prerenal AKI, which may evolve to ischemic ATN. However, treatment with hydralazine lowers the systemic vascular resistance, which reduces cardiac afterload. This will lead to an increase in overall cardiac output.

Educational objective:
In patients with long-standing hypertension, a chronic autoregulatory shift in the blood pressure–flow relationship occurs, affording less perfusion (flow) at any given pressure. Therefore, excessively rapid correction of blood pressure toward normal may induce relative ischemia.

Pathophysiology	Cardiovascular System	Acute kidney injury
Subject	System	Topic

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A 65-year-old man with a history of coronary artery disease comes to the physician complaining of progressive exertional shortness of breath, fatigue, and lower extremity swelling. His medical history is significant for a myocardial infarction 5 years ago and an electronic pacemaker implanted 2 years ago. His chest x-ray is shown below. A segment of one of the leads is highlighted (arrow).

The highlighted segment most likely lies within which of the following structures?

- A. Anterior interventricular sulcus
- B. Atrioventricular groove
- C. Pulmonary artery
- D. Right atrium
- E. Right ventricle

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A 65-year-old man with a history of coronary artery disease comes to the physician complaining of progressive exertio
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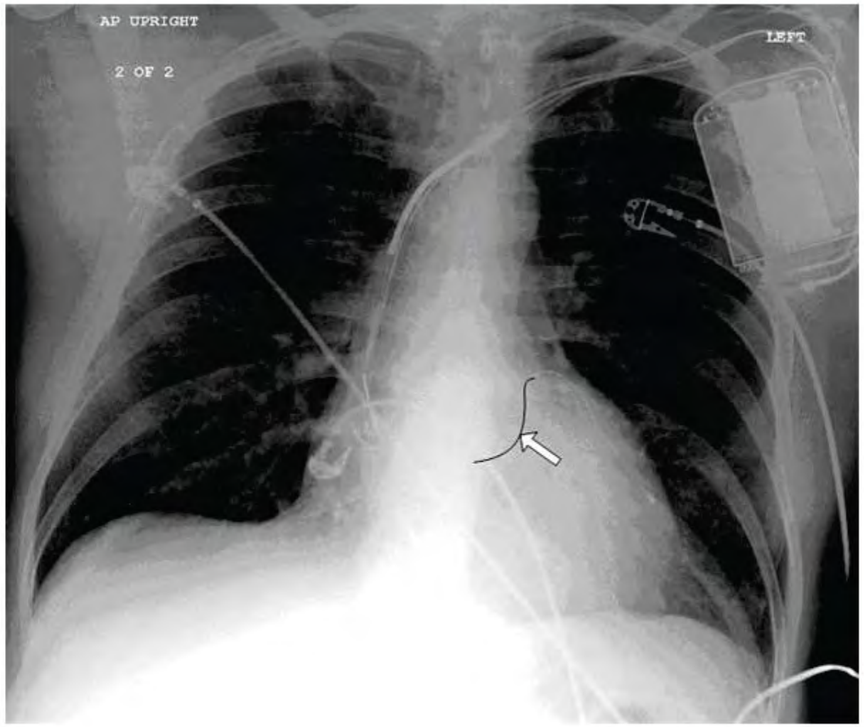
below.

Exhibit Display



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B. Atrioventricular groove
C. Pulmonary artery
D. Right atrium

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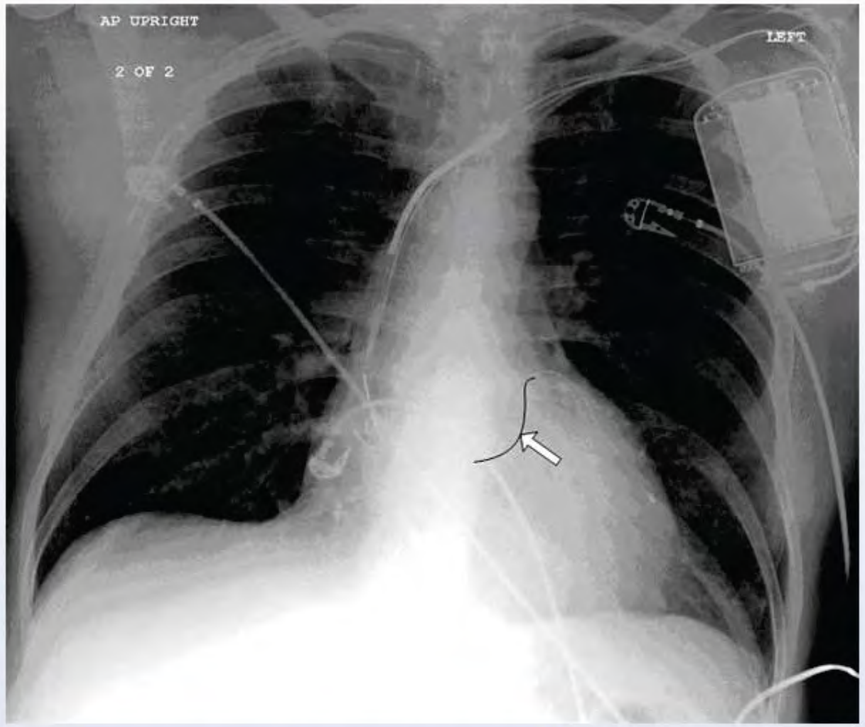
below. A segment of one of the leads is highlighted (arrow).



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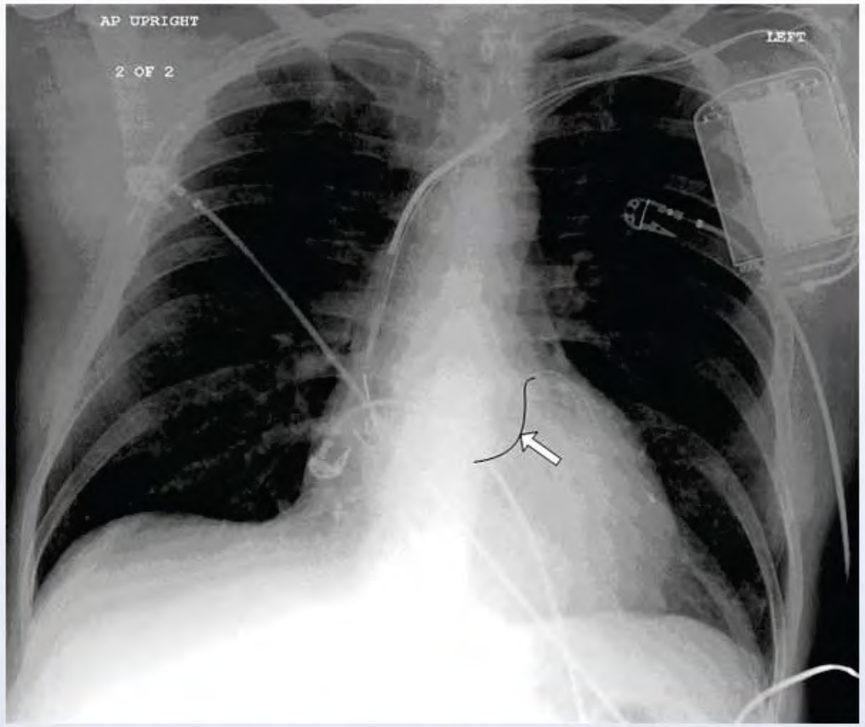

The highlighted segment most likely lies within which of the following structures?

- A. Anterior interventricular sulcus (7%)
- ✓ B. Atrioventricular groove (53%)
- C. Pulmonary artery (7%)
- D. Right atrium (20%)
- E. Right ventricle (10%)

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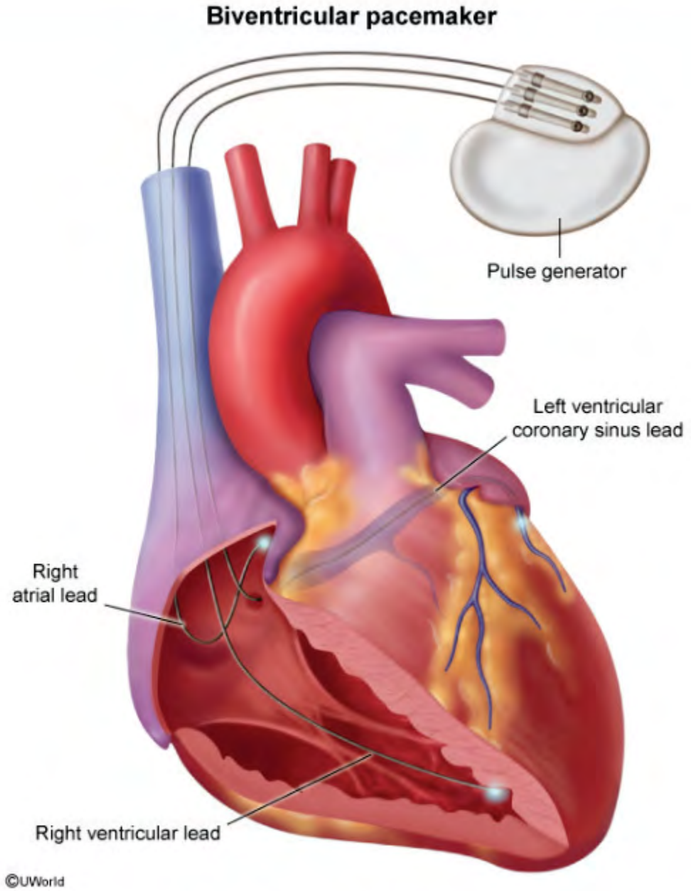

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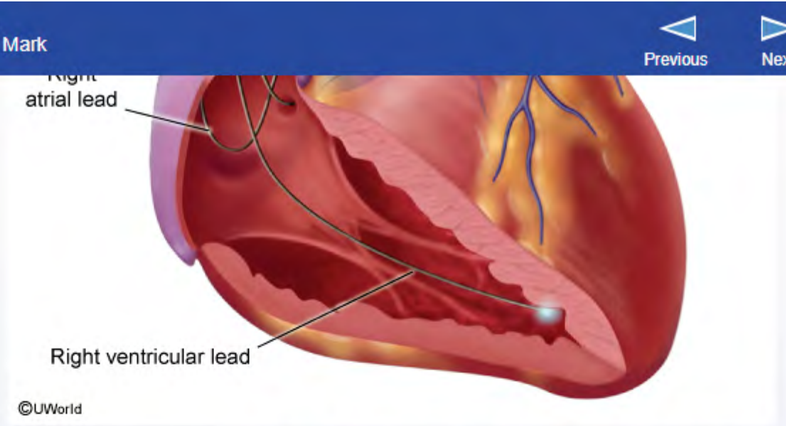

This patient has a biventricular pacemaker, a device that requires 2 or 3 leads. If 3 leads are used, the first 2 are placed in the right atrium and right ventricle. The third lead is used to pace the left ventricle. Right atrial and ventricular leads are easy to place as they only need to traverse the left subclavian vein and superior vena cava to reach these cardiac chambers. In contrast, the lead that paces the left ventricle is more difficult to position. The preferred transvenous approach involves passing the left ventricular pacing lead from the right atrium into the coronary sinus, which resides in the atrioventricular groove on the posterior aspect of the heart. It is then advanced into one of the lateral venous tributaries in order to optimize left ventricular pacing.

(Choice A) The anterior interventricular sulcus courses toward the apex of the heart on its anterior surface. The anterior descending vessels lie in this sulcus.

(Choice C) The pulmonary artery lies just to the left of center on the anterior surface of the heart. It courses from the right ventricle toward the aortic arch. Swan-Ganz catheters traverse the pulmonary artery.

(Choices D & E) The right atrium and ventricle are visible on the rightmost section of the cardiac silhouette.

Educational objective:
Left ventricular leads in biventricular pacemakers course through the coronary sinus, which resides in the atrioventricular groove on the posterior aspect of the heart.

Anatomy	Cardiovascular System	Implantable cardioverter defibrillator
Subject	System	Topic

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A 24-year-old woman comes to the physician with a 3-week history of progressive fatigue. Physical examination shows lesions involving the nail beds, as seen in the photograph below.

Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

- A. Neck palpation
- B. Lung auscultation
- C. Cardiac auscultation
- D. Abdominal palpation
- E. Rectal examination

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A 24-year-old woman comes to the physician with a 3-week history of progressive fatigue. Physical examination shows

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E. Rectal examination

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Further evaluation would most likely reveal additional abnormalities during which part of the physical examination?

- A. Neck palpation (1%)
- B. Lung auscultation (4%)
- ✓ C. Cardiac auscultation (89%)
- D. Abdominal palpation (3%)
- E. Rectal examination (0%)

Correct

89%
Answered correctly

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Explanation

Exhibit Display

Splinter hemorrhages

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These
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microemboli in bacterial endocarditis. Patients with suspected IE should therefore be evaluated for a regurgitant murmur during cardiac auscultation.

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These lesions are subungual splinter hemorrhages, splinter- or flame-shaped hemorrhagic streaks in the nail bed that appear as a consequence of microemboli. Janeway lesions (small, macular, erythematous or hemorrhagic, nontender lesions on the palms and soles) are also a sign of microembolism. Infectious endocarditis (IE) is the usual cause of such microemboli, and progressive fatigue may be the presenting symptom of subacute bacterial endocarditis. Chronically destructive, infected vegetations involving the cardiac valves are the source of the microemboli in bacterial endocarditis. Patients with suspected IE should therefore be evaluated for a regurgitant murmur during cardiac auscultation.

(Choices A, B, and D) Unless the primary source of the bacteremia initiating the IE is a focal neck, pulmonary, or abdominal infection, one would not expect to find any abnormalities in these systems.

(Choice E) Unless the primary source of the original bacteremia was a peri-rectal infection or necrotic, infected rectal neoplasm, one would not expect to find any abnormalities on rectal examination.

Educational objective:
Microemboli from the valvular vegetations of bacterial endocarditis are the most common cause of subungual splinter hemorrhages. The presence of these lesions necessitates careful cardiac auscultation to detect a possible new-onset regurgitant murmur.

Pathology	Cardiovascular System	Endocarditis
Subject	System	Topic

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An 82-year-old woman comes to the office due to fatigue, abdominal discomfort, and lower extremity swelling. She also occasionally feels an uncomfortable pulsation in her neck. Six weeks ago, the patient underwent pacemaker implantation due to sick sinus syndrome. Episodes of lightheadedness that she had experienced before the procedure have now resolved. The patient is afebrile, and other vital signs are normal. Physical examination reveals bilateral, lower extremity edema and a tender, pulsatile liver. The lungs are clear on auscultation. The pacemaker implantation site has healed normally. Complete blood count is normal. What is the most likely diagnosis?

- A. Aortic valve regurgitation
- B. Constrictive pericarditis
- C. Infective endocarditis
- D. Pulmonic valve regurgitation
- E. Tricuspid valve regurgitation

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- ✗ A. Aortic valve regurgitation (15%)
- B. Constrictive pericarditis (13%)
- C. Infective endocarditis (3%)
- D. Pulmonic valve regurgitation (6%)
- ✓ E. Tricuspid valve regurgitation (60%)

Incorrect
Correct answer
E

60%
Answered correctly

03 secs
Time Spent

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Explanation

Dual chamber pacemaker

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An 82-year-old woman comes to the office due to fatigue, abdominal discomfort, and lower extremity swelling. She also o
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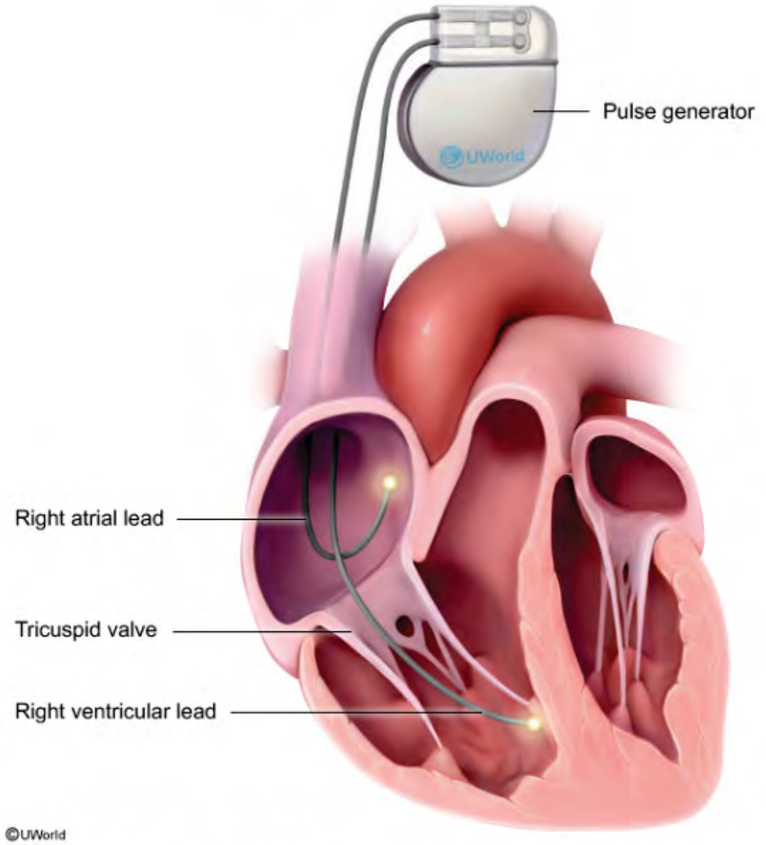
Dual chamber pacemaker



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This patient likely has **tricuspid valve regurgitation** (TR) due to an adverse effect of her permanent pacemaker. The right ventricular lead of an **implantable pacemaker or cardioverter-defibrillator** passes through the superior vena cava into the right atrium and then through the tricuspid valve orifice to terminate on the endocardium of the right ventricle. Damage to the tricuspid valve leaflets or inadequate leaflet coaptation can sometimes occur, leading to severe TR in some patients.

Severe TR typically presents with **right-sided heart failure**. Patients can have **distended jugular veins**, pulsatile and **tender hepatomegaly**, abdominal distension with **ascites**, and **lower extremity edema**. The lungs are clear on auscultation in the absence of concomitant left-sided heart disease. Cardiac examination typically reveals a **holosystolic murmur** best heard at the left lower sternal border; the murmur intensifies with maneuvers that increase right ventricular preload (eg, deep inspiration, leg raise).

(Choice A) Causes of aortic valve regurgitation include rheumatic heart disease and aortic root dilation. Severe disease can lead to left-sided heart failure; however, pulmonary edema with crackles on lung auscultation would be expected.

(Choice B) Constrictive pericarditis can result from chronic pericardial inflammation in the setting of infection or rheumatologic disease. It typically manifests with signs and symptoms of right-sided heart failure; however, the relationship between this patient's heart failure symptoms and recent pacemaker placement makes TR more likely.

(Choice C) Infective endocarditis may affect the tricuspid valve and lead to TR; however, tricuspid involvement typically develops in patients with a history of injection drug use. In addition, this patient's lack of fever makes infective endocarditis unlikely.

(Choice D) Severe pulmonic valve regurgitation can lead to right-sided heart failure. Causes of pulmonic valve regurgitation include rheumatic heart disease and endocarditis, but it is not associated with pacemaker placement as pacemaker leads do not cross the pulmonic valve.

Educational objective:
Severe tricuspid valve regurgitation (TR) can lead to right-sided heart failure, evidenced by jugular venous distension, hepatomegaly, lower extremity edema, and the absence of pulmonary edema. Permanent pacemaker placement can cause TR because the right ventricular lead passes through the tricuspid valve orifice and can

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(Choice D) Severe pulmonic valve regurgitation can lead to right-sided heart failure. Causes of pulmonic valve regurgitation include rheumatic heart disease and endocarditis, but it is not associated with pacemaker placement as pacemaker leads do not cross the pulmonic valve.

Educational objective:
Severe tricuspid valve regurgitation (TR) can lead to right-sided heart failure, evidenced by jugular venous distension, hepatomegaly, lower extremity edema, and the absence of pulmonary edema. Permanent pacemaker placement can cause TR because the right ventricular lead passes through the tricuspid valve orifice and can disrupt valve closure.

Pathophysiology | Cardiovascular System | Tricuspid regurgitation

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A 60-year-old man with known coronary artery disease comes to the office due to exertional chest tightness for the last 6 months. The symptoms occur when he walks more than 3 blocks, especially in cold weather. The patient underwent coronary artery bypass graft surgery 3 years ago for progressive angina. Past medical history also includes asthma, benign prostate hyperplasia, and peripheral artery disease. The physician discusses adding isosorbide dinitrate to his current therapy but the patient is concerned about adverse effects. Which of the following is most likely to occur in this patient with the add-on therapy?

- A. Atrioventricular conduction delay
- B. Cold extremities
- C. Constipation
- D. Joint pains
- E. Nocturnal wheezing
- F. Throbbing headaches
- G. Urinary retention

Submit

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- A. Atrioventricular conduction delay (4%)
- B. Cold extremities (11%)
- ✗ C. Constipation (3%)
- D. Joint pains (1%)
- E. Nocturnal wheezing (4%)
- ✓ F. Throbbing headaches (67%)
- G. Urinary retention (6%)

Incorrect
Correct answer
F

67%
Answered correctly

03 secs
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Explanation

Nitrates are frequently used for symptomatic relief in patients with chronic stable angina. The anti-ischemic effect of nitrates is mediated by systemic vasodilation (predominantly venodilation) with a decrease in left ventricular end-diastolic volume and wall stress, resulting in decreased myocardial oxygen demand and relief of angina symptoms.

The main adverse effects seen with nitrate therapy include **headaches, cutaneous flushing**, lightheadedness, **hypotension**, and reflex tachycardia. Nitrates must be avoided in patients with hypertrophic cardiomyopathy (due to increased outflow tract obstruction), right ventricular infarction (due to reduction in preload, impairing cardiac

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Explanation

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The main adverse effects seen with nitrate therapy include **headaches, cutaneous flushing**, lightheadedness, **hypotension**, and reflex tachycardia. Nitrates must be avoided in patients with hypertrophic cardiomyopathy (due to increased outflow tract obstruction), right ventricular infarction (due to reduction in preload, impairing cardiac output), and those on phosphodiesterase inhibitors (synergism increases the risk of severe hypotension).

(Choice A) Atrioventricular conduction delay is a potential adverse effect of several drugs used for management of chronic stable angina. These include calcium channel blockers (eg, verapamil, diltiazem) and beta blockers (eg, metoprolol, propranolol).

(Choice B) Cold extremities due to Raynaud phenomenon are associated with medications such as amphetamines, ergotamine, and chemotherapeutic agents.

(Choice C) Constipation is a common side effect of opioid analgesics, iron supplements, calcium channel blockers such as verapamil, and anticholinergic medications such as atropine.

(Choice D) Thiazide diuretics can lead to hyperuricemia and precipitate an acute gout attack.

(Choice E) Nocturnal wheezing can be a symptom of asthma or gastroesophageal reflux disease. In addition, the use of beta blockers in patients with underlying bronchospastic disease can cause increased bronchoconstriction and lead to wheezing.

(Choice G) Urinary retention is typically associated with medications that exhibit anticholinergic properties such as tricyclic antidepressants (eg, amitriptyline) and antihistamines (eg, diphenhydramine).

Educational objective:
The main adverse effects seen with nitrate therapy include headaches and cutaneous flushing along with lightheadedness and hypotension due to systemic vasodilation.

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A 47-year-old, previously healthy man is transported to the emergency department after he was involved in a high-speed, head-on motor vehicle collision. The patient was wearing a seat belt; he did not hit his head or lose consciousness. However, he has had chest pain and shortness of breath since the incident. On arrival, blood pressure is 160/90 mm Hg and pulse is 115/min. Bruising and tenderness are noted over the anterior chest wall. Neck veins are flat, and the trachea is central. Breath sounds are symmetric, and heart sounds are normal. The abdomen is soft and nontender. There are no significant extremity injuries, but bilateral femoral pulses are weak compared to the radial pulses. Chest x-ray reveals a widened mediastinum. While being prepared for surgery, the patient becomes unresponsive. The pulse is no longer detectable, and the cardiac monitor demonstrates sinus tachycardia. Despite aggressive resuscitation efforts, the patient dies. Autopsy examination is most likely to reveal an injury involving which of the following areas of the thoracic aorta?

- A. A
- B. B
- C. C
- D. D
- E. E

Submit

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A 47-year-old, previously healthy man is transported to the emergency department after he was involved in a high-
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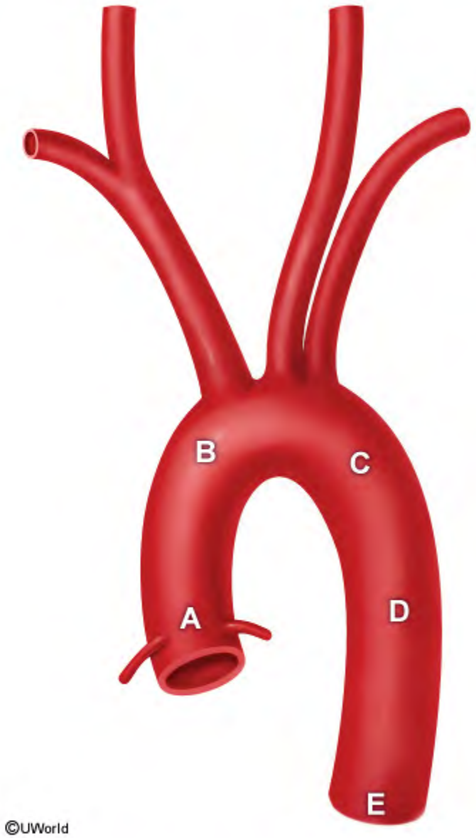

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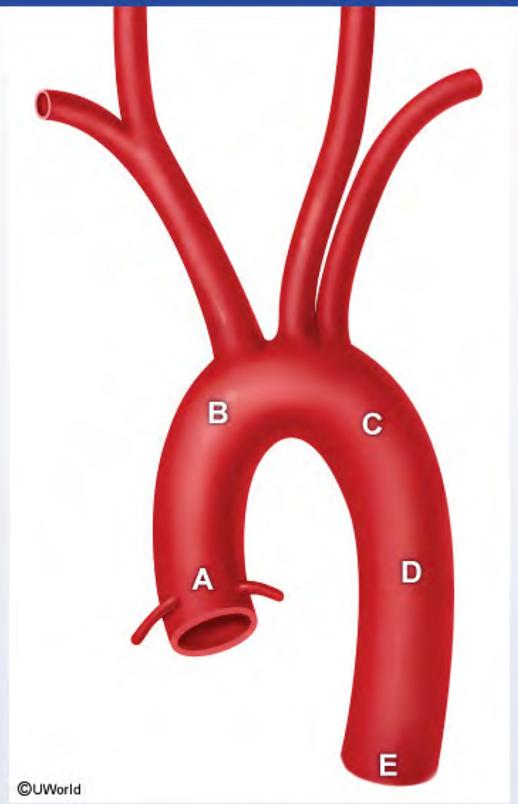

- A. A (6%)
- B. B (14%)
- ✓ C. C (52%)
- ✗ D. D (15%)
- E. E (8%)

Incorrect
Correct answer
C

52%
Answered correctly

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Incorrect
Correct answer
C

52%
Answered correctly

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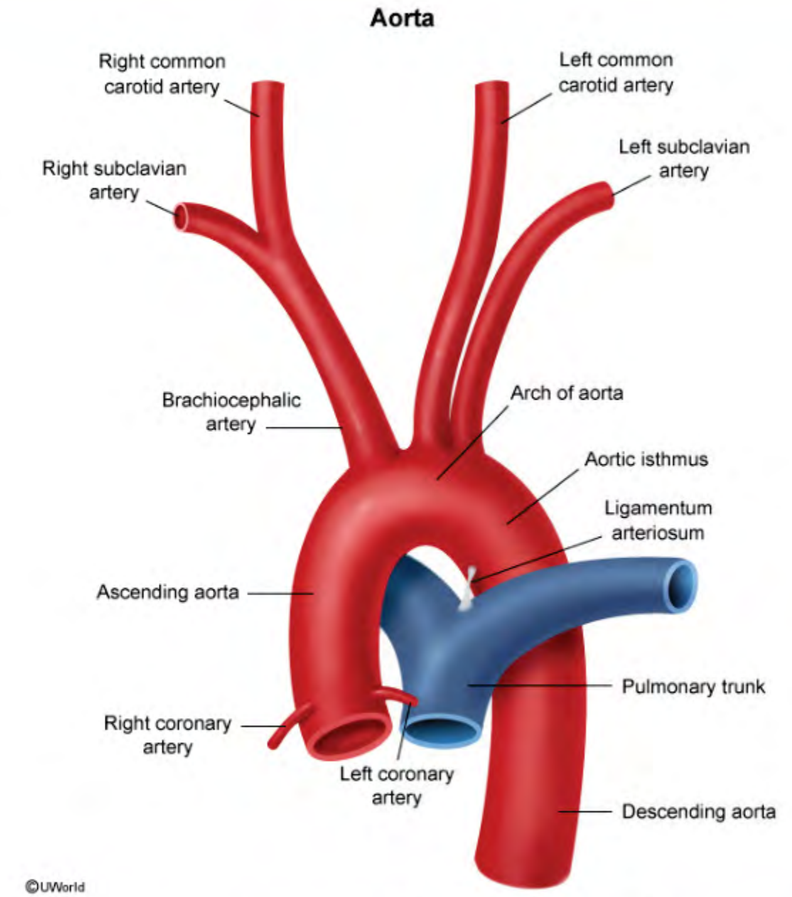

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Rapid deceleration during a high-speed motor vehicle collision can lead to **blunt aortic injury** (BAI) due to a combination of forces (eg, **shearing**) on the aorta. These forces can affect some or all of the layers (ie, intima, media, adventitia). Most patients exsanguinate rapidly (due to full-thickness injury that ruptures the adventitia); however, in a minority of cases (~20%), delayed rupture can allow survival until surgical treatment. A high index of suspicion is crucial because symptoms may be nonspecific (eg, chest pain, shortness of breath).

Depending on the extent and location of vessel injury, some findings suggestive of BAI include:

- **Widened mediastinum**
- **Pulse discrepancy** between the upper and lower extremities (similar to aortic coarctation)
- Intrascapular murmur (due to aortic flow impingement)

BAI occurs most often at **transition zones** between mobile and fixed portions of the aorta. The most common site of injury is the **aortic isthmus** (C), which is tethered by the ligamentum arteriosum and is relatively immobile compared to the adjacent descending aorta; the isthmus is particularly susceptible to BAI due to intrinsic weakness of the aortic wall in this area.

(Choices A and B) The aortic root (A) and the ascending aorta near the arch (B) are both transition zones and can be damaged by high-speed traumatic injury. However, these areas are affected less often than the aortic isthmus. In addition, this patient's circulation is preserved in the upper extremities compared to the lower extremities, which is more characteristic of an injury distal to the brachiocephalic and left subclavian arteries. Injury to the proximal aorta is classically associated with complications such as hemopericardium (eg, jugular vein distension), coronary artery dissection (eg, ST-segment elevation), and aortic valve disruption (eg, diastolic regurgitation murmur).

(Choices D and E) The distal thoracic aorta is also susceptible to injury just proximal to the diaphragm due to tethering at the diaphragmatic hiatus. However, this is a less common site for traumatic rupture than the more proximal portions of the aorta.

Educational objective:

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combination of forces (eg, **shearing**) on the aorta. These forces can affect some or all of the layers (ie, intima, media, adventitia). Most patients exsanguinate rapidly (due to full-thickness injury that ruptures the adventitia); however, in a minority of cases (~20%), delayed rupture can allow survival until surgical treatment. A high index of suspicion is crucial because symptoms may be nonspecific (eg, chest pain, shortness of breath).

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Educational objective:
Impacts causing the thorax to rapidly decelerate (eg, high-speed motor vehicle collisions) can result in blunt aortic injury, especially at transitions between relatively mobile and fixed zones. The most common site of injury is the aortic isthmus, just distal to the attachment of the ligamentum arteriosum.

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A 75-year-old man comes to the office due to worsening dyspnea and fatigue on exertion over the last 6 months. Recently, he has had severe lightheadedness during physical activity. Blood pressure is 125/65 mm Hg and pulse is 65/min and regular. Physical examination reveals a harsh ejection-type systolic murmur at the base of the heart radiating to the neck. The second heart sound is diminished in intensity. A fourth heart sound is heard at the cardiac apex. Which of the following is the most likely cause of this patient's heart condition?

- A. Extensive valve calcification with impaired leaflet mobility
- B. Fusion of valve commissures due to repetitive inflammation
- C. Hypertrophy of the basal interventricular septum with subaortic obstruction
- D. Infectious vegetations attached to the valve cusps
- E. Myxomatous valve degeneration with leaflet prolapse

Submit

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- ✓ A. Extensive valve calcification with impaired leaflet mobility (77%)
- B. Fusion of valve commissures due to repetitive inflammation (2%)
- ✗ C. Hypertrophy of the basal interventricular septum with subaortic obstruction (14%)
- D. Infectious vegetations attached to the valve cusps (1%)
- E. Myxomatous valve degeneration with leaflet prolapse (4%)

Incorrect
Correct answer
A

77%
Answered correctly

03 secs
Time Spent

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Explanation

This patient's presentation (fatigue, progressive dyspnea, presyncope with exertion, and a cardiac murmur) is suggestive of **aortic valve stenosis** (AS). In AS, the heart is unable to adequately increase cardiac output during exercise, causing pulmonary vascular congestion and reduced mean arterial pressure. These changes cause dyspnea and lightheadedness, respectively. AS can also chronically lower cardiac output, causing an increased tendency toward fatigue in general. AS murmur is usually a harsh ejection-type systolic murmur heard best at the base of the heart in the "aortic area" (second right intercostal space) with radiation to the carotid arteries. Second heart sound (S2) is diminished in intensity due to reduced mobility of the aortic leaflets, and a fourth heart sound (S4) may be heard due to decreased compliance of the hypertrophic myocardium.

Calcific degeneration of the trileaflet aortic valve is the most common cause of valvular AS in developed nations.

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Explanation

This patient's presentation (fatigue, progressive dyspnea, presyncope with exertion, and a cardiac murmur) is suggestive of **aortic valve stenosis** (AS). In AS, the heart is unable to adequately increase cardiac output during exercise, causing pulmonary vascular congestion and reduced mean arterial pressure. These changes cause dyspnea and lightheadedness, respectively. AS can also chronically lower cardiac output, causing an increased tendency toward fatigue in general. AS murmur is usually a harsh ejection-type systolic murmur heard best at the base of the heart in the "aortic area" (second right intercostal space) with radiation to the carotid arteries. Second heart sound (S2) is diminished in intensity due to reduced mobility of the aortic leaflets, and a fourth heart sound (S4) may be heard due to decreased compliance of the hypertrophic myocardium.

Calcific degeneration of the trileaflet aortic valve is the most common cause of valvular AS in developed nations. AS is characterized by progressive aortic valve leaflet thickening and calcification, leading to restricted leaflet excursion and mobility. Congenital heart disease, such as bicuspid aortic valve, can also lead to AS due to accelerated valve degeneration. On the other hand, rheumatic valve disease is a common cause of AS in developing nations and is characterized by fusion of valve commissures due to repetitive inflammation **(Choice B)**. Rheumatic valve disease usually presents at an earlier age and involves the mitral valve as well.

(Choice C) Hypertrophy of the basal interventricular septum with subaortic obstruction is the pathophysiologic process in patients with hypertrophic cardiomyopathy. Physical examination reveals a harsh crescendo-decrescendo murmur heard best at the apex and lower sternal border. The murmur does not radiate to the neck, and S2 is usually normal.

(Choice D) Infective endocarditis can lead to aortic regurgitation (AR) due to inadequate coaptation of the aortic leaflets. AR murmur is an early, decrescendo, high-pitched, blowing diastolic murmur that begins immediately after A2. It is best heard along the left sternal border at the third and fourth intercostal spaces.

(Choice E) Myxomatous degeneration of the mitral valve leaflets leads to mitral valve prolapse. Cardiac auscultation usually reveals a non-ejection click and mid-to-late systolic murmur of mitral regurgitation.

Educational objective:

Calcific degeneration of the trileaflet aortic valve is the most common cause of aortic stenosis (AS) in developed

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(Choice E) Myxomatous degeneration of the mitral valve leaflets leads to mitral valve prolapse. Cardiac auscultation usually reveals a non-ejection click and mid-to-late systolic murmur of mitral regurgitation.

Educational objective:
Calcific degeneration of the trileaflet aortic valve is the most common cause of aortic stenosis (AS) in developed nations. AS is characterized by progressive aortic valve leaflet thickening and calcification, leading to restricted leaflet excursion and mobility. AS murmur is usually a harsh ejection-type systolic murmur heard best at the base of the heart in the "aortic area" (second right intercostal space) with radiation to the carotid arteries.

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A 52-year-old man comes to the office with concerns about a "heart problem." Three years ago, the patient was told during a routine physical examination that he had a heart murmur. Currently, he has no specific complaints, but he mentions that he has become tired more easily in the past year. He has no chest pain or lower extremity swelling or significant medical history. The patient was healthy as a child and received all his childhood vaccinations. Blood pressure is 145/90 mm Hg and pulse is 80/min and regular. Physical examination reveals a holosystolic murmur best heard at the apex of the heart that radiates to the axilla. The lungs are clear on auscultation and abdominal examination is unremarkable. Which of the following is the best indicator of the severity of this patient's valve disease?

- A. Presence of audible S3
- B. Presence of audible S4
- C. Presence of mid-systolic click
- D. S2-to-opening snap time interval
- E. Splitting of S2 accentuated by inspiration

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- ✓ A. Presence of audible S3 (40%)
- ✗ B. Presence of audible S4 (11%)
- C. Presence of mid-systolic click (15%)
- D. S2-to-opening snap time interval (23%)
- E. Splitting of S2 accentuated by inspiration (8%)

Incorrect
Correct answer
A

40%
Answered correctly

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Time Spent

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Version

Explanation

A holosystolic murmur at the apex with radiation to the axilla is consistent with **mitral regurgitation** (MR). The murmur is generated by regurgitant blood flow from the left ventricle back to the left atrium during systole. This results in elevated pressure and blood volume in the left atrium, which increases the amount of blood reentering the left ventricle during diastole; an **audible S3 gallop** occurs when the left ventricle is unable to accommodate the excess blood flow.

An S3 is generated by the sudden cessation of blood flow into the left ventricle during the passive filling phase of

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Incorrect
Correct answer
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40%
Answered correctly

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Time Spent

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Version

Explanation

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An S3 is generated by the sudden cessation of blood flow into the left ventricle during the passive filling phase of diastole. A higher volume of blood flow or a more dilated left ventricle is more likely to produce an S3, and although an S3 can be a normal finding in healthy young adults, it is usually a pathologic finding in older adults that is classically associated with heart failure. In patients with MR, an S3 is the best indicator of severe MR with left-sided volume overload; the **absence of an S3** can be used to **exclude severe chronic MR**.

(Choice B) An S4 is a low-frequency diastolic sound that occurs during the atrial kick of ventricular diastole, and it reflects blood colliding with a stiff ventricular wall. It often indicates pathology such as hypertrophic cardiomyopathy or concentric left ventricular hypertrophy (eg, due to hypertension or aortic stenosis).

(Choice C) A mid-systolic click is characteristic of mitral valve prolapse. It is caused by sudden tensing of the chordae tendineae as they are pulled taut by the valve leaflets ballooning into the left atrium. The timing of the click varies with left ventricular volume; it occurs earlier in systole with physical maneuvers that decrease left ventricular volume.

(Choice D) An opening snap is an early diastolic sound heard after S2 in patients with mitral or tricuspid stenosis. The S2-to-opening snap interval is the time between the closure of the aortic valve and the abrupt halting of leaflet motion during opening of a stenotic mitral valve; a shorter S2-to-opening snap interval is indicative of more severe mitral stenosis.

(Choice E) Pulmonic valve stenosis delays pulmonic valve closure leading to widened splitting of S2. The splitting is further accentuated by the increase in venous return to the right side of the heart that occurs with inspiration.

Educational objective:

Patients with severe mitral regurgitation develop left-sided volume overload with an S3 gallop due to the large

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Educational objective:
Patients with severe mitral regurgitation develop left-sided volume overload with an S3 gallop due to the large volume of regurgitant flow reentering the ventricle during mid-diastole. The absence of an S3 gallop excludes severe chronic MR.

References

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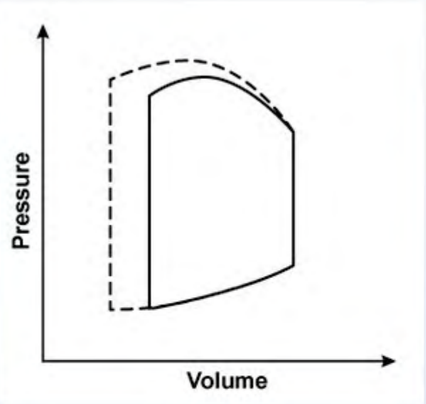
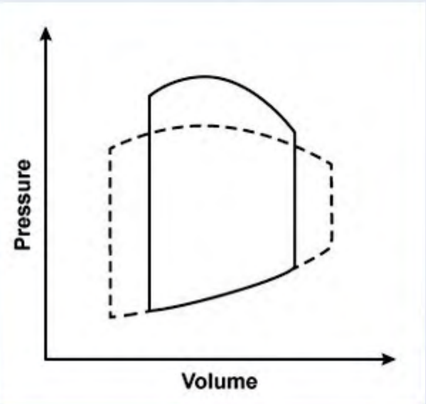
A 44-year-old man is hospitalized with multiple injuries following a motor vehicle collision. An arteriovenous fistula created by an injury would most likely result in which of the following changes to the left ventricular pressure-volume loop?

Solid line = preinjury
Dashed line = postinjury

A.



B.



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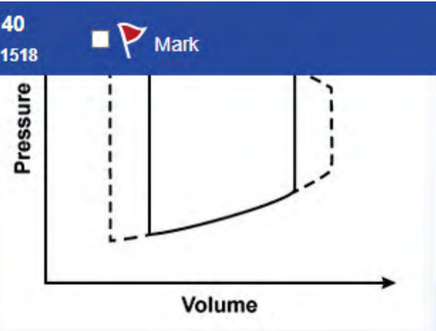

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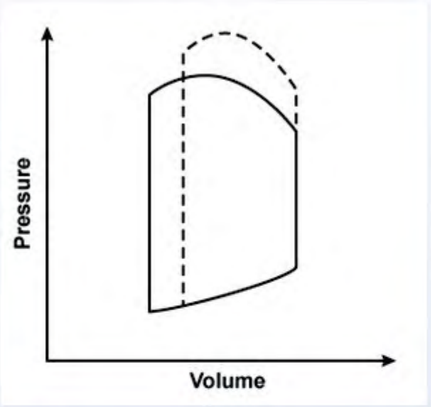

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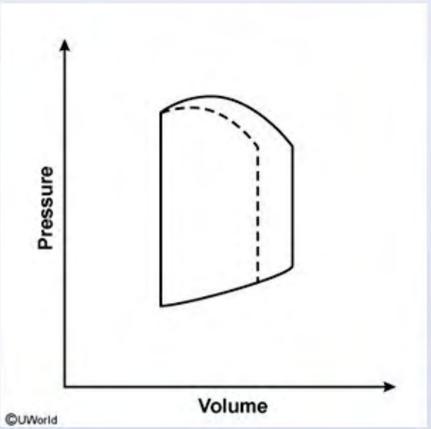

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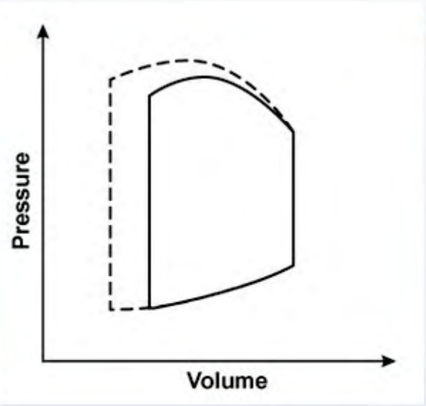
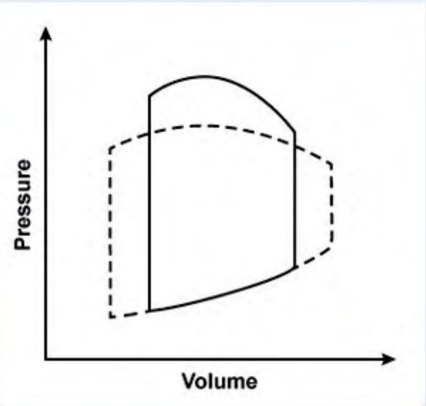


✓ B. (58%)



C. (11%)

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Explanation

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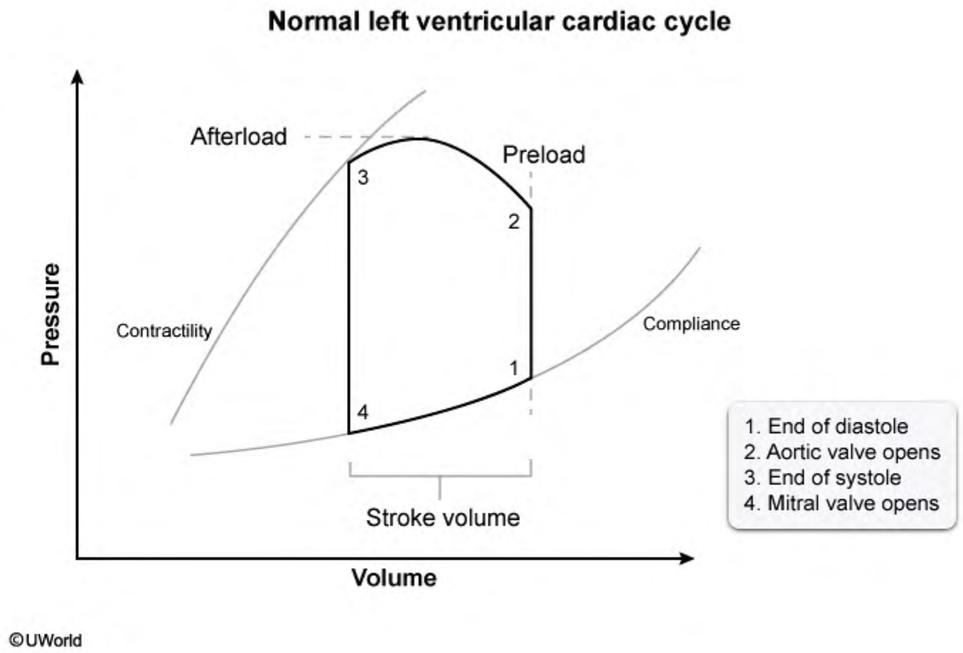

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circulation without passing through the high resistance of the systemic arterioles. This has the following effects on the LV pressure-volume loop:

- The low-resistance fistula pathway creates overall reduced systemic vascular resistance (ie, **reduced**

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The **left ventricular (LV) pressure-volume loop** follows the cardiac cycle. At point 1, the left ventricle is full of blood and begins isovolumetric contraction until the aortic valve opens at point 2, causing blood to eject into the aorta and LV volume to decrease. LV contraction ends at point 3, and LV pressure rapidly decreases to point 4, where the mitral valve opens and LV diastolic filling begins again.

An **arteriovenous fistula**, which can be congenital (eg, arteriovenous malformation) or acquired (eg, traumatic injury, surgical placement for hemodialysis), allows blood to shunt from the arterial circulation to the venous circulation without passing through the high resistance of the systemic arterioles. This has the following effects on the LV pressure-volume loop:

- The low-resistance fistula pathway creates overall reduced systemic vascular resistance (ie, **reduced afterload**).
- Blood quickly and easily passes through the reduced peripheral resistance, resulting in increased venous return to the right atrium (ie, **increased preload**).
- Both the reduced afterload and increased preload facilitate **increased stroke volume**, which increases cardiac output and helps maintain blood pressure.
- Contractility is unchanged initially but may eventually increase via the baroreceptor reflex if there is sustained hypotension.

With a large arteriovenous fistula, the left ventricle may be unable to keep up with the increased venous return despite a sustained increase in cardiac output, leading to high-output heart failure.

(Choice A) This pressure-volume loop shows increased contractility with a resulting increase in stroke volume and slight increase in afterload. Such changes are expected with administration of a positive inotropic agent such as digoxin.

(Choice C) This pressure-volume loop shows increased afterload with a resulting decrease in stroke volume. Contractility is unchanged. Such changes are expected with the obstruction to LV outflow that occurs with aortic stenosis.

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(Choice D) This pressure-volume loop shows decreased preload with a resulting decrease in stroke volume and slight decrease in afterload. Such changes are expected with administration of a primarily venous vasodilator such as nitroglycerin.

Educational objective:
An arteriovenous fistula allows blood to shunt from the arterial circulation to the venous circulation, bypassing the high resistance of the systemic arterioles. This leads to reduced systemic vascular resistance (ie, reduced afterload), increased venous return (ie, increased preload), and increased stroke volume. Left ventricular contractility is unchanged.

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2. Aortic valve opens
3. End of systole

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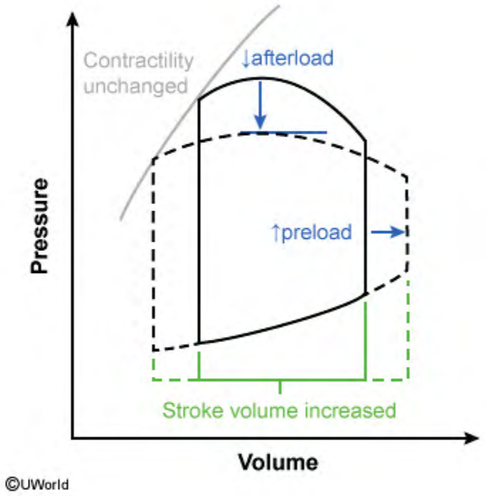

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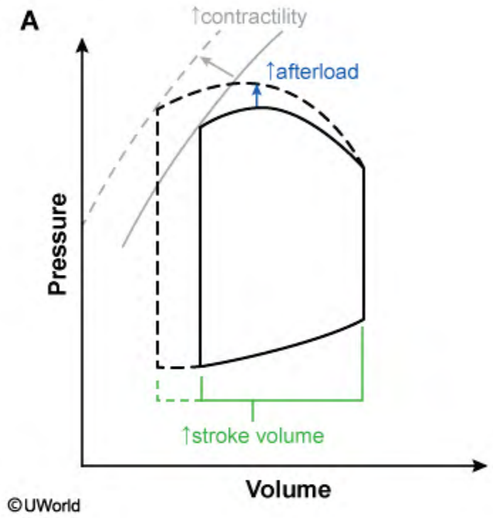

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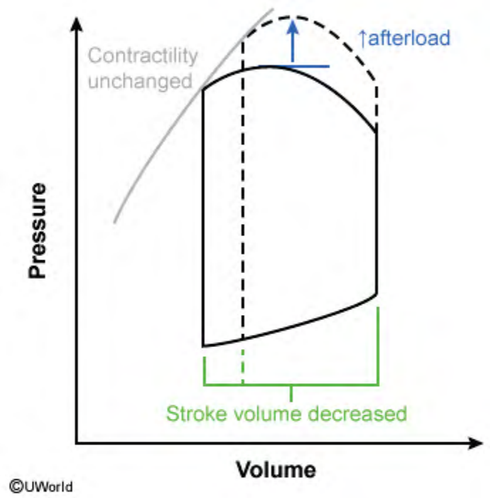

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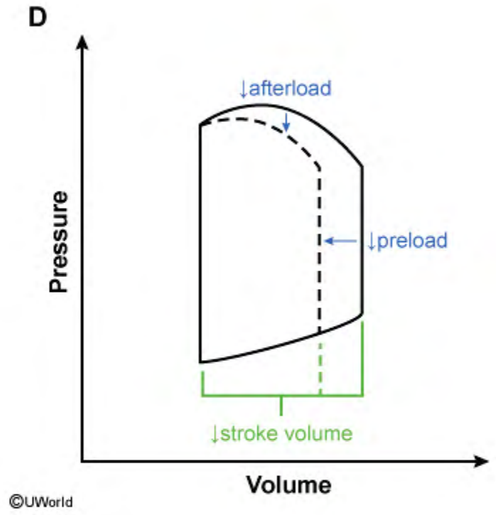

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Physiology
Subject

Cardiovascular System
System

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Topic

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A 33-year-old woman who recently immigrated to the United States is brought to the emergency department with severe shortness of breath and hemoptysis. Physical examination reveals a diastolic murmur. Chest x-ray shows severe pulmonary vascular congestion and edema. She is admitted to the hospital, treated with diuretics, and begins to feel better. However, during her hospitalization, she develops right-sided hemiparesis. Which of the following additional findings in this patient would be most suggestive of combined disease involving the mitral and aortic valves rather than exclusive mitral involvement?

- A. Increased left ventricular diastolic pressure
- B. Increased pulmonary artery pressure
- C. Increased pulmonary capillary wedge pressure
- D. Reduced pulmonary vascular compliance
- E. Right ventricular dilation
- F. Tricuspid regurgitation

Submit

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- ✓ A. Increased left ventricular diastolic pressure (74%)
- B. Increased pulmonary artery pressure (3%)
- ✗ C. Increased pulmonary capillary wedge pressure (13%)
- D. Reduced pulmonary vascular compliance (2%)
- E. Right ventricular dilation (4%)
- F. Tricuspid regurgitation (1%)

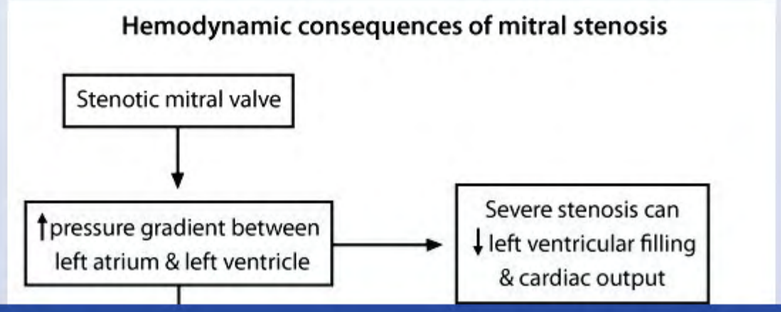
Incorrect
Correct answer
A

74%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation



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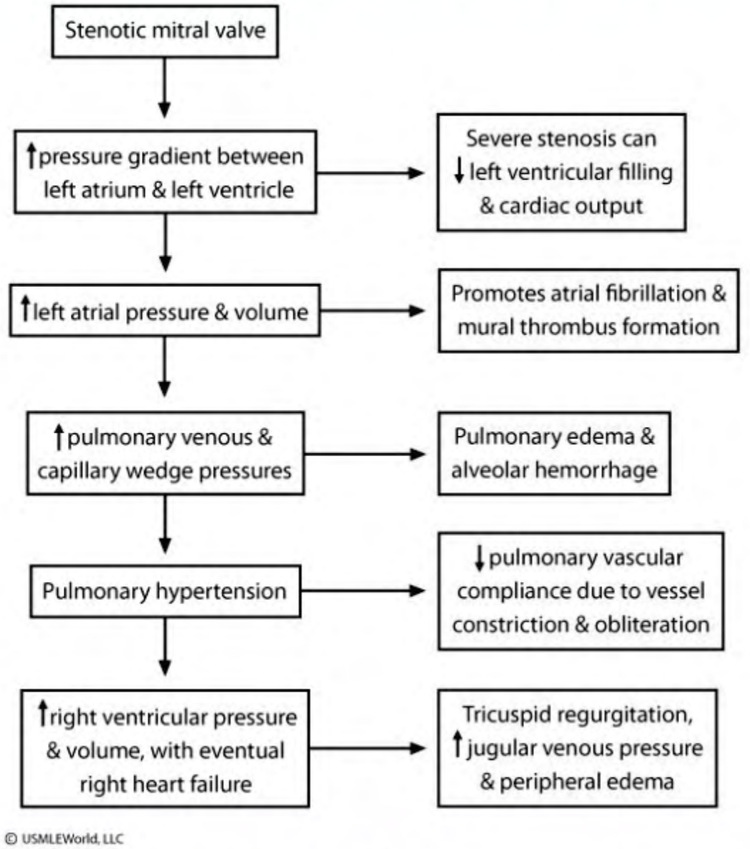
Hemodynamic consequences of mitral stenosis



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left atrium & left ventricle
& cardiac output

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This patient's physical examination findings are suggestive of **mitral stenosis** (MS). In isolated MS, cardiac and pulmonary pressures proximal to the stenotic mitral valve are markedly elevated. However, diastolic pressure in the left ventricle (LV) is usually near normal or even decreased with severe stenosis. **Increased LV diastolic pressure** in a patient with MS suggests concomitant **aortic valve dysfunction**.

Rheumatic heart disease almost always affects the mitral valve, but both the mitral and aortic valves are affected in about 25% of cases. Aortic valve involvement typically involves combined aortic stenosis and regurgitation, both of which can increase LV diastolic pressure. Rheumatic deformation of the aortic valve can also predispose to infective endocarditis even when the valve is functionally normal. This patient's right-sided **hemiparesis** is most likely due to **embolism**, which could have originated from an atrial mural thrombus secondary to atrial dilation from MS or an endocarditis-related valvular vegetation (less likely).

(Choices B and D) Severe, isolated MS can cause increased pulmonary artery pressure (pulmonary hypertension) via backward transmission of elevated pulmonary capillary wedge pressure. Longstanding pulmonary hypertension can result in reduced pulmonary vascular compliance due to endothelial remodelling and capillary obliteration.

(Choice C) Pulmonary capillary wedge pressure (an estimate of left atrial pressure) will increase with any significant left-sided valvular pathology (ie, both isolated MS and combined aortic and mitral valve disease).

(Choices E and F) Right ventricular dilatation can occur in MS when the resulting pulmonary hypertension is severe enough to cause right heart failure. Tricuspid regurgitation can occur as a complication of right ventricular dilatation.

Educational objective:
Isolated mitral stenosis causes elevated upstream pressures in the left atrium and pulmonary veins and arteries. Left ventricular end diastolic pressure (LVEDP) is normal or decreased due to obstruction of blood flow through the stenotic valve. An elevated LVEDP suggests additional downstream pathology (eg, aortic valve disease, left ventricular failure).

Pathophysiology	Cardiovascular System	Mitral stenosis
Subject	System	Topic

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A 35-year-old man comes to the office with progressive fatigue, dyspnea on exertion, and lower extremity edema over the last 2 weeks. Preceding these symptoms, he had an episode of fever, runny nose, and myalgias that resolved after several days. The patient has no other medical history, takes no medications, and has no significant family history. He occasionally drinks alcohol and does not use tobacco. Blood pressure is 112/74 mm Hg, and pulse is 98/min and regular. Physical examination reveals jugular venous distension, bibasilar crackles on lung auscultation, and 2+ pitting edema involving the lower extremities. Which of the following echocardiographic findings is most likely to be seen in this patient?

- A. Concentric left ventricle thickening with abnormal diastolic relaxation
- B. Dilated ventricles with abnormal systolic ventricular function
- C. High systolic pressure gradient across the aortic valve
- D. Regional wall motion abnormality
- E. Systolic anterior motion of the mitral valve

Submit

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- A. Concentric left ventricle thickening with abnormal diastolic relaxation (8%)
- ✓ B. Dilated ventricles with abnormal systolic ventricular function (58%)
- C. High systolic pressure gradient across the aortic valve (4%)
- D. Regional wall motion abnormality (7%)
- E. Systolic anterior motion of the mitral valve (20%)

Correct

58% Answered correctly

03 secs Time Spent

2023 Version

Explanation

Causes of nonvalvular heart failure				
	Ventricular wall thickness	**Ventricular cavity size**	**Contractile function**	**Diastolic function**
Ischemic heart disease	↓ or normal	↑	↓	↓ or normal
Dilated cardiomyopathy*				
Hypertensive heart				

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Causes of nonvalvular heart failure				
	Ventricular wall thickness	**Ventricular cavity size**	**Contractile function**	**Diastolic function**
Ischemic heart disease	↓ or normal	↑	↓	↓ or normal
Dilated cardiomyopathy*				
Hypertensive heart disease	↑	↓	normal	↓
Hypertrophic cardiomyopathy*	↑ septal thickening	↓	normal	↓
Restrictive cardiomyopathy*	↑ or normal	↓ or normal	normal	↓

*Cardiomyopathies are characterized by primary myocardial dysfunction (eg, viral myocarditis, genetic disorder, infiltrative disease). Myocardial disease caused by secondary factors (eg, hypertension, coronary artery disease, valvular dysfunction) is not classified as a cardiomyopathy.

This patient's presentation with dyspnea, lower extremity edema, jugular venous distension, and pulmonary crackles is consistent with **decompensated heart failure**. In a young patient who develops heart failure following a symptomatic viral prodrome (eg, fever, sinus congestion, myalgias), **dilated cardiomyopathy** (DCM) due to **viral myocarditis** should be suspected. A number of viruses have been implicated in causing DCM including coxsackievirus, adenovirus, and influenza virus. It is believed that an inadequate immune response to the viral infection allows virus to infect and persist inside cardiomyocytes, resulting in damage due to a direct cytotoxic effect or a destructive autoimmune reaction.

Depletion of cardiomyocytes with subsequent fibrosis causes weakened myocardial contractility (**systolic dysfunction**), volume overload, and ventricular dilation (**eccentric hypertrophy**) as surviving myocytes respond to increased wall stress. Other causes of DCM include genetic disease, pregnancy, infiltrative disease (eg, late

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(Choice A) Concentric left ventricular (LV) hypertrophy with thickened walls and abnormal diastolic relaxation is characteristic of hypertensive heart disease due to long-standing uncontrolled hypertension. Initially, the LV cavity is reduced in size and there is normal systolic function; however, over time, high afterload pressures can cause progressive systolic dysfunction, LV cavity enlargement, and heart failure.

(Choice C) High systolic pressure gradient across the aortic valve is present in aortic stenosis; concentric LV hypertrophy can occur due to LV pressure overload. Aortic stenosis can develop in relatively young patients due to calcification of a bicuspid aortic valve; however, a crescendo-decrescendo systolic murmur is expected on examination.

(Choice D) Regional wall motion abnormality is suggestive of ischemic heart disease (eg, myocardial infarction). Contractile function is impaired in the damaged portion of myocardium, resulting in systolic dysfunction with LV volume overload, eccentric hypertrophy, and LV cavity enlargement. Ischemic heart disease is unlikely in this young patient without significant risk factors for cardiovascular disease.

(Choice E) In patients with hypertrophic cardiomyopathy, systolic anterior motion of the mitral valve may worsen LV outflow tract obstruction. Clinical features of hypertrophic cardiomyopathy include exercise-induced syncope (due to outflow obstruction) and sudden cardiac death (due to ventricular arrhythmia) in young athletes.

Educational objective:
Dilated cardiomyopathy results from direct damage to cardiomyocytes leading to myocardial contractile dysfunction (systolic dysfunction), volume overload, and ventricular dilation. Viral myocarditis is a common cause of dilated cardiomyopathy and should be suspected in young patients who develop heart failure following a symptomatic viral prodrome.

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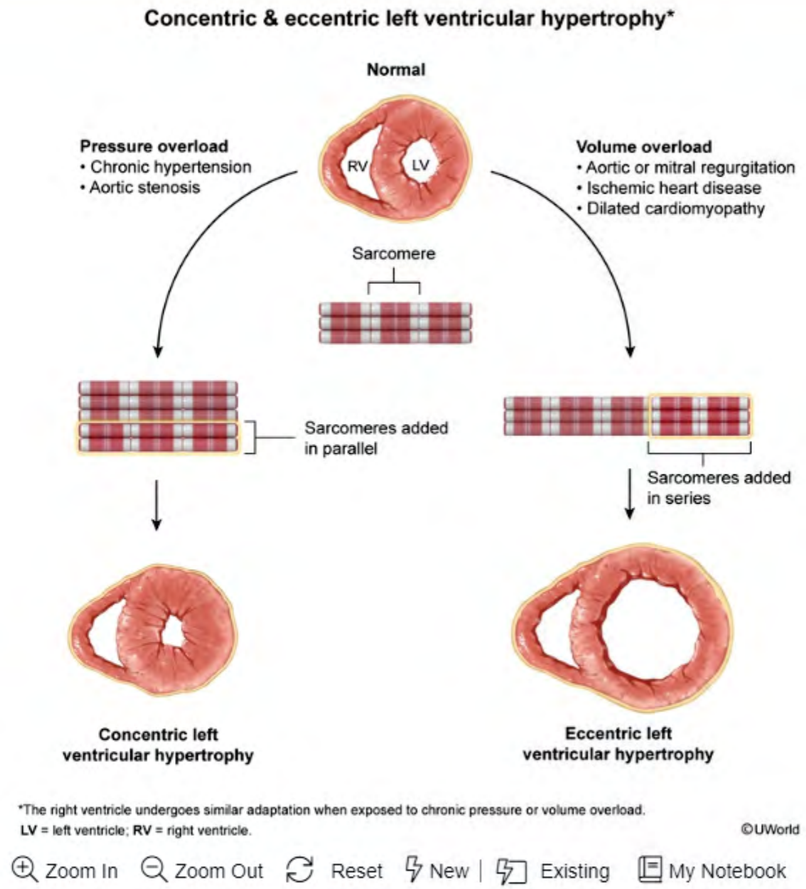

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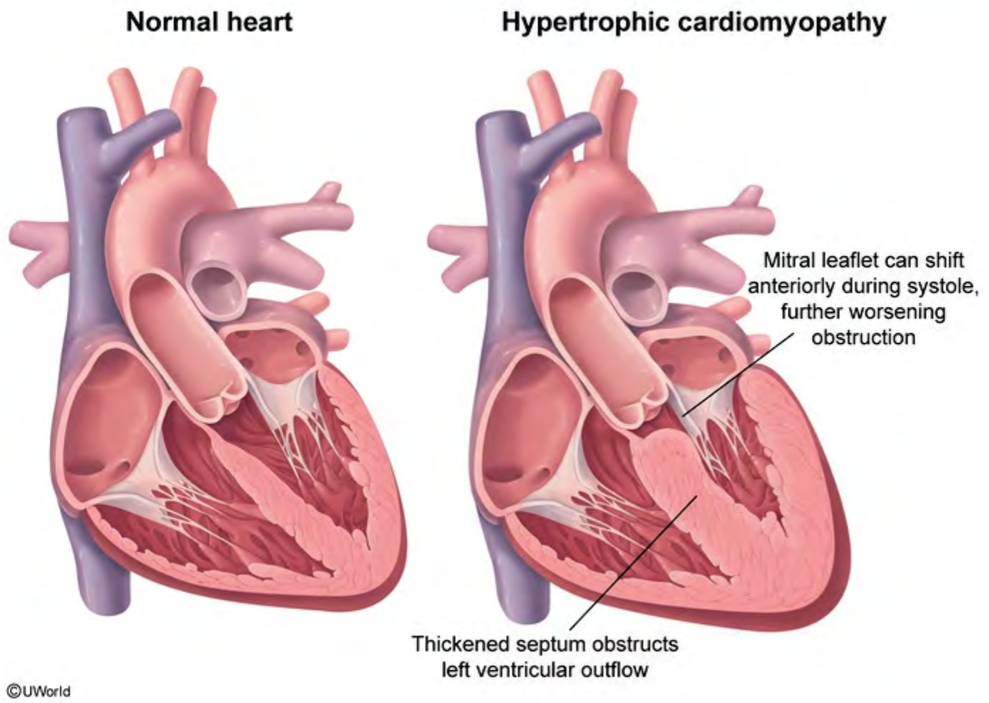

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A 50-year-old man is brought to the ER with severe dizziness and confusion. He states that he had an episode of chest pain and took several tablets of nitroglycerin. His current medications include a daily aspirin for heart attack prevention, an occasional acetaminophen for headaches and occasionally tadalafil for erectile dysfunction. His blood pressure is 50/20 mmHg and his heart rate is 120 beats/min. Which of the following cellular changes is most likely responsible for this patient's symptoms?

- A. Receptor downregulation
- B. G_s protein phosphorylation
- C. Cyclic GMP accumulation
- D. Tyrosine kinase overactivity
- E. Enhanced phospholipid metabolism
- F. Tolerance development

Submit

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- ✗ A. Receptor downregulation (2%)
- B. G_s protein phosphorylation (4%)
- ✓ C. Cyclic GMP accumulation (87%)
- D. Tyrosine kinase overactivity (2%)
- E. Enhanced phospholipid metabolism (1%)
- F. Tolerance development (2%)

Incorrect
Correct answer
C

87%
Answered correctly

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Explanation

The interaction between nitrates and phosphodiesterase inhibitors used in erectile dysfunction (tadalafil, sildenafil, vardenafil) is well known and very important to understand. Nitrates are converted to nitric oxide by vascular smooth muscle cells, and nitric oxide causes increased intracellular cGMP as a second-messenger. Increased cGMP concentration leads to vascular smooth muscle relaxation. Additionally, cGMP is metabolized within the cells by phosphodiesterase, and phosphodiesterase inhibitors will lead to increased intracellular cGMP. cGMP accumulation in vascular smooth muscle cells due to both enhanced synthesis (nitrates) and inhibited degradation (PDE inhibitors) is responsible for profound hypotension due to extreme vasodilatation when these drugs are used together.

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Explanation

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(Choice A) Receptor downregulation or tachyphylaxis occurs with many drug classes including topical glucocorticoids, opiates, L-dopa and more. It is one mechanism by which the body develops tolerance to drugs on a cellular level, but it is not the cause of hypotension in this patient who took nitrates together with a PDE inhibitor.

(Choice B) G_s protein phosphorylation leads to activation of the G_s protein, a stimulatory protein which acts as a part of the adenylate cyclase second messenger system. It has no role in cGMP pathways that are affected by nitrates and PDE inhibitors.

(Choice D) Tyrosine kinase overactivity would lead to an exaggeration of the effects of insulin or insulin-like growth factor. Tyrosine kinase has no role in the effect of nitrates or PDE inhibitors on vascular smooth muscle.

(Choice E) Enhanced phospholipid metabolism would lead to an increase in the production of prostaglandins (via diacylglycerol) and inositol triphosphate (leading to calcium release from the endoplasmic reticulum). This is the inositol triphosphate second-messenger system, and it mediates the effects of alpha-1 adrenergic receptors, angiotensin II receptors, oxytocin receptors and vascular vasopressin receptors. It is not involved in the action of nitrates or PDE inhibitors.

(Choice F) Tolerance refers to the need for a larger dose of drug to achieve the same clinical effect achieved previously with a smaller dose of drug. Development of tolerance can occur by multiple mechanisms such as a

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(PDE inhibitors) is responsible for profound hypotension due to extreme vasodilatation when these drugs are used together.

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(Choice F) Tolerance refers to the need for a larger dose of drug to achieve the same clinical effect achieved previously with a smaller dose of drug. Development of tolerance can occur by multiple mechanisms such as a change in cellular response to drug (decreased receptors, decreased response to receptor stimulation), as well as increased rate of drug elimination by the body. Tolerance is not the reason for this patient to experience hypotension with this drug combination.

Educational Objective:
Using nitrates together with phosphodiesterase (PDE) inhibitors used for erectile dysfunction and pulmonary hypertension causes a profound systemic hypotension because they both increase intracellular cGMP which causes vascular smooth muscle relaxation. Their use together is absolutely contraindicated.

Pharmacology	Cardiovascular System	Nitrates
Subject	System	Topic

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A 67-year-old man with nonischemic cardiomyopathy comes to the office for follow-up. He recently was hospitalized for acute decompensated heart failure. The patient's symptoms have improved with multidrug treatment, but he has persistent shortness of breath on mild exertion. He has a history of hypertension and hypercholesterolemia. Blood pressure is 115/70 mm Hg and pulse is 66/min. There is a third heart sound on heart auscultation and mild lower extremity pitting edema. A recent echocardiogram showed a left ventricular ejection fraction of 30%. Which of the following diuretics would most likely improve survival if added to this patient's current regimen?

- A. Acetazolamide
- B. Furosemide
- C. Hydrochlorothiazide
- D. Mannitol
- E. Spironolactone
- F. Triamterene

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- A. Acetazolamide (1%)
- ✗ B. Furosemide (27%)
- C. Hydrochlorothiazide (16%)
- D. Mannitol (1%)
- ✓ E. Spironolactone (50%)
- F. Triamterene (2%)

Incorrect
Correct answer
E

50%
Answered correctly

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Explanation

Mineralocorticoid receptor antagonists (eg, spironolactone, eplerenone) prevent aldosterone from binding to its receptor in the distal renal tubules. This leads to increased sodium and water excretion while conserving potassium ions (**potassium-sparing diuresis**). These antagonists also block the deleterious effect of aldosterone on the heart, causing regression of myocardial fibrosis and **improvement in ventricular remodeling**.

Mineralocorticoid receptor antagonists reduce morbidity and **improve survival** in patients with congestive heart failure and decreased ejection fraction. Therefore, they are recommended in addition to standard heart failure

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Mineralocorticoid receptor antagonists reduce morbidity and **improve survival** in patients with congestive heart failure and decreased ejection fraction. Therefore, they are recommended in addition to standard heart failure therapy (ACE inhibitors and beta blockers). They should not be used in patients with hyperkalemia or renal failure. The most common side effects are hyperkalemia and gynecomastia (spironolactone >> eplerenone).

(Choices A, C, and F) Acetazolamide (carbonic anhydrase inhibitor), hydrochlorothiazide (thiazide diuretic), and triamterene (epithelial sodium channel blocker) have a variably lower diuretic effect compared to loop diuretics and are not as efficacious for treating heart failure symptoms.

(Choice B) Furosemide is a loop diuretic frequently used for treatment of pulmonary congestion and fluid retention in heart failure patients. Although loop diuretics improve symptoms significantly, they do not provide survival benefit (ie, improved morbidity but not mortality) in these patients.

(Choice D) Mannitol is an intravenous osmotic diuretic used primarily to lower intracranial pressure and treat acute glaucoma. It is not used to treat heart failure as it can cause an initial rise in extracellular fluid volume, exacerbating pulmonary congestion and peripheral fluid retention.

Educational objective:
Mineralocorticoid receptor antagonists (eg, spironolactone, eplerenone) improve survival in patients with congestive heart failure and reduced left ventricular ejection fraction. They should not be used in patients with hyperkalemia or renal failure.

References

- The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators.

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A 52-year-old woman with a history of locally advanced breast cancer comes to the office for follow-up. The patient is undergoing trastuzumab-based chemotherapy but has not received radiation therapy. She has no symptoms except mild fatigue. Blood pressure is 120/72 mm Hg and pulse is 85/min and regular. The patient is afebrile. Transthoracic echocardiography reveals a left ventricular ejection fraction of 40% with no evidence of valvular disease. Cardiac assessment prior to chemotherapy initiation was normal. Which of the following pathologic findings is most likely to be present in this patient's heart?

- A. Abnormal protein accumulation in the extracellular space
- B. Focal ischemic cardiomyocyte necrosis
- C. Myocardial tumor cell infiltration
- D. Patchy cardiomyocyte necrosis with diffuse myocardial fibrosis
- E. Reduced cardiomyocyte contractility with no fibrosis

Submit

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- A. Abnormal protein accumulation in the extracellular space (11%)
- ✗ B. Focal ischemic cardiomyocyte necrosis (3%)
- C. Myocardial tumor cell infiltration (3%)
- D. Patchy cardiomyocyte necrosis with diffuse myocardial fibrosis (27%)
- ✓ E. Reduced cardiomyocyte contractility with no fibrosis (53%)

Incorrect
Correct answer
E

53%
Answered correctly

03 secs
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Explanation

Trastuzumab is a monoclonal antibody used in the treatment of **breast cancer** caused by tumor cells that overexpress human epidermal growth factor receptor-2 (**HER2**). By binding to HER2, trastuzumab blocks downstream signaling that promotes cellular proliferation and thereby encourages malignant cell apoptosis.

The major adverse effect of trastuzumab is a risk of **cardiotoxicity**, likely because HER2 signaling plays a role in minimizing oxidative stress on cardiomyocytes and preserving cardiomyocyte function. Cardiotoxicity typically manifests as a **decrease in myocardial contractility** (myocardial stunning) **without cardiomyocyte destruction** or myocardial fibrosis. Patients usually experience an asymptomatic decline in left ventricular ejection fraction; however, overt heart failure can also occur. Unlike the cardiotoxicity that occurs with anthracyclines (eg,

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(Choice A) Abnormal protein accumulation in the cardiac extracellular space is seen in patients with amyloidosis, namely with the light-chain (AL) and transthyretin (ATTR) subtypes. The disease typically manifests initially as restrictive cardiomyopathy with impaired diastolic function and preserved ejection fraction.

(Choice B) Focal ischemic cardiomyocyte necrosis leading to left ventricular systolic dysfunction is seen in patients with a prior myocardial infarction due to coronary heart disease.

(Choice C) Breast cancer is one of the most common malignancies to metastasize to the heart, typically causing pericardial effusion that may gradually progress to cardiac tamponade. However, tamponade predominantly affects diastolic filling of the right-sided heart chambers and does not decrease left ventricular ejection fraction.

(Choice D) Anthracycline-induced cardiotoxicity occurs due to oxidative damage of cellular membranes, leading to cardiomyocyte necrosis and death with eventual myocardial fibrosis.

Educational objective:
Trastuzumab is a monoclonal antibody that blocks human epidermal growth factor receptor-2 (HER2) to disrupt malignant cell signaling and encourage apoptosis. Because HER2 helps preserve cardiomyocyte function, trastuzumab can cause cardiotoxicity that manifests as a decrease in myocardial contractility without cardiomyocyte destruction or myocardial fibrosis.

References

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Incorrect
Correct answer
E

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Answered correctly

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References

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A 2-month-old boy is found unresponsive in his crib. He had previously been well and had a normal physical examination at his 2-month visit approximately 1 week earlier. An autopsy is performed to determine the cause of death. The pathologist concludes that the infant likely died of sudden infant death syndrome. The autopsy also shows an incidental finding of bicuspid aortic valve. If this patient had survived, he would have been at greatest risk for which of the following events?

- A. Aortic dissection in his 30s
- B. Aortic stenosis in his 50s
- C. Atrial fibrillation in his 40s
- D. Endocarditis in his 20s
- E. Severe aortic stenosis in infancy
- F. Ventricular arrhythmia in childhood

Submit

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- ✗ A. Aortic dissection in his 30s (9%)
- ✓ B. Aortic stenosis in his 50s (73%)
- C. Atrial fibrillation in his 40s (1%)
- D. Endocarditis in his 20s (6%)
- E. Severe aortic stenosis in infancy (5%)
- F. Ventricular arrhythmia in childhood (2%)

Incorrect
Correct answer
B

73%
Answered correctly

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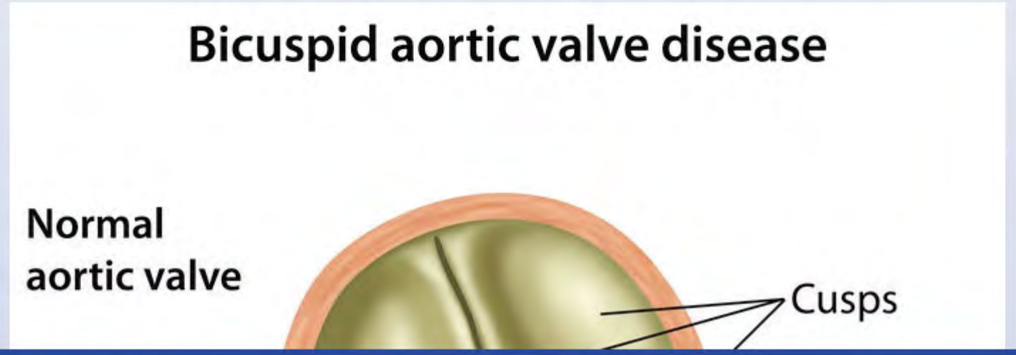
Explanation



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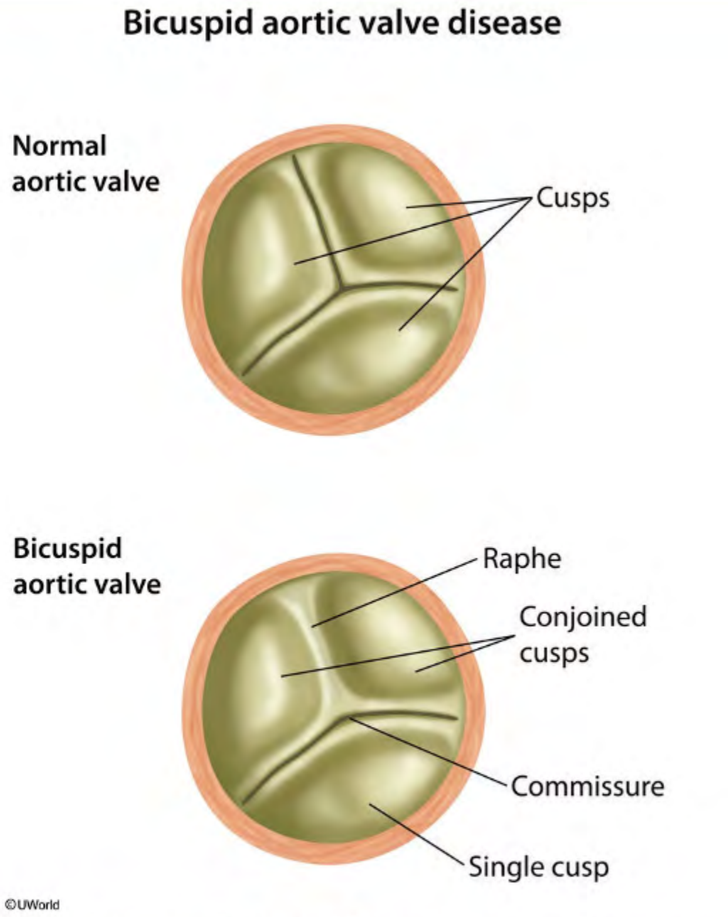

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Cusps

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Bicuspid aortic valves (right and left aortic cusp fusion) occur in approximately 1%-2% of live births, making it one of the **most common** congenital heart defects. The abnormally shaped valve experiences increased hemodynamic stress, which accelerates the normal aging process and causes premature **atherosclerosis** and **calcification** of the aortic valve. These deposits begin accumulating as early as adolescence and lead to **aortic stenosis** in **>50%** of affected patients. Symptomatic aortic stenosis develops on average around age 50 (10 years earlier than the average onset of senile calcific aortic stenosis in patients with normal aortic valves).

(Choice A) Patients with bicuspid aortic valves have a higher risk of aortic dissection. However, the absolute risk of aortic dissection is still <1%. As with other complications of bicuspid aortic valves, the risk increases with advancing age.

(Choice C) Atrial fibrillation has not been associated with bicuspid aortic valves.

(Choice D) Patients with bicuspid aortic valves are at increased risk for infective endocarditis. The risk is 2%-3% and rises with age due to increasingly turbulent blood flow across the abnormal valve.

(Choice E) In early childhood, isolated bicuspid aortic valves usually are asymptomatic, without any hemodynamically significant degree of aortic outflow tract obstruction or murmurs. In contrast, congenital aortic stenosis usually produces a systolic murmur as a result of severe subvalvular, valvular, or supravalvular anatomic defects.

(Choice F) The most common cause of ventricular fibrillation at age <30 is hypertrophic cardiomyopathy. This condition is not associated with bicuspid aortic valves.

Educational objective:
Aortic stenosis is the most common complication of bicuspid aortic valves. Patients with bicuspid aortic valves develop clinically significant aortic stenosis on average around age 50. In comparison, senile calcific stenosis of normal aortic valves generally becomes symptomatic age >65.

Pathophysiology	Cardiovascular System	Bicuspid aortic valve
Subject	System	Topic

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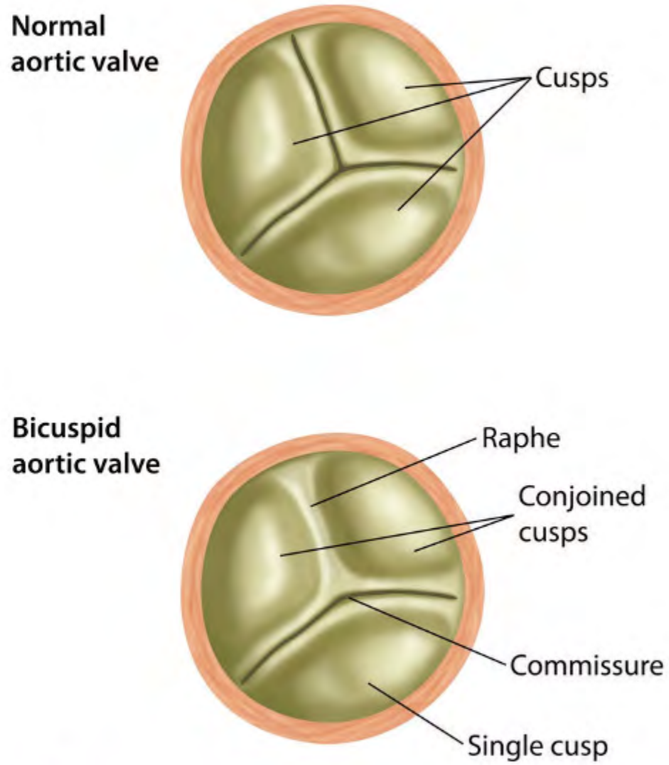
Bicuspid aortic valve disease



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A 17-year-old boy collapses while jogging and dies despite resuscitation efforts. He has no medical history; however, his family history is significant for an uncle who died suddenly at age 20. The boy is 185.4 cm (6 ft 1 in) tall and weighs 80 kg (176.4 lb). Autopsy reveals significant left ventricular hypertrophy predominantly affecting the interventricular septum. There are no valvular abnormalities. Assuming the boy died of an inherited condition, which of the following proteins was most likely affected by the relevant mutation?

- A. Beta-myosin heavy chain
- B. Dystrophin
- C. Fibrillin-1
- D. Membrane potassium channel
- E. Transthyretin

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- ✓ A. Beta-myosin heavy chain (80%)
- ✗ B. Dystrophin (3%)
- C. Fibrillin-1 (10%)
- D. Membrane potassium channel (1%)
- E. Transthyretin (3%)

Incorrect
Correct answer
A

80%
Answered correctly

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Explanation

Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain
Phenotypic	• Myocardial disarray & coronary microvascular dysfunction • Asymmetric septal hypertrophy with dynamic LVOT

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Explanation

Hypertrophic cardiomyopathy	
Inheritance	• Autosomal dominant with incomplete penetrance • Mutations affecting proteins of the cardiac sarcomere • Most common: myosin-binding protein C & β-myosin heavy chain
Phenotypic manifestations	• Myocardial disarray & coronary microvascular dysfunction • Asymmetric septal hypertrophy with dynamic LVOT obstruction • LV stiffening with diastolic dysfunction • Increased risk of ventricular arrhythmias & SCD
Clinical presentation	• Often asymptomatic • Exertional dyspnea, angina, palpitations, light-headedness, syncope • Systolic murmur that increases in intensity with ↓ LV blood volume • Audible S4
LV = left ventricular; **LVOT** = left ventricular outflow tract; **SCD** = sudden cardiac death.	

Sudden cardiac death (SCD) in a young patient with a family history of SCD and autopsy findings of asymmetric septal hypertrophy is highly suggestive of **hypertrophic cardiomyopathy** (HCM). HCM is an **autosomal dominant** genetic disorder caused by mutations in genes encoding **myocardial contractile proteins** of the sarcomere. The most common mutations are single-point missense mutations affecting myosin-binding protein C and **beta-myosin heavy chain**.

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Sudden cardiac death (SCD) in a young patient with a family history of SCD and autopsy findings of asymmetric septal hypertrophy is highly suggestive of **hypertrophic cardiomyopathy** (HCM). HCM is an **autosomal dominant** genetic disorder caused by mutations in genes encoding **myocardial contractile proteins** of the sarcomere. The most common mutations are single-point missense mutations affecting myosin-binding protein C and **beta-myosin heavy chain**.

SCD in HCM typically results from **ventricular arrhythmia**, which is encouraged by the following:

- Conduction disturbances caused by cardiomyocyte disarray altering the spatial relationship of intercalated discs (the primary mediators of organized cardiac conduction)
- Myocardial ischemia resulting from increased oxygen demand of the hypertrophied myocardium and decreased oxygen supply due to both microvascular dysfunction and dynamic left ventricular outflow tract obstruction

(Choices B, C, D, and E) X-linked recessive mutations affecting the dystrophin protein are responsible for both Duchenne and Becker muscular dystrophy and can lead to dilated cardiomyopathy. Autosomal dominant mutations affecting glycoprotein fibrillin-1 cause Marfan syndrome, which is characterized by abnormalities of the skeleton (eg, disproportionately long extremities, pectus deformity, scoliosis), eyes (lens dislocation), and cardiovascular system (eg, aortic root dilation, aortic regurgitation). Autosomal dominant or recessive mutations affecting inward rectifier potassium channels cause long QT syndrome. Acquired mutations affecting the transthyretin protein cause cardiac amyloidosis and resulting restrictive or dilated cardiomyopathy in older patients (eg, age >60). All these conditions can cause SCD, but none involve *asymmetric septal hypertrophy*.

Educational objective:
Hypertrophic cardiomyopathy (HCM) is an autosomal dominant inherited condition resulting from mutations affecting genes that encode for myocardial contractile proteins of the sarcomere; myosin-binding protein C and beta-myosin heavy chain are the most commonly affected proteins. HCM is characterized by asymmetric septal hypertrophy and increased risk of sudden cardiac death.

References

- The molecular genetic basis for hypertrophic cardiomyopathy.

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A 56-year-old man comes to the hospital due to 24 hours of burning substernal chest pain. The most intense pain occurred approximately 6 hours ago, and it has started to subside. The patient has a history of diabetes mellitus. ECG shows sinus rhythm with ST-segment elevation in leads V3 to V5. In the cardiac catheterization laboratory, he is found to have total occlusion of the left anterior descending artery. Successful intervention is performed to restore blood flow in the artery. The next day, the patient's echocardiogram shows a normal-sized left ventricle with no evidence of hypertrophy; there is apical and anterior akinesia with a reduced left ventricular ejection fraction of 38%. Which of the following is the most likely long-term effect of ACE inhibitor therapy in this patient?

- A. Attenuation of left ventricular chamber dilation
- B. Enhanced collagen deposition in the apical myocardium
- C. Enhanced collagen deposition in the peri-infarct myocardium
- D. Enhanced left ventricular hypertrophy in basal segments
- E. Increase in left ventricular stroke work

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- ✓ A. Attenuation of left ventricular chamber dilation (68%)
- ✗ B. Enhanced collagen deposition in the apical myocardium (3%)
- C. Enhanced collagen deposition in the peri-infarct myocardium (6%)
- D. Enhanced left ventricular hypertrophy in basal segments (3%)
- E. Increase in left ventricular stroke work (17%)

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Correct answer
A

68%
Answered correctly

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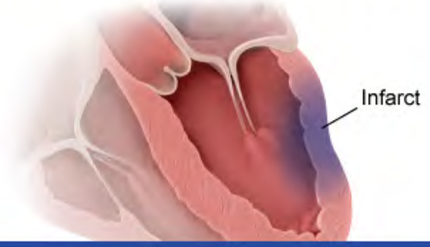
Explanation

Post–myocardial infarction ACE inhibition



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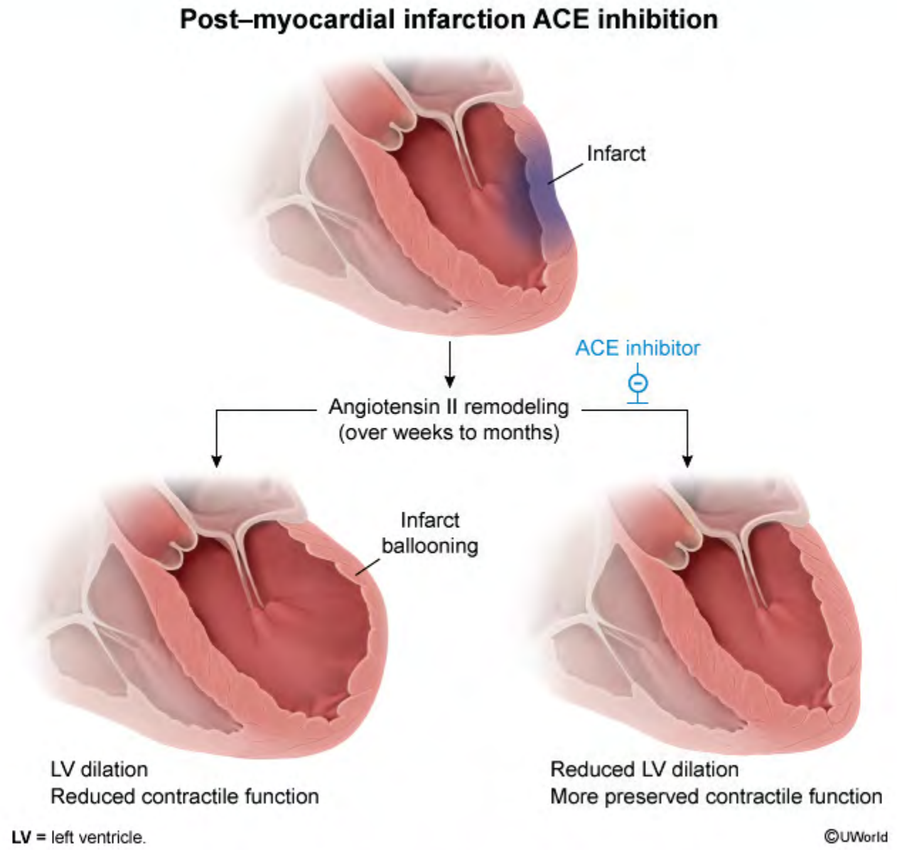

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LV = left ventricle.

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This patient has a **reduced left ventricular (LV) ejection fraction** (<45%) due to acute **myocardial infarction**; echocardiography shows akinesia of the anterior and apical segments, findings consistent with infarction in those locations. In the months following infarction, the affected myocardium undergoes fibroblastic proliferation and fibrosis to repair the necrotic region (ie, initial **remodeling** phase); disproportionate thinning of the infarcted area can lead to ballooning of affected segments and marked **LV cavity enlargement**. Although LV cavity enlargement is initially beneficial as it acts as a compensatory mechanism for LV volume overload, progressive cavity enlargement (eg, eccentric hypertrophy) leads to overwhelming wall stress and further worsening of LV contractile dysfunction. With progressive dilation, the LV begins to assume a spherical, rather than its normal elliptical, shape.

Much of the deleterious remodeling that occurs following myocardial infarction is likely driven by neurohormonal signaling via **angiotensin II**. Accordingly, **ACE inhibitors** (eg, lisinopril) **reduce the deleterious remodeling** that takes place following myocardial infarction, **minimizing LV dilation** and helping preserve LV function.

(Choices B and C) Angiotensin II enhances post–myocardial infarction collagen deposition to contribute to deleterious cardiac remodeling. This effect is especially prominent in peri-infarct cardiomyocytes (cardiomyocytes at the edge of an infarction), which have high angiotensin II levels. ACE inhibitors reduce deleterious cardiac remodeling at least in part via inhibition of myocardial collagen deposition and fibrosis.

(Choice D) Noninfarcted cardiac segments (eg, the basal segments in this patient) are subject to postinfarction remodeling caused by LV hemodynamic influences (eg, pressure and volume changes). ACE inhibitors also reduce remodeling of these segments, further contributing to maintenance of the normal LV elliptical shape and preservation of LV contractile function.

(Choice E) Stroke work is the work done by the left ventricle with each contraction and is estimated by the product of stroke volume and LV systolic pressure (ie, stroke volume × LV systolic pressure). ACE inhibitors reduce stroke work by causing peripheral vasodilation and reducing systemic blood pressure (ie, reducing the pressure the left ventricle must overcome during each contraction).

Educational objective:
Following myocardial infarction, collagen deposition and fibrosis within the infarct region and eccentric hypertrophy

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Educational objective:
Following myocardial infarction, collagen deposition and fibrosis within the infarct region and eccentric hypertrophy of surviving myocytes can lead to progressive left ventricular (LV) dilation and worsening contractile dysfunction. Because much of this remodeling is driven by neurohormonal signaling via angiotensin II, ACE inhibitors (eg, lisinopril) reduce deleterious cardiac remodeling following myocardial infarction, minimizing LV dilation and helping preserve contractile function.

Pharmacology	Cardiovascular System	ACE inhibitors
Subject	System	Topic

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Reduced contractile function

More preserved contractile function

LV = left ventricle

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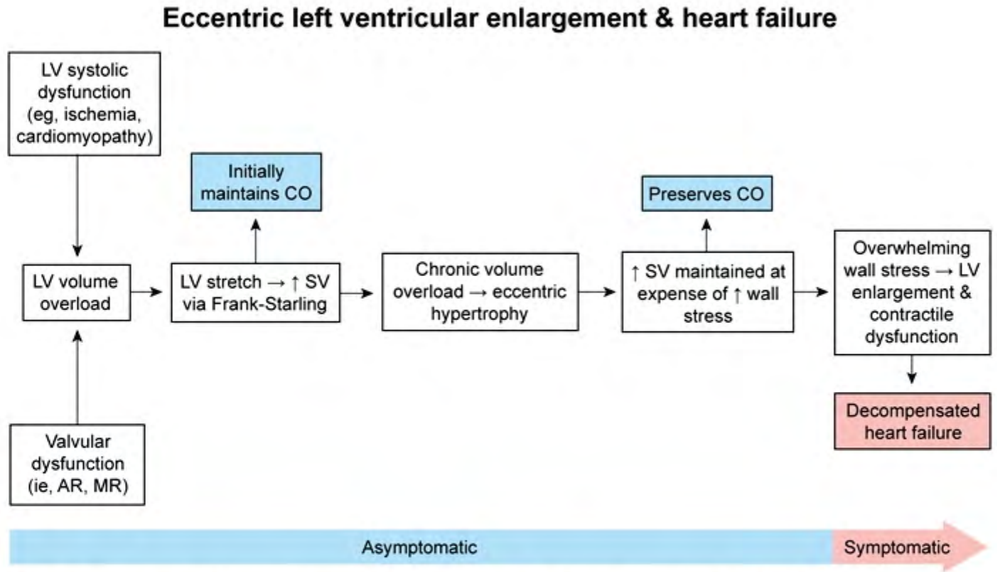

AR = aortic regurgitation; **CO** = cardiac output; **LV** = left ventricular; **MR** = mitral regurgitation; **SV** = stroke volume.
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Educational objective:
Following myocardial infarction, collagen deposition and fibrosis within the infarct region and eccentric hypertrophy

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A 38-year-old man dies suddenly at his workplace and cannot be resuscitated. He had experienced chest discomfort, shortness of breath, and fatigue in the week preceding the event. The man was overweight but otherwise had no significant medical history. He had no established primary care physician. He did not use alcohol, tobacco, or illicit drugs. Both parents are alive and healthy; a maternal uncle reportedly died of a heart attack at age 50. Autopsy was performed and myocardial findings are shown in the slide below.

Which of the following is the most likely diagnosis?

- A. Acute myocarditis
- B. Cardiac amyloidosis
- C. Congenital long QT syndrome
- D. Early-onset coronary artery disease
- E. Hypertrophic cardiomyopathy

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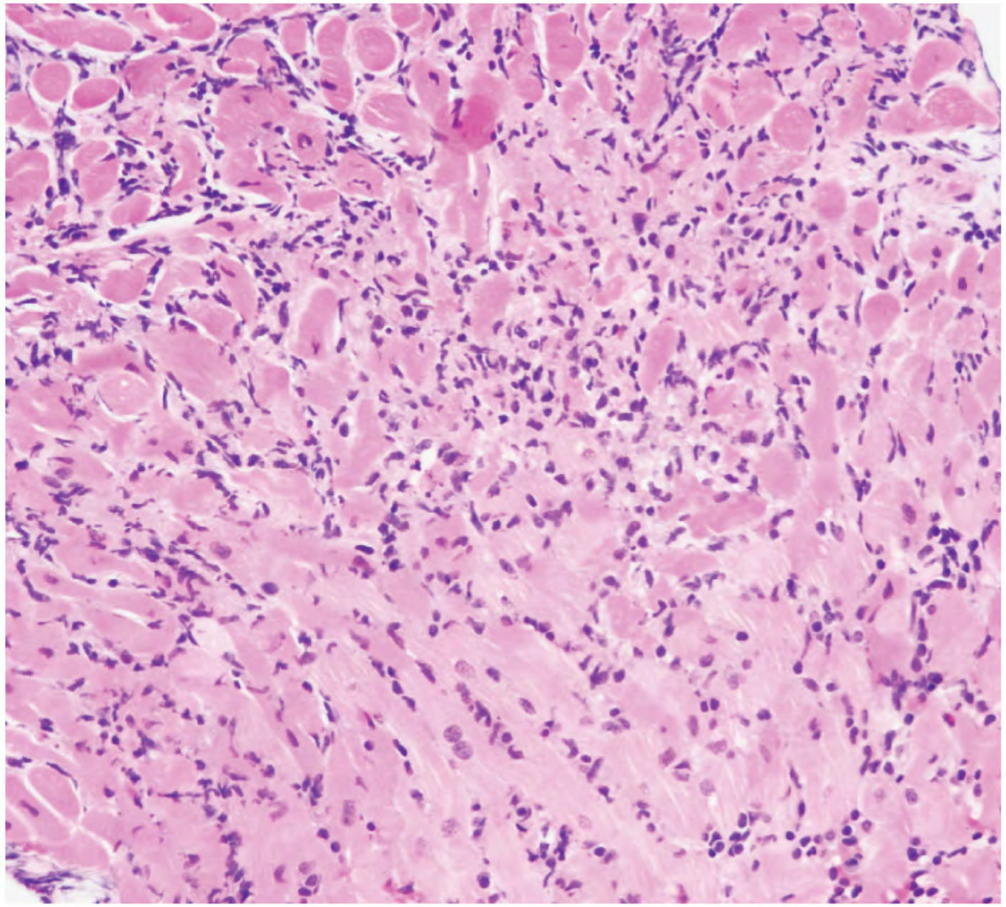

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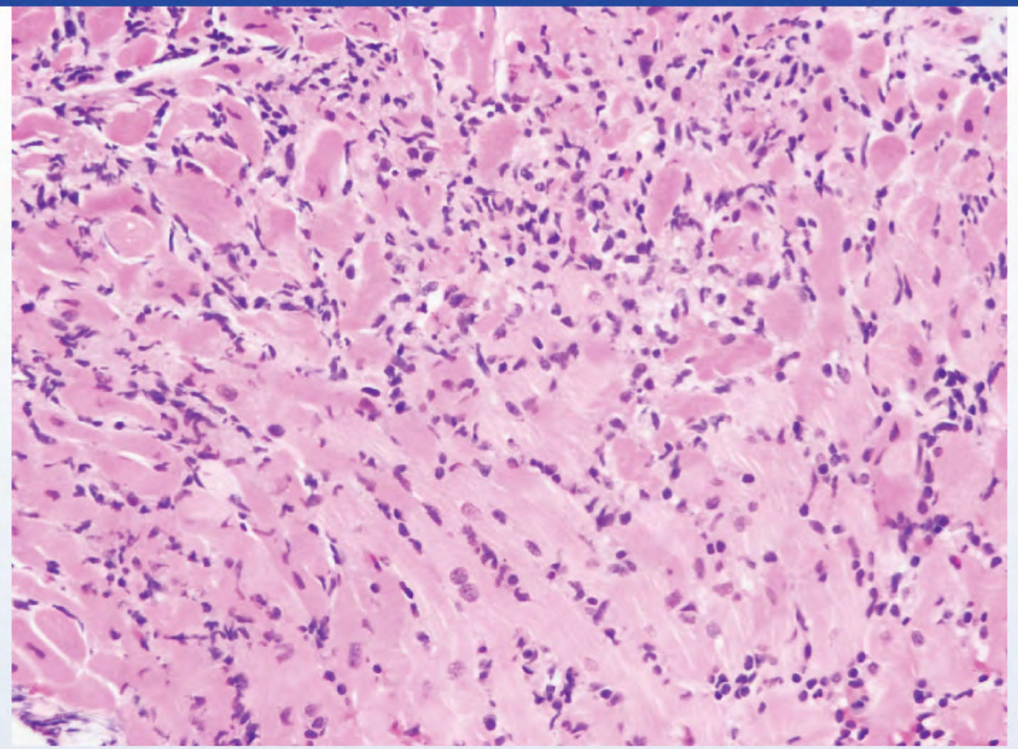

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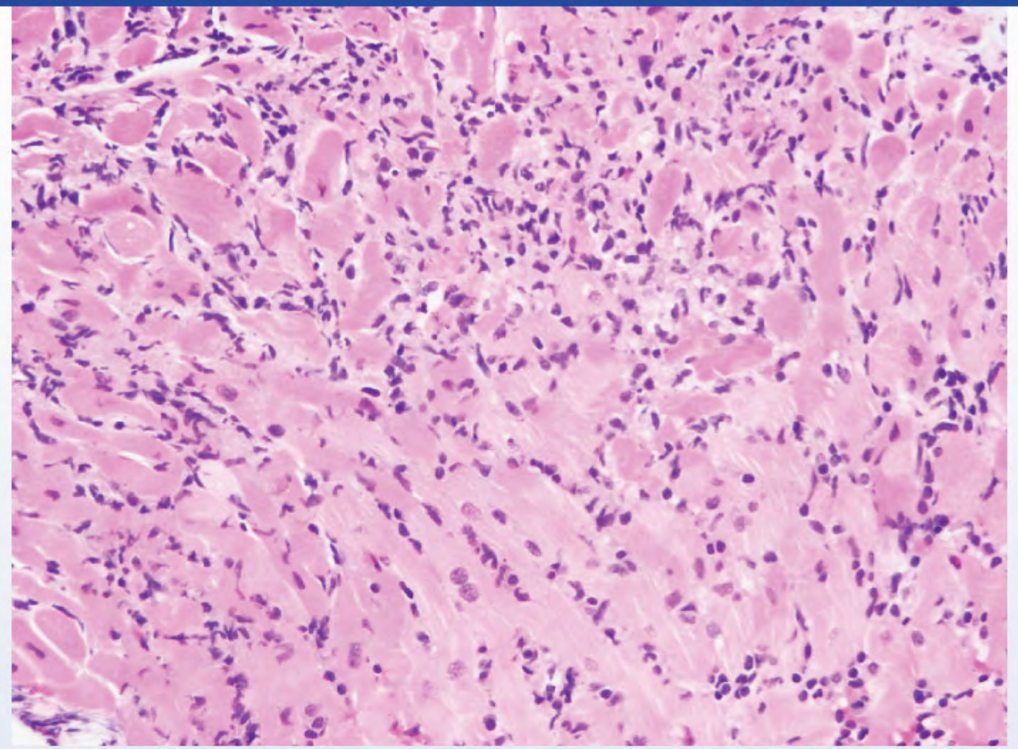

Which of the following is the most likely diagnosis?

- ✓ A. Acute myocarditis (42%)
- ✗ B. Cardiac amyloidosis (16%)
- C. Congenital long QT syndrome (2%)
- D. Early-onset coronary artery disease (5%)
- E. Hypertrophic cardiomyopathy (31%)

Incorrect
Correct answer
A

42%
Answered correctly

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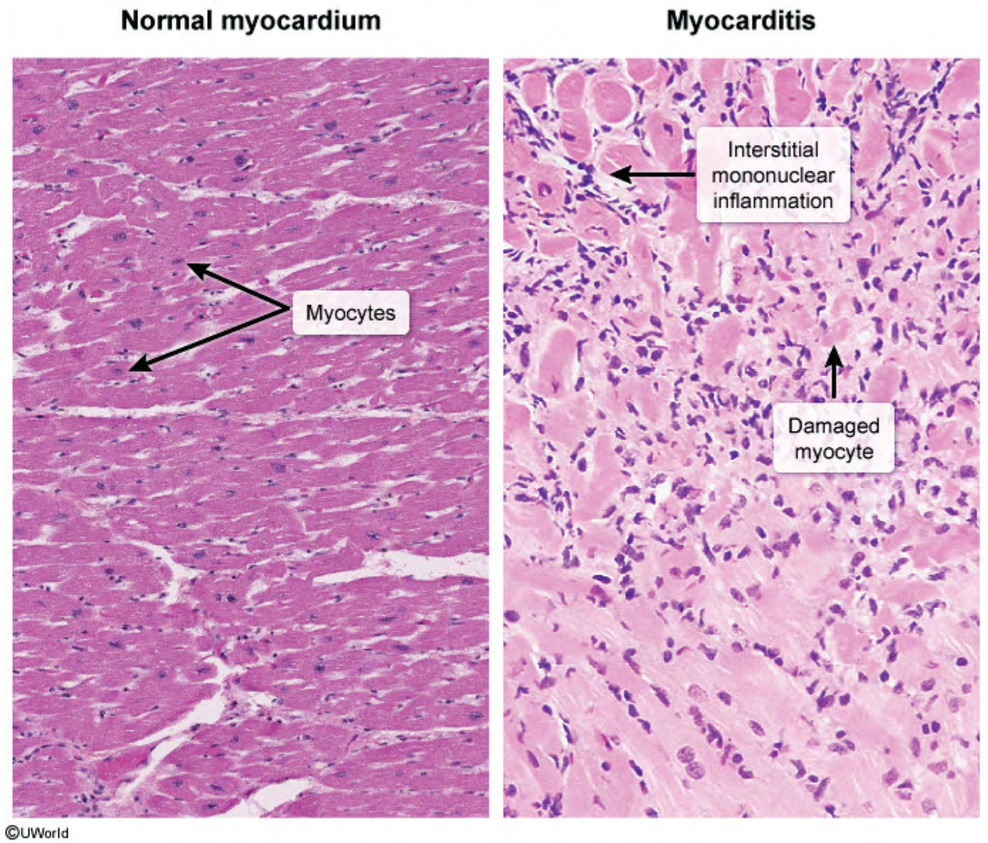

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viruses, including coxsackievirus, adenovirus, influenza, and HIV have been implicated. Histopathology of viral myocarditis typically shows **myocyte necrosis or degeneration** accompanied by an **inflammatory mononuclear**

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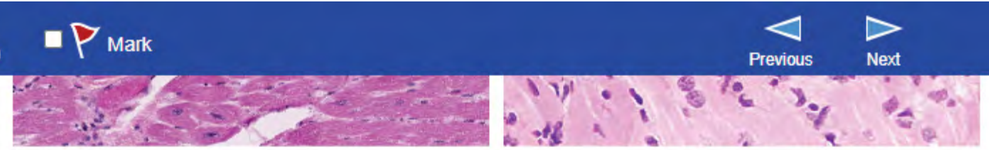

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This patient likely died from **acute myocarditis**, a condition most often caused by **viral infection**. Numerous viruses, including coxsackievirus, adenovirus, influenza, and HIV have been implicated. Histopathology of viral myocarditis typically shows **myocyte necrosis or degeneration** accompanied by an **inflammatory mononuclear cell infiltrate** primarily consisting of lymphocytes with some macrophages. Other less common etiologies of acute myocarditis, including bacterial, parasitic, and rheumatologic, can have a similar histologic appearance but may demonstrate additional findings (eg, granulomas in mycobacterial disease, visible organisms in parasitic disease).

The clinical presentation of acute myocarditis can vary significantly; many patients remain asymptomatic with subclinical infection, but some experience severe complications including decompensated **heart failure** (eg, dyspnea, fatigue, peripheral edema) due to **dilated cardiomyopathy**. Ventricular arrhythmias leading to **sudden cardiac death** (SCD) can also occur.

(Choice B) Cardiac amyloidosis involves deposition of abnormal proteins in myocardial tissue and typically manifests as restrictive cardiomyopathy initially. SCD can occur, but histopathology demonstrates pink amorphous material (amyloid) surrounding cardiomyocytes.

(Choice C) Congenital long QT syndrome can trigger ventricular arrhythmias and lead to SCD; however, histopathology is generally unremarkable and would not demonstrate myocardial necrosis with inflammatory infiltrate.

(Choice D) Coronary artery disease can lead to SCD by triggering ventricular arrhythmia, either in the setting of acute myocardial infarction or chronic ischemic heart disease. However, microscopic changes in the early stages of acute myocardial infarction include areas of coagulative necrosis and subsequent neutrophilic infiltrate, and chronic ischemic heart disease typically shows evidence of healed myocardial infarctions (eg, collagenous scars). In addition, this patient's relatively young age and short duration of preceding exertional symptoms make acute myocarditis more likely.

(Choice E) Hypertrophic cardiomyopathy is characterized by cardiac myocyte disarray and interstitial fibrosis that can predispose to ventricular arrhythmia and SCD. An inflammatory infiltrate is not expected.

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(Choice E) Hypertrophic cardiomyopathy is characterized by cardiac myocyte disarray and interstitial fibrosis that can predispose to ventricular arrhythmia and SCD. An inflammatory infiltrate is not expected.

Educational objective:
Acute myocarditis is most commonly caused by a viral infection (eg, coxsackievirus, adenovirus, influenza). It often resolves without noticeable symptoms, but patients can develop serious complications, including decompensated heart failure due to dilated cardiomyopathy or sudden cardiac death due to ventricular arrhythmia. Histopathology typically demonstrates myocyte necrosis with inflammatory mononuclear infiltrate.

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Educational objective:

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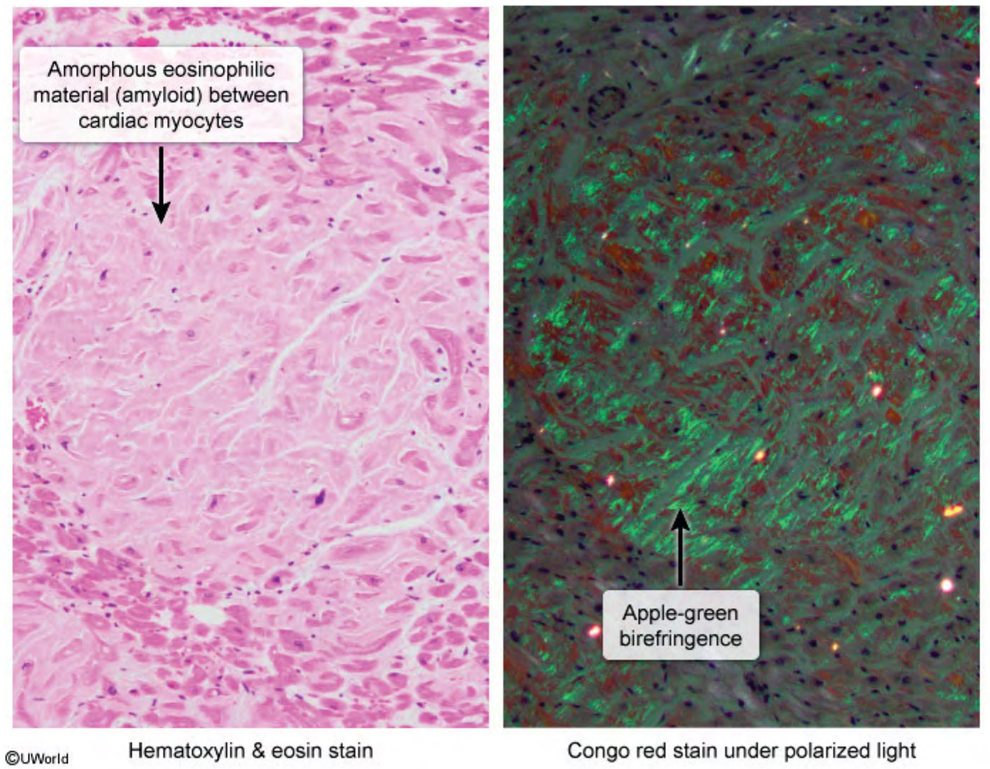

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Educational objective:
Acute myocarditis is most commonly caused by a viral infection (eg, coxsackievirus, adenovirus, influenza). It

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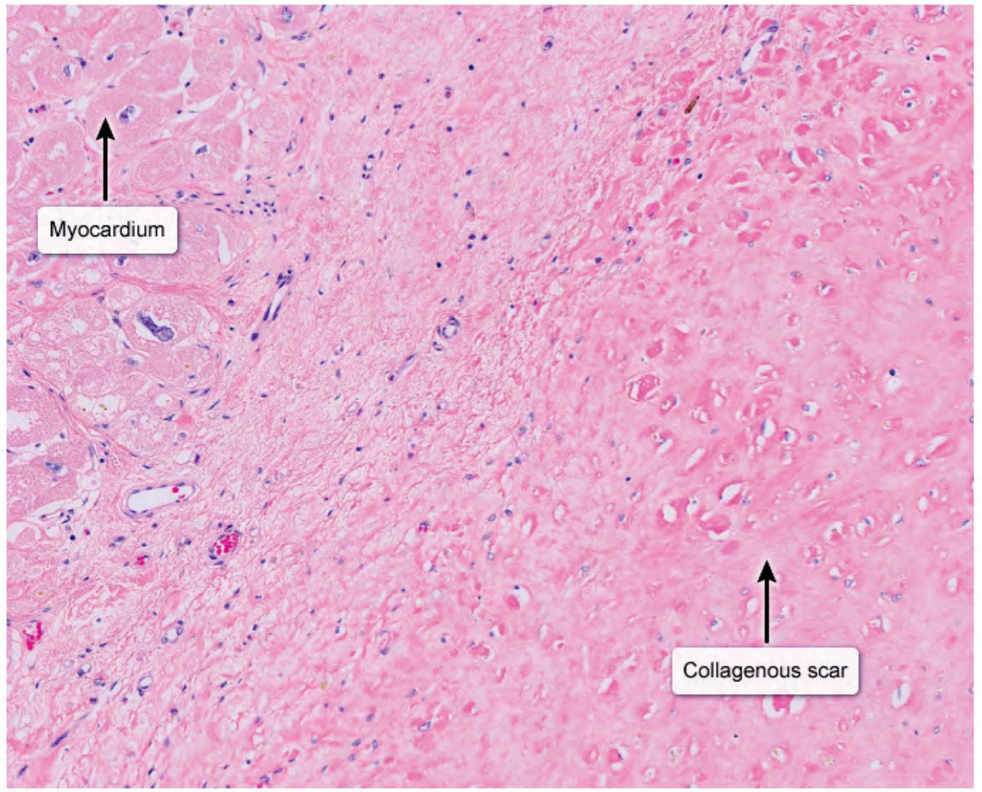
Healed myocardial infarction



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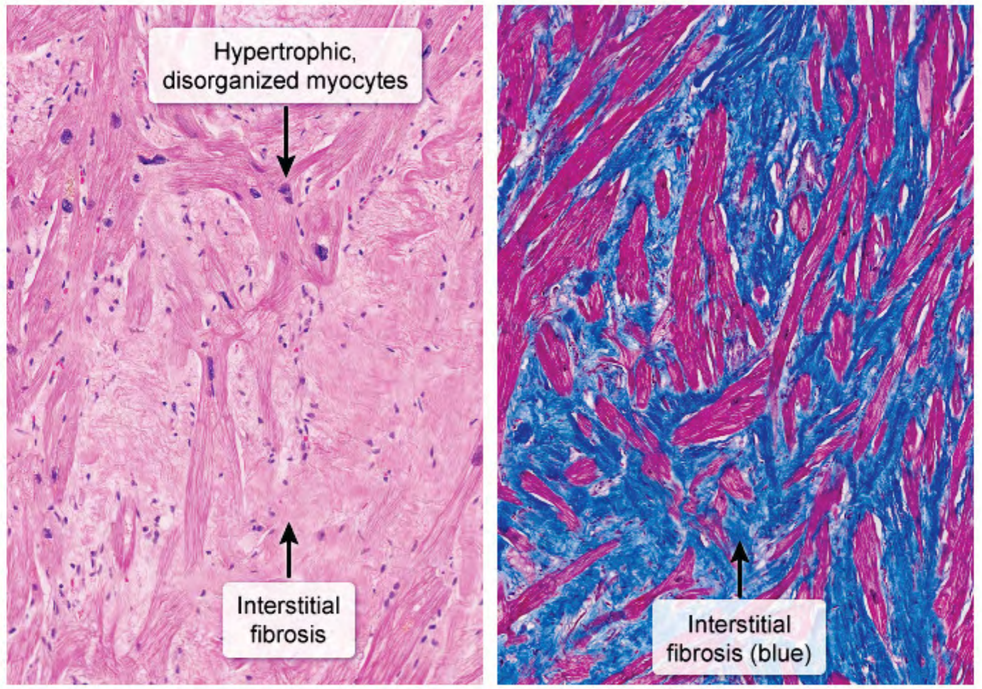
Hypertrophic cardiomyopathy



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Hematoxylin & eosin stain

Trichrome stain

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A 45-year-old man comes to the emergency department because of severe chest pain, diaphoresis, and palpitations. The patient dies two hours after the onset of his symptoms. Autopsy reveals 100% occlusion of the left anterior descending artery. At the time of the patient's death, light microscopy of the affected myocardium would most likely demonstrate which of the following?

- A. Edema and punctate hemorrhages
- B. Myocyte hypereosinophilia
- C. Dense interstitial neutrophil infiltrate
- D. Extensive macrophage phagocytosis of the dead cells
- E. Fibrovascular granulation tissue with neovascularization
- F. Dense collagen scar
- G. Normal myocardium

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- ✗ A. Edema and punctate hemorrhages (12%)
- B. Myocyte hypereosinophilia (7%)
- C. Dense interstitial neutrophil infiltrate (9%)
- D. Extensive macrophage phagocytosis of the dead cells (1%)
- E. Fibrovascular granulation tissue with neovascularization (0%)
- F. Dense collagen scar (0%)
- ✓ G. Normal myocardium (67%)

Incorrect
Correct answer
G

67%
Answered correctly

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Explanation

Time after myocardial infarction	Predominant light microscopic changes
0-4 hrs	minimal change
4-12 hrs	early coagulation necrosis, edema, hemorrhage, wavy fibers
12-24 hrs	coagulation necrosis and marginal contraction band necrosis
1 to 5 days	coagulation necrosis and neutrophilic infiltrate
5 to 10 days	macrophage phagocytosis of dead cells

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Answered correctly

Time Spent

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Explanation

Time after myocardial infarction	Predominant light microscopic changes
0-4 hrs	minimal change
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1 to 5 days	coagulation necrosis and neutrophilic infiltrate
5 to 10 days	macrophage phagocytosis of dead cells
10 to 14 days	granulation tissue and neovascularization
2 weeks to 2 months	collagen deposition / scar formation

The cause of death was most likely an acute myocardial infarction. In myocardial infarction, changes on light microscopy are usually not apparent until 4 hours after the onset of severe ischemia. Although a variable waviness of myofibrils at the border of the infarct (due to myofibril relaxation) might be observed before this, more definite signs of early coagulative necrosis, such as cytoplasmic eosinophilia and nuclear pyknosis, take at least 4 hours to develop.

Other potentially lethal causes of chest pain, diaphoresis, and palpitations/tachycardia include aortic dissection and/or rupture, massive pulmonary embolism, and tension pneumothorax. The chest pain of aortic dissection is typically tearing in nature and often radiates to the back. Dyspnea is typically the most prominent symptom in the event of a pulmonary embolus or tension pneumothorax.

(Choices A & B) Cytoplasmic hypereosinophilia on light microscopy is one of the earliest signs of coagulative necrosis of cardiac myocytes. It begins approximately 4 hours after the onset of lethal ischemia. Light microscopy may also reveal edema and punctate hemorrhages in infarcted myocardium starting also at about 4 hours after the

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(Choices A & B) Cytoplasmic hypereosinophilia on light microscopy is one of the earliest signs of coagulative necrosis of cardiac myocytes. It begins approximately 4 hours after the onset of lethal ischemia. Light microscopy may also reveal edema and punctate hemorrhages in infarcted myocardium starting also at about 4 hours after the ischemic event.

(Choice C) An interstitial infiltrate of neutrophils around a zone of myocardial infarction is not seen until 1 to 3 days after the onset of severe ischemia.

(Choice D) Extensive macrophage phagocytosis of the dead cells generally does not develop until at least 5 days after myocardial infarction, and is most prominent during post-infarction days 7 to 10.

(Choice E) Fibrovascular granulation tissue with neovascularization generally begins to develop 7 days after myocardial infarction, and is most prominent on post-infarction days 10 to 14.

(Choice F) Increased collagen deposition and decreased cellularity in a zone of infarcted myocardium is not generally evident until 2 weeks post-infarction. Fibrosis continues during weeks 2 to 8, producing a dense collagenous scar by two months post-infarction.

Educational objective:
After the onset of severe ischemia leading to myocardial infarction (MI), early signs of coagulative necrosis do not become apparent on light microscopy until 4 hours after the onset of MI.

Pathology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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A 60-year-old man comes to the office for follow-up due to persistent atrial fibrillation. He reports occasional palpitations and poor exercise tolerance. For the past year, the patient's atrial fibrillation has been managed with a rate-control strategy using metoprolol. Three months ago, his metoprolol dosage was increased for improved rate control, but today in the office, he reports that he was unable to tolerate the new dosage due to dizziness. Blood pressure is 110/70 mm Hg and pulse is 105/min and irregular. Examination shows no abnormalities. Echocardiography reveals left atrial enlargement, an ejection fraction of 59%, and no significant valvular disease. The patient is advised to remain on his previously tolerated metoprolol dose, and digoxin is added to the medication regimen. Four weeks later, he reports symptom improvement. Resting pulse is 84/min and irregular. Which of the following best explains digoxin's effect on heart rate in this patient?

- A. Decreased atrial refractoriness
- B. Decreased cardiomyocyte action potential duration
- C. Decreased Purkinje fiber automaticity
- D. Increased parasympathetic tone
- E. Increased ventricular contractility

Submit

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- ✗ A. Decreased atrial refractoriness (5%)
- B. Decreased cardiomyocyte action potential duration (6%)
- C. Decreased Purkinje fiber automaticity (4%)
- ✓ D. Increased parasympathetic tone (55%)
- E. Increased ventricular contractility (29%)

Incorrect
Correct answer
D

55%
Answered correctly

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Explanation

Long-term management of atrial fibrillation	
Rate-control strategy*	**Rhythm-control strategy***
• Atrioventricular node blockade ◦ Beta blockers	• Electrical cardioversion • Pharmacologic antiarrhythmics

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Long-term management of atrial fibrillation	
Rate-control strategy*	**Rhythm-control strategy***

• Atrioventricular node blockade
  ◦ Beta blockers
  ◦ Nondihydropyridine CCBs
  ◦ Digoxin
• Anticoagulation usually indicated
	• Electrical cardioversion
	• Pharmacologic antiarrhythmics
	  ◦ Flecainide, amiodarone
	• Radiofrequency ablation
	• Anticoagulation often indicated

*Clinical outcomes for each strategy are roughly equivalent. Choice of strategy is typically based on case particulars (eg, duration of atrial fibrillation) & patient preference. **CCBs** = calcium channel blockers.	

Adequate **rate control** (eg, resting heart rate <85/min) is important in **atrial fibrillation** because it minimizes symptoms and helps decrease the risk of tachycardia-induced cardiomyopathy. Beta blockers (eg, metoprolol) **slow conduction** through the **atrioventricular (AV) node** via inhibition of sympathetic stimulation and are typically first-line therapy for rate control in patients with atrial fibrillation. This patient was unable to tolerate the dose of metoprolol needed to maintain an appropriate resting heart rate; therefore, **digoxin** was added for increased rate control. Digoxin slows conduction through the AV node via **increased parasympathetic (vagal) tone**.

Several mechanisms may explain the parasympathetic effects of digoxin. Inhibition of the sodium-potassium ATPase pump in vagal afferent fibers sensitizes arterial baroreceptors (eg, carotid, aortic) and cardiac volume receptors, augmenting afferent input from the cardiovascular system to the brain. In addition, digoxin enhances efferent parasympathetic ganglionic transmission, leading to **increased vagal output**.

This mechanism of increased parasympathetic tone is effective in controlling heart rate at rest but is limited in controlling heart rate during exercise, when sympathetic tone is high. Therefore, digoxin is rarely used as monotherapy for rate control and is more typically combined with a beta blocker or nondihydropyridine calcium channel blocker (eg, verapamil, diltiazem).

(Choices A and B) Decreased atrial refractoriness and decreased cardiomyocyte action potential duration both occur with digoxin, increasing the automaticity of the atrial myocardium and encouraging the development of atrial

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(Choices A and B) Decreased atrial refractoriness and decreased cardiomyocyte action potential duration both occur with digoxin, increasing the automaticity of the atrial myocardium and encouraging the development of atrial fibrillation and other atrial arrhythmias. However, these effects are typically problematic only at toxic digoxin levels and at therapeutic levels are outweighed by the beneficial effect of increased parasympathetic tone.

(Choice C) Digoxin increases (rather than decreases) Purkinje fiber automaticity, encouraging the development of ventricular arrhythmias (eg, ventricular tachycardia). Like the proarrhythmic effects on the atrial myocardium, this effect is typically problematic only at toxic digoxin levels.

(Choice E) Digoxin increases ventricular contractility (positive inotropy) by inhibiting the sodium-potassium ATPase pump to increase intracellular sodium and consequently increase intracellular calcium. This effect can improve cardiac output and symptoms (eg, dyspnea, orthopnea) in patients with left ventricular systolic dysfunction, but it does not contribute to improved rate control.

Educational objective:
Digoxin can be used to improve ventricular rate control in patients with atrial fibrillation, as it decreases atrioventricular node conduction by increasing parasympathetic (vagal) tone. Digoxin also has a positive inotropic effect that can be useful in patients with left ventricular systolic dysfunction.

References

- Digoxin in the management of cardiovascular disorders.

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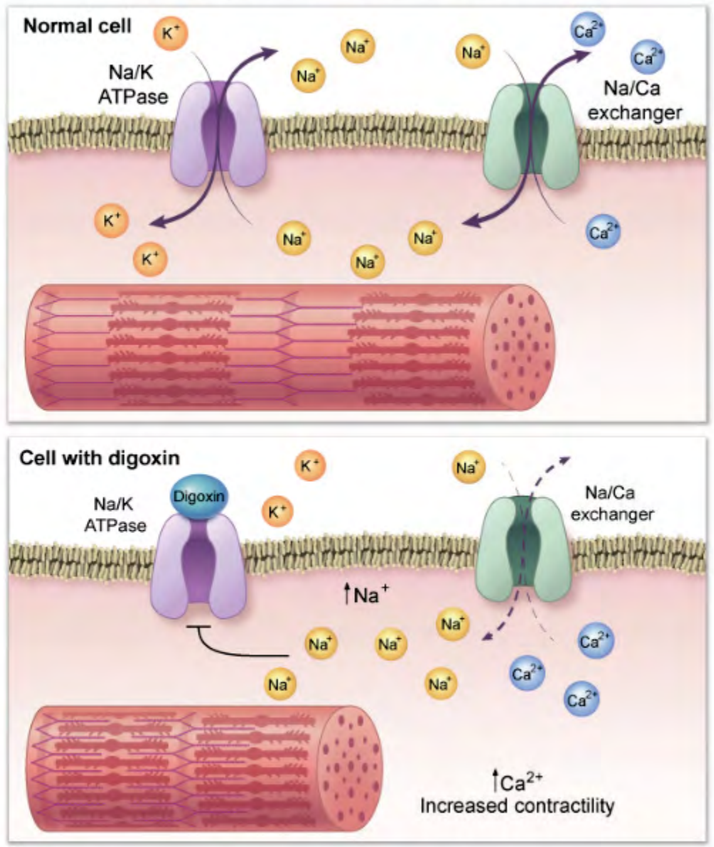
Digoxin mechanism of action



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A 56-year-old man is admitted to a tertiary care center after being involved in a high-speed motor vehicle accident. The patient was thrown from his vehicle and suffered severe head trauma along with multiple fractures involving his ribs and extremities. Despite aggressive treatment, he dies the following day from massive cerebral edema and brainstem herniation. Autopsy shows the following heart findings:

Left atrium: enlarged
Left ventricular myocardial mass: increased
Left ventricular wall thickness: increased

The structural changes observed in this patient's heart are most likely associated with which of the following conditions?

- A. Atrial septal defect
- B. Long-standing hypertension
- C. Mitral insufficiency
- D. Mitral stenosis
- E. Normal aging
- F. Recent myocardial infarction
- G. Wolff-Parkinson-White syndrome

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A 56-year-old man is admitted to a tertiary care center after being involved in a high-speed motor vehicle accident. The patient was thrown from his vehicle and suffered severe head trauma along with multiple fractures involving his ribs and extremities. Despite aggressive treatment, he dies the following day from massive cerebral edema and brainstem herniation. Autopsy shows the following heart findings:

Left atrium: enlarged
Left ventricular myocardial mass: increased
Left ventricular wall thickness: increased

The structural changes observed in this patient's heart are most likely associated with which of the following conditions?

- A. Atrial septal defect (1%)
- ✓ B. Long-standing hypertension (65%)
- C. Mitral insufficiency (22%)
- D. Mitral stenosis (6%)
- E. Normal aging (2%)
- F. Recent myocardial infarction (0%)
- G. Wolff-Parkinson-White syndrome (0%)

Correct | 65% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Concentric & eccentric left ventricular hypertrophy*

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E. Normal aging (2%)

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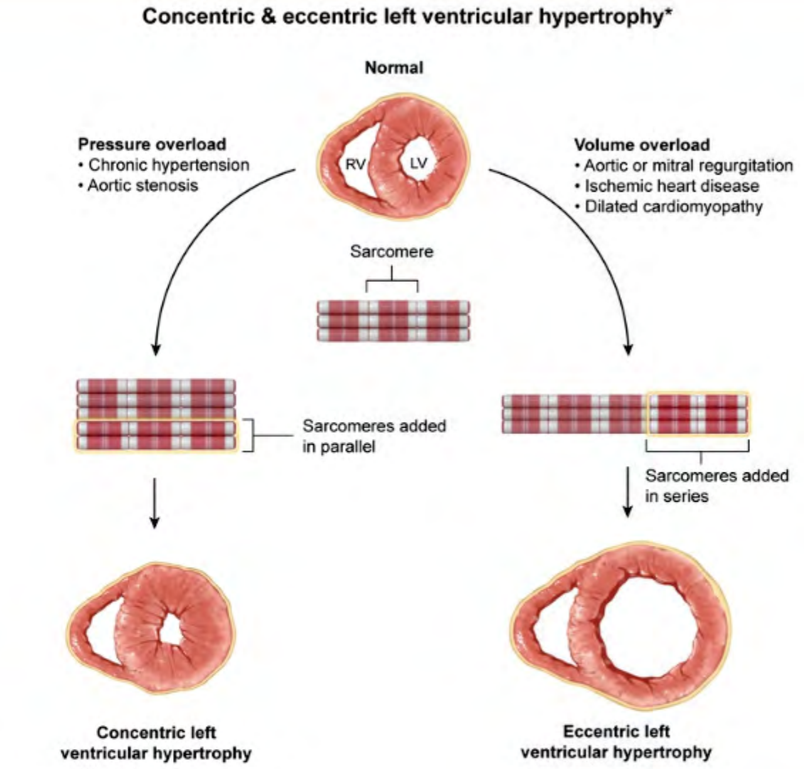

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*The right ventricle undergoes similar adaptation when exposed to chronic pressure or volume overload.
LV = left ventricle; **RV** = right ventricle.

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The autopsy findings of this patient's left ventricle (LV) are consistent with **concentric hypertrophy** most likely due to chronic hypertension.

LV hypertrophy is generally defined as an increase in the left ventricular mass due to **increased wall thickness** (concentric) or cavity size (eccentric). Concentric hypertrophy is characterized by **uniform** thickening of the ventricular wall with the outer dimensions of the ventricle remaining almost unchanged, resulting in a narrowed ventricular cavity size. It is due to chronic elevation of ventricular pressures during systole, which is usually caused by **long-standing hypertension** or aortic stenosis (**increased LV afterload**). Patients may develop diastolic dysfunction with left atrial enlargement and congestive heart failure due to impaired ventricular compliance and filling.

(Choice A) An atrial septal defect causes a shunting of blood flow from the left to right atrium. This typically results in volume overload of the right ventricle, producing right atrial enlargement and eccentric (dilated) right ventricular hypertrophy.

(Choice C) Mitral insufficiency causes LV volume overload as regurgitant flow through the mitral valve during systole increases the amount of blood returning to the LV during diastole. The net structural consequence of mitral insufficiency is enlargement of the LV cavity and eccentric hypertrophy.

(Choice D) Mitral stenosis leads to left atrial enlargement due to elevated pressure in the left atrium. The LV cavity is typically small/normal in size with normal wall thickness due to restriction of blood flow across the mitral valve.

(Choice E) Aging is associated with a decrease in LV chamber size, predominantly through shortening of the apex-to-base dimension accompanied by a leftward bowing of the lower interventricular (sigmoid) septum. The aortic valve may also become calcified, resulting in aortic stenosis; however, structural cardiac changes due to aging are generally not prominent before age 65.

(Choice F) The ventricular remodeling that occurs after an acute myocardial infarction involves expansion, thinning, and fibrous healing of the infarcted zone of myocardium. Regional dysfunction of the infarcted myocardium causes volume overload for the remaining viable myocardium. The net result is usually eccentric

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filling.

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(Choice C) Mitral insufficiency causes LV volume overload as regurgitant flow through the mitral valve during systole increases the amount of blood returning to the LV during diastole. The net structural consequence of mitral insufficiency is enlargement of the LV cavity and eccentric hypertrophy.

(Choice D) Mitral stenosis leads to left atrial enlargement due to elevated pressure in the left atrium. The LV cavity is typically small/normal in size with normal wall thickness due to restriction of blood flow across the mitral valve.

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(Choice F) The ventricular remodeling that occurs after an acute myocardial infarction involves expansion, thinning, and fibrous healing of the infarcted zone of myocardium. Regional dysfunction of the infarcted myocardium causes volume overload for the remaining viable myocardium. The net result is usually eccentric hypertrophy, with enlargement of the LV cavity.

(Choice G) Patients with Wolff-Parkinson-White syndrome have an accessory pathway that bypasses the atrioventricular node and directly connects the atria and ventricles. This results in ventricular preexcitation and the development of tachyarrhythmias. The LV usually appears grossly normal.

Educational objective:
Concentric hypertrophy is characterized by uniform thickening of the ventricular wall and narrowing of the ventricular cavity due to increased afterload (eg, chronic hypertension, aortic stenosis). Eccentric hypertrophy is characterized by reduced ventricular wall thickness with an associated increase in chamber size due to volume overload.

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A newborn girl is being evaluated in the nursery. The patient was born at 39 weeks gestation via spontaneous vaginal delivery following an uncomplicated pregnancy. She has been breastfeeding well and has urinated 3 times and stooled once. Approximately 1 hour after delivery, a 2/6 systolic murmur could be heard at the left upper sternal border. At 8 hours, the murmur could be heard during both systole and diastole. Examination at 24 hours revealed no murmur. Which of the following most likely occurred immediately prior to the disappearance of this patient's murmur?

- A. Closure of a ventricular left-to-right shunt
- B. Closure of a ventricular right-to-left shunt
- C. Closure of an arterial left-to-right shunt
- D. Closure of an arterial right-to-left shunt
- E. Closure of an atrial left-to-right shunt
- F. Closure of an atrial right-to-left shunt

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- ✗ A. Closure of a ventricular left-to-right shunt (4%)
- B. Closure of a ventricular right-to-left shunt (2%)
- ✓ C. Closure of an arterial left-to-right shunt (43%)
- D. Closure of an arterial right-to-left shunt (18%)
- E. Closure of an atrial left-to-right shunt (17%)
- F. Closure of an atrial right-to-left shunt (12%)

Incorrect
Correct answer
C

43%
Answered correctly

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Time Spent

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Explanation

Evolution of the pressure gradient & murmur associated with ductus arteriosus

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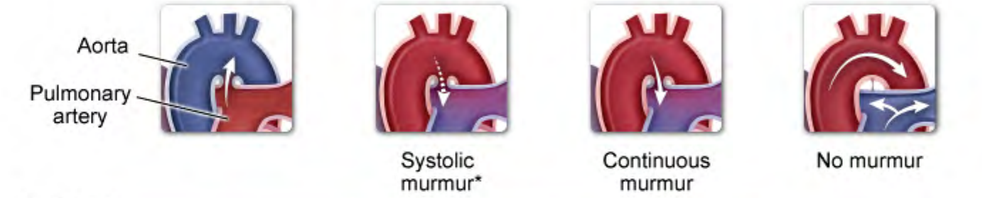
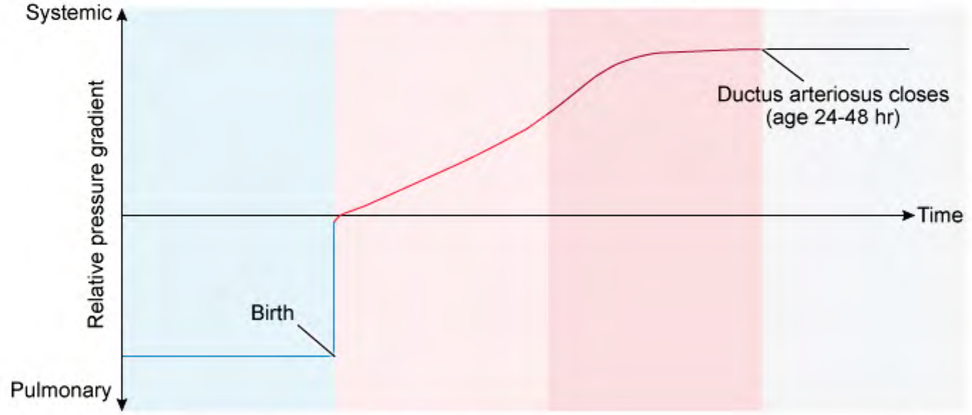
Evolution of the pressure gradient & murmur associated with ductus arteriosus





*Systemic pressure > pulmonary pressure only during systole.

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Systolic murmur* | Continuous murmur | No murmur

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The changes in this newborn's heart murmur throughout the first day of life are most likely explained by the normal evolution of the **ductus arteriosus**.

In the fetal circulation, blood flows right to left through the ductus arteriosus from the pulmonary artery to the aorta, bypassing the lungs. This occurs due to high pulmonary vascular resistance (PVR) caused by the constricted pulmonary arteries and low systemic vascular resistance (SVR) caused by the low resistance of the placenta.

After delivery, increased oxygen tension in the lungs causes pulmonary vasodilation and **reduced PVR**. At the same time, the placenta becomes disconnected from the newborn's systemic circulation, leading to **increased SVR**. These changes decrease pulmonary pressure and increase systemic pressure, reversing right-to-left blood flow through the ductus arteriosus to left-to-right blood flow.

Just after birth, pulmonary diastolic pressure is still high relative to systemic diastolic pressure, so the murmur is heard best (or only) during systole. As PVR continues to fall, systemic pressure exceeds pulmonary pressure throughout the cardiac cycle, and the classic machinery-like, **continuous murmur** is heard best at the left upper sternal border or left infraclavicular area. When the ductus arteriosus closes (usually 24-48 hr after birth), the murmur disappears.

(Choice A) Left-to-right flow through a ventricular septal defect (VSD) causes a holosystolic murmur at the left sternal border; this murmur does not have a diastolic component.

(Choices B, D, and F) Incomplete closure of the ductus arteriosus causes a patent ductus arteriosus. Long-standing left-to-right shunting through a patent ductus arteriosus (or atrial/ventricular septal defects) can lead to pulmonary vascular remodeling with gradually increased PVR and eventual reversal to right-to-left shunting (ie, Eisenmenger syndrome). However, right-to-left shunting usually causes cyanosis (not noted in this patient), and the pressure gradient is not typically large enough to cause a murmur.

(Choice E) Left-to-right flow through an ASD itself does not generate a murmur due to the low velocity of shunted blood. The characteristic murmur of an ASD is a systolic murmur at the left upper sternal border caused by increased flow through the pulmonic valve. However, this murmur does not have a diastolic component (ie, not continuous).

Educational objective:

After birth, pulmonary vascular resistance progressively decreases as systemic vascular resistance progressively

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bypassing the lungs. This occurs due to high pulmonary vascular resistance (PVR) caused by the constricted pulmonary arteries and low systemic vascular resistance (SVR) caused by the low resistance of the placenta.

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Educational objective:
After birth, pulmonary vascular resistance progressively decreases as systemic vascular resistance progressively increases. As the relative pressures in the pulmonary artery and aorta change, the murmur associated with the ductus arteriosus evolves from systolic only to continuous before the duct closes, at which time the murmur disappears.

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Pulmonary

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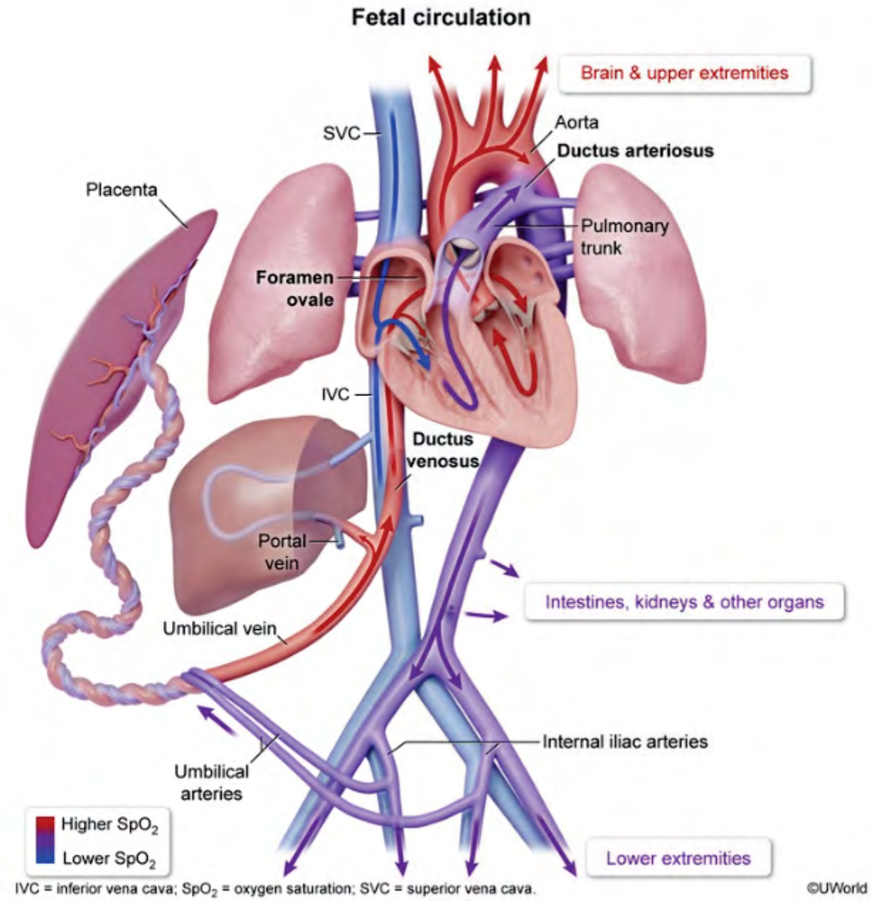

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Pulmonary

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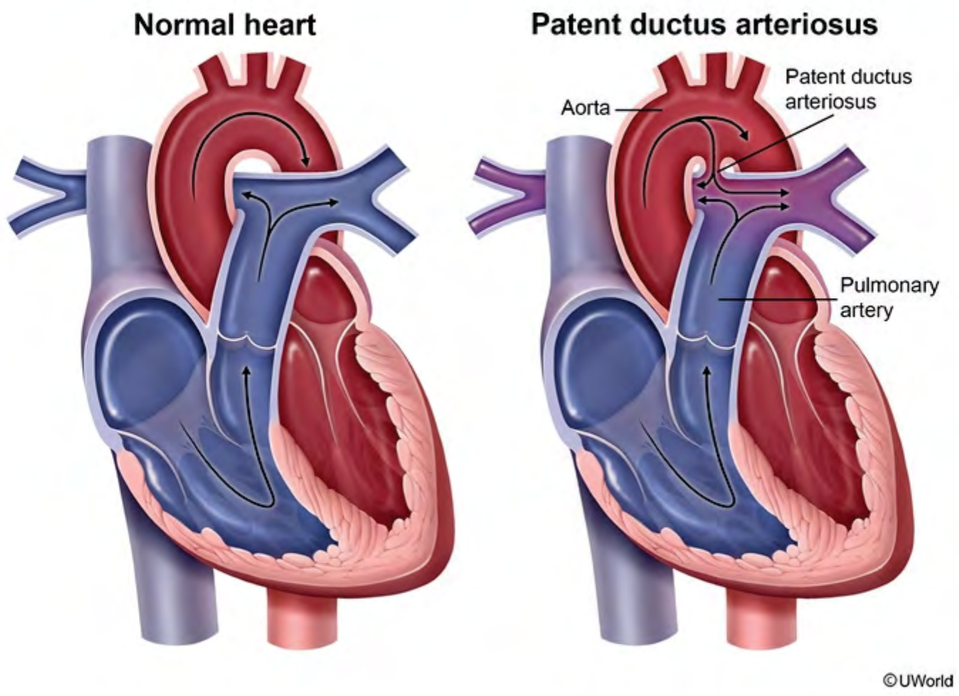

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A 62-year-old woman comes to the emergency department due to palpitations and shortness of breath that began an hour ago. Medical history includes hypertension, coronary artery disease, and paroxysmal atrial flutter. Blood pressure is 108/76 mm Hg and pulse is rapid. ECG shows atrial flutter with a ventricular rate of approximately 150/min. The estimated atrial rate based on ECG flutter waves is 300/min. Which of the following is most likely responsible for the rate difference between the atria and the ventricles?

- A. Calcium current within the atrioventricular node
- B. Calcium current within the sinoatrial node
- C. Sodium current within an atrioventricular accessory pathway
- D. Sodium current within the atria
- E. Sodium current within the ventricles

Submit

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- ✓ A. Calcium current within the atrioventricular node (51%)
- ✗ B. Calcium current within the sinoatrial node (11%)
- C. Sodium current within an atrioventricular accessory pathway (18%)
- D. Sodium current within the atria (13%)
- E. Sodium current within the ventricles (4%)

Incorrect
Correct answer
A

51%
Answered correctly

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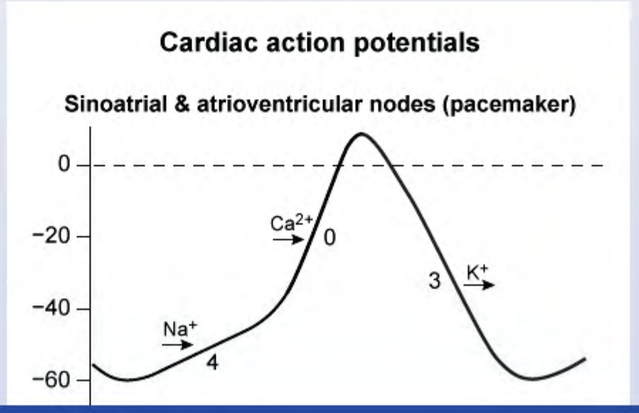
Explanation



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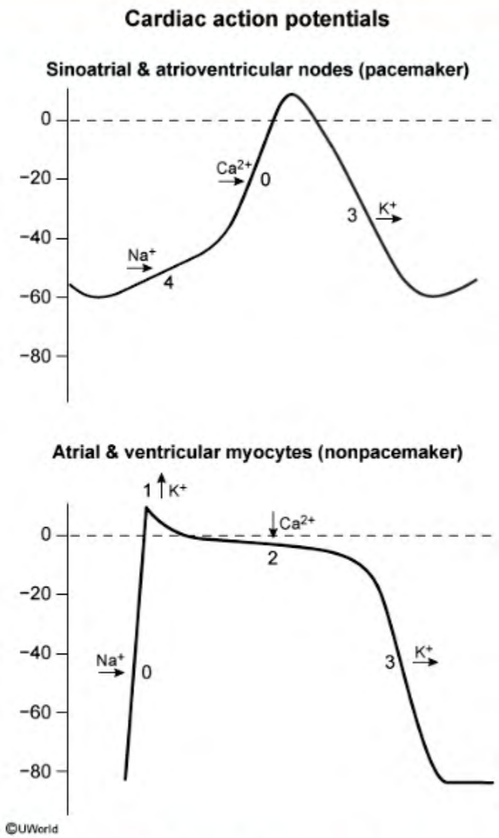

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Atrial flutter usually involves a reentrant circuit that develops around the cavotricuspid isthmus of the right atrium, resulting in **rapid atrial conduction** at a rate of approximately 300/min (ie, flutter waves on ECG). Transmission of all of these electrical impulses to the ventricles would be highly problematic as a ventricular rate of 300/min would allow little time for diastolic filling, causing a profound drop in cardiac output and likely leading to sudden cardiac arrest. However, the **atrioventricular (AV) node** reduces the transmission of impulses from the atria to the ventricles due to the **slow inward calcium current** that drives the **phase 0 depolarization** phase of action potentials through the AV node.

A similar AV node slowing effect occurs with atrial fibrillation, which can have atrial rates up to 500-600/min.

(Choice B) A slow inward calcium current also controls the phase 0 depolarization of action potentials in the sinoatrial node. However, the sinoatrial node is suppressed during atrial flutter (and atrial fibrillation) as atrial impulses are driven by abnormal foci of conduction.

(Choices C, D, and E) Fast inward sodium channels control the phase 0 depolarization of both the atrial and the ventricular myocardium, allowing for rapid conduction speeds to be generated. However, the slow calcium channels in the AV node act as a buffer, preventing the transmission of rapid impulses from the atria to the ventricles. Some patients can have an abnormal accessory pathway (driven by fast sodium channels) that directly connects the atria to the ventricles and bypasses the AV node (ie, Wolff-Parkinson-White syndrome). In such patients, rapid atrial arrhythmias can generate dangerously high ventricular rates.

Educational objective:
The slow inward calcium current within the atrioventricular node decreases the transmission rate of rapid impulses generated by the atrial myocardium (eg, atrial fibrillation/flutter), acting as a conduction rate buffer between the atria and the ventricles.

Physiology	Cardiovascular System	Atrial fibrillation
Subject	System	Topic

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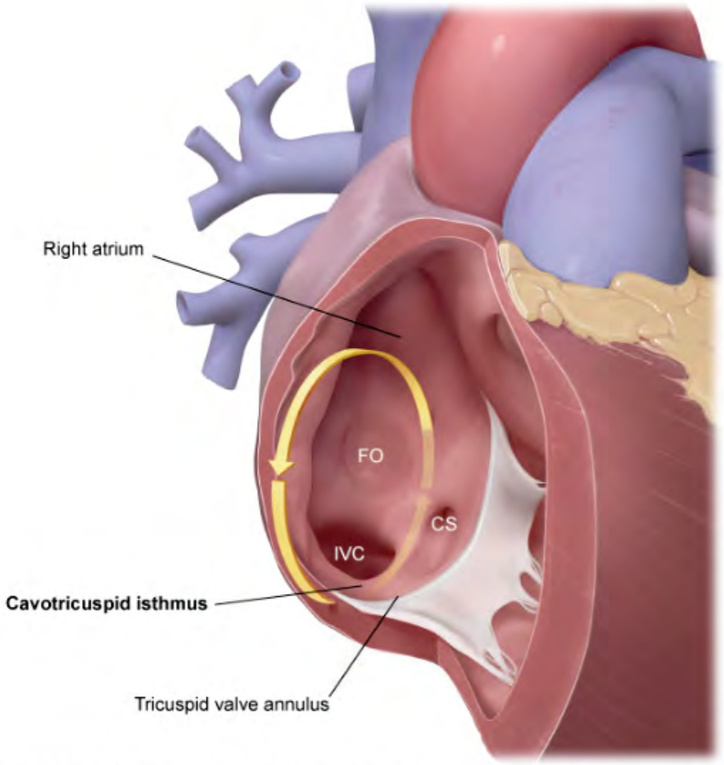
Reentrant circuit in atrial flutter



CS = coronary sinus; **FO** = fossa ovalis; **IVC** = inferior vena cava.
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Atrial flutter with consistent 2:1 conduction

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Physiology
Subject

Cardiovascular System
System

Atrial fibrillation
Topic

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A 44-year-old man with progressive dyspnea is diagnosed with dilated cardiomyopathy. Despite optimal medical therapy, he continues to have symptoms and disease progression is noted. He undergoes cardiac transplantation after a suitable donor becomes available. Permission is obtained from the patient to study his diseased heart for intracellular calcium regulation. Microelectrodes placed into cardiac muscle cells detect a rapid decrease in cytoplasmic calcium level immediately preceding relaxation. Which of the following proteins is most likely responsible for the observed change in electrolyte levels?

- A. Calmodulin
- B. Na^{+}/Ca^{2+} exchanger
- C. Ryanodine receptors
- D. Troponin C
- E. Voltage-dependent calcium channels

Submit

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A 44-year-old man with progressive dyspnea is diagnosed with dilated cardiomyopathy. Despite optimal medical therapy, he continues to have symptoms and disease progression is noted. He undergoes cardiac transplantation after a suitable donor becomes available. Permission is obtained from the patient to study his diseased heart for intracellular calcium regulation. Microelectrodes placed into cardiac muscle cells detect a rapid decrease in cytoplasmic calcium level immediately preceding relaxation. Which of the following proteins is most likely responsible for the observed change in electrolyte levels?

- A. Calmodulin (11%)
- ✓ B. Na^+/Ca^{2+} exchanger (45%)
- C. Ryanodine receptors (21%)
- D. Troponin C (5%)
- E. Voltage-dependent calcium channels (15%)

Correct

45% Answered correctly

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Explanation

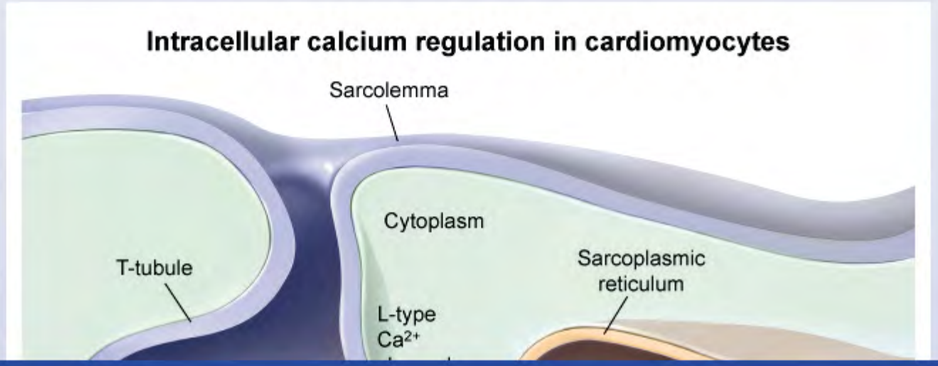

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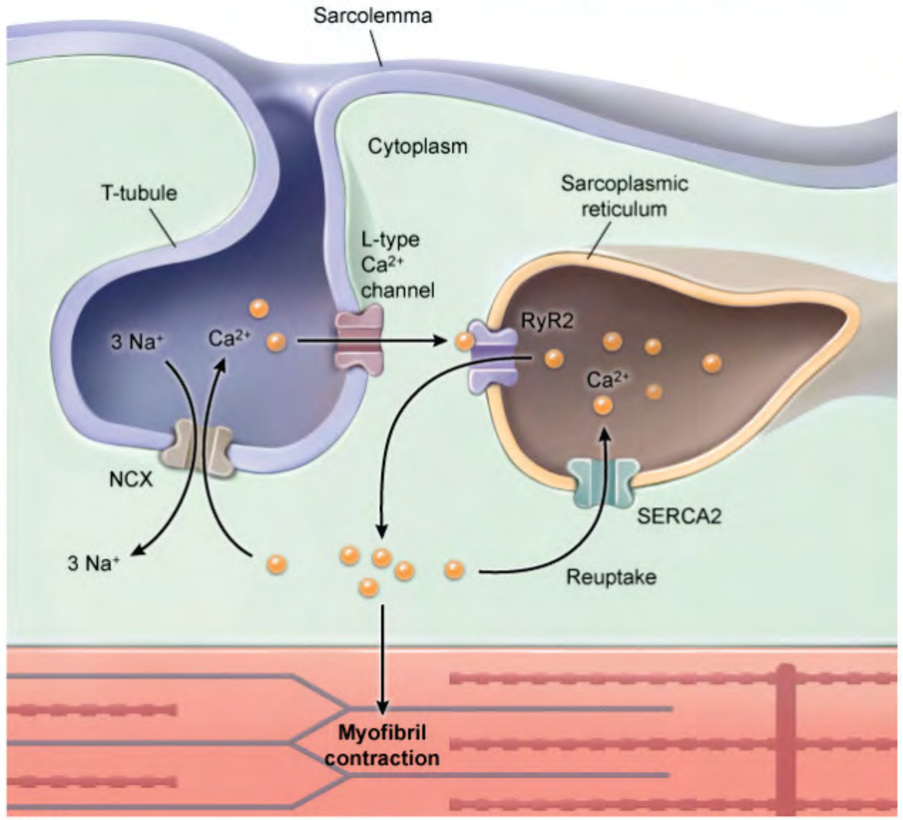
Exhibit Display

Intracellular calcium regulation in cardiomyocytes



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Intracellular calcium regulation plays an important role in **excitation-contraction coupling**. Voltage-dependent calcium channels (L-type) are activated during phase 2 of cardiac action potential (depolarization) and permit influx of calcium into the cardiac myocytes **(Choice E)**. This initial calcium influx is sensed by the ryanodine receptors in the sarcoplasmic reticulum, which triggers further release of Ca^{2+} (calcium-induced calcium release) into the cytoplasm, thereby increasing intracellular calcium concentration 100-fold **(Choice C)**. The calcium released from the sarcoplasmic reticulum diffuses through the myofilament network and binds to troponin C **(Choice D)**. Tropomyosin is then moved out of the way so that actin and myosin can interact, leading to muscle contraction.

The final stage of **excitation**-contraction coupling is **myocyte relaxation**, which occurs subsequent to **calcium efflux** from the cytoplasm. Intracellular calcium is removed primarily via an **Na^+/Ca^{2+} exchange pump** (NCX) and sarcoplasmic reticulum **Ca^{2+}-ATPase pump** (SERCA). NCX uses the large extracellular Na^+ concentration gradient to help pump Ca^{2+} out of the cell and, in the process, removes one intracellular Ca^{2+} in exchange for 3 extracellular Na^+ ions. In contrast, SERCA is a Ca^{2+}-ATPase pump that actively transfers Ca^{2+} from the cytosol to the lumen of sarcoplasmic reticulum at the expense of ATP hydrolysis.

(Choice A) Calmodulin (**cal**cium **modul**ated protein) is a calcium-binding messenger protein that is present in all cells and mediates many of the regulatory effects of Ca^{2+}. Calmodulin is important for excitation-contraction coupling in smooth muscle cells, which lack troponin, unlike cardiac and skeletal muscles. In cardiac muscle, calmodulin is not directly involved in excitation-contraction coupling; rather, it helps regulate intracellular calcium activity and transcription factor signaling.

Educational objective:

Calcium efflux from cardiac cells prior to relaxation is primarily mediated via an Na^+/Ca^{2+} exchange pump and sarcoplasmic reticulum Ca^{2+}-ATPase pump.

References

- The therapeutic potential of new insights into myocardial excitation-contraction coupling.

Physiology Cardiovascular System Muscle structure & physiology

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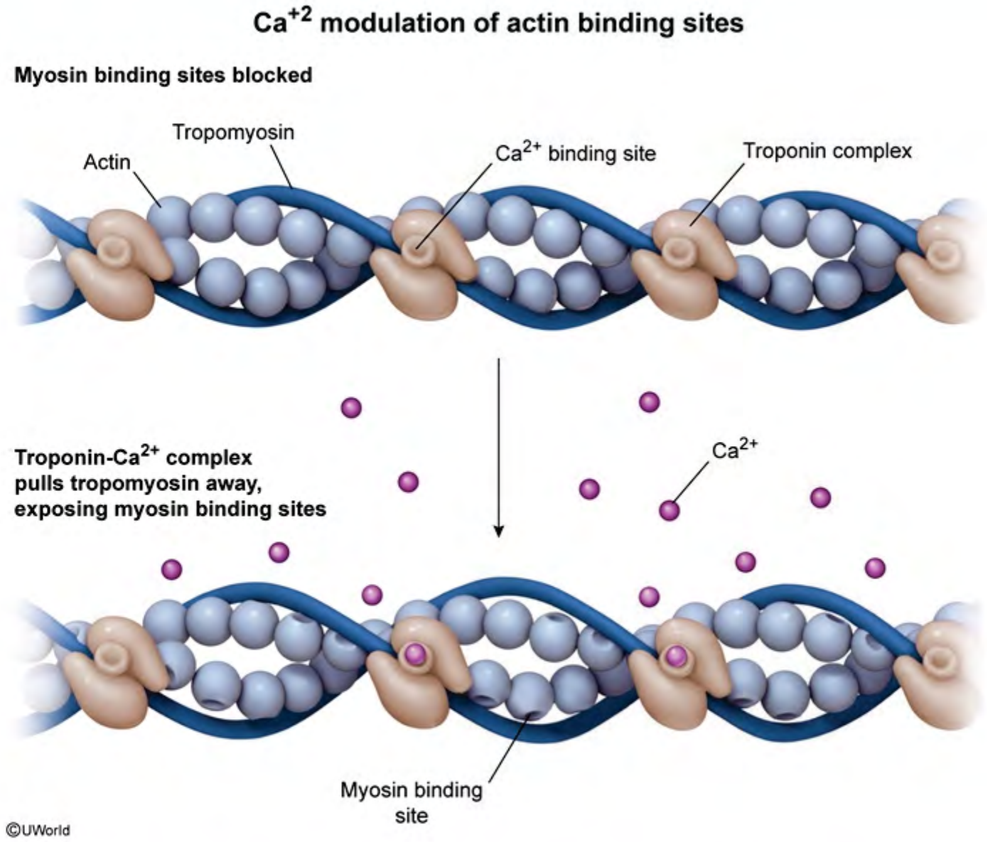

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Physiology Cardiovascular System Muscle structure & physiology

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A 63-year-old man comes to the emergency department due to increasing dyspnea. He was unable to mow his lawn the previous day due to shortness of breath. The patient has a history of hypertension and a 20-pack-year smoking history. He is retired and traveled to Hawaii 2 months ago. Blood pressure is 160/86 mm Hg, pulse is 94/min, and respirations are 24/min; oxygen saturation is 94% on room air. Physical examination reveals bilateral lung crackles and occasional wheezes. Laboratory testing reveals elevated brain natriuretic peptide levels. Which of the following is the primary trigger for the release of this substance in this patient?

- A. Activation of alveolar juxtacapillary receptors
- B. Decrease in renal perfusion pressure
- C. Decrease in vena cava blood return
- D. Focal myocardial necrosis
- E. Increase in ventricular wall stress
- F. Stretching of aortic baroreceptors

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- A. Activation of alveolar juxtacapillary receptors (1%)
- B. Decrease in renal perfusion pressure (3%)
- ✗ C. Decrease in vena cava blood return (2%)
- D. Focal myocardial necrosis (0%)
- ✓ E. Increase in ventricular wall stress (75%)
- F. Stretching of aortic baroreceptors (16%)

Incorrect
Correct answer
E

75%
Answered correctly

03 secs
Time Spent

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Explanation

In addition to their contractile and electrical conduction properties, **cardiomyocytes** possess endocrine capabilities to help regulate blood volume. Volume overload leads to stretching and **increased wall stress** of the ventricular walls. In response, the ventricular myocardium releases **brain natriuretic peptide** (BNP); similarly, the atrial myocardium releases atrial natriuretic peptide (ANP) in response to atrial stretching. These natriuretic peptides stimulate both venous and arterial **vasodilation** to decrease cardiac preload and afterload and reduce strain on the myocardium. In addition, ANP and BNP stimulate salt and water excretion by the kidneys to facilitate **diuresis**.

Clinically, BNP has diagnostic utility in patients with shortness of breath of unknown cause. For patients in whom

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Clinically, BNP has diagnostic utility in patients with shortness of breath of unknown cause. For patients in whom the serum BNP level is not elevated, shortness of breath is unlikely to be due to heart failure.

(Choice A) Alveolar juxtacapillary receptors increase respiratory rate in response to pathologic alveolar processes (eg, pulmonary edema, pneumonia).

(Choice B) Decreased renal perfusion pressure triggers the juxtaglomerular apparatus to release renin; activation of the renin-angiotensin-aldosterone system stimulates vasoconstriction and fluid retention, opposing the effects of natriuretic peptides.

(Choice C) Decrease in vena cava blood return reduces preload and leads to a consequent reduction in cardiac output. The reduced cardiac output is sensed by carotid baroreceptors, and an increase in sympathetic drive is triggered to stimulate vasoconstriction and increased heart rate and contractility.

(Choice D) Focal myocardial necrosis causes release of troponin into the bloodstream, which acts as a sensitive marker of myocardial damage. BNP levels often increase during myocardial infarction; however, this increase is likely secondary to ventricular dysfunction leading to volume overload and myocardial stretch.

(Choice F) Elevated blood pressure causes stretching of aortic baroreceptors, which triggers a reduction in sympathetic drive to alleviate vasoconstriction and reduce myocardial contractility.

Educational objective:
The ventricular myocardium secretes brain natriuretic peptide (BNP) in response to the ventricular stretch and strain that typically occurs with volume overload. BNP, along with atrial natriuretic peptide secreted by the atrial myocardium, stimulates vasodilation and salt and water excretion to help relieve volume overload.

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A 30-year-old Asian man comes to the clinic due to painful, ulcerated fingers. His symptoms began with pain and discoloration of the fingers several weeks ago, which progressed to ulceration at the fingertips. He also reports pain in the hands that occurs with activity and is relieved by rest. Medical history is unremarkable. The patient has smoked 2 packs of cigarettes a day for the last 5 years. He occasionally drinks alcohol but does not use illicit drugs. Vital signs are within normal limits. No heart murmurs are heard and the lungs are clear. Examination of the hands is shown in the exhibit. Radial pulses are diminished. There is erythema and tenderness along a superficial vein of the left forearm. Hemoglobin A1c and a fasting lipid panel are within normal limits. Which of the following pathologic findings is most likely to be seen within the affected vessels?

- A. Granulomatous inflammation of the media
- B. Lipid-filled intimal plaques
- C. Onion skin–appearing, concentric thickening of the arteriolar wall
- D. Segmental vasculitis extending into contiguous veins and nerves
- E. Transmural inflammation of the arterial wall with fibrinoid necrosis

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- A. Granulomatous inflammation of the media (8%)
- B. Lipid-filled intimal plaques (1%)
- C. Onion skin–appearing, concentric thickening of the arteriolar wall (4%)
- ✓ D. Segmental vasculitis extending into contiguous veins and nerves (63%)
- E. Transmural inflammation of the arterial wall with fibrinoid necrosis (22%)

Correct

63% Answered correctly

30 secs Time Spent

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Explanation

Thromboangiitis obliterans (Buerger disease)	
Etiology	• Segmental small & medium-sized vessel vasculitis • Likely triggered by tobacco smoke
Clinical features	• Age <45, predominantly men • Digital ischemic ulcerations, limb claudication

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Explanation

Thromboangiitis obliterans (Buerger disease)	
Etiology	• Segmental small & medium-sized vessel vasculitis • Likely triggered by tobacco smoke
Clinical features	• Age <45, predominantly men • Digital ischemic ulcerations, limb claudication • Raynaud phenomenon, superficial thrombophlebitis
Histopathology	• Inflammatory intraluminal thrombi with vessel wall sparing • Extension into contiguous veins & nerves

This patient's presentation is consistent with **thromboangiitis obliterans** (Buerger disease), a segmental, inflammatory vasculitis that affects the small- and medium-sized arteries and veins of the distal extremities. It typically affects **young**, **heavy smokers** (age <45) and is more common in men. Toe and/or finger ischemia and **ischemic ulcerations** are the most common manifestation, and patients with involvement of proximal arteries can develop claudication affecting the lower legs, feet, or hands. **Raynaud phenomenon** and superficial thrombophlebitis (erythema, tenderness along veins) may also occur as early manifestations of the disease.

In thromboangiitis obliterans, chronic exposure to tobacco products is thought to cause direct endothelial injury or trigger a delayed-type hypersensitivity reaction against the endothelium. Histopathology shows highly cellular, inflammatory **intraluminal thrombi** (ie, containing neutrophils, multinucleated giant cells) in the arteries and veins with **sparing of the vessel wall** and internal elastic lamina. This segmental, thrombosing vasculitis often **extends contiguously** into veins and nerves, a feature rarely seen in other types of vasculitis.

(Choice A) Granulomatous inflammation of the media occurs in the large vessel vasculitides Giant cell arteritis (temporal arteritis) and Takayasu arteritis. Giant cell arteritis typically affects patients age >50 and presents with unilateral headache, jaw claudication, and transient vision loss. Takayasu arteritis usually affects young women of Asian descent and presents with diminished pulses often accompanied by angina and/or carotid artery pain.

(Choice B) Lipid-filled intimal plaques that bulge into the arterial lumen are seen in patients with atherosclerosis

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(Choice B) Lipid-filled intimal plaques that bulge into the arterial lumen are seen in patients with atherosclerosis.

(Choice C) Hyperplastic arteriolosclerosis is characterized by thickening of the arteriolar wall due to the concentric proliferation of smooth muscle cells and reduplicated basement membranes, giving the arterioles an onion-skin appearance. These changes occur as an adaptive response of arterioles to severe (malignant) hypertension.

(Choice E) Transmural inflammation of the arterial wall with fibrinoid necrosis can be seen in several types of systemic vasculitis, including polyarteritis nodosa (PAN). PAN affects small and medium-sized arteries and can involve numerous organs (eg, kidney, gastrointestinal system, skin). Patients commonly present with fever, hypertension, abdominal pain, and/or cutaneous lesions (eg, nodules, livedo reticularis).

Educational objective:
Thromboangiitis obliterans (Buerger disease) is a segmental, inflammatory vasculitis that affects the small- and medium-sized arteries and veins of the distal extremities with inflammatory, intraluminal thrombi and sparing of the vessel wall. It is usually seen in young, heavy smokers, and can present with digital ischemia and ulceration, extremity claudication, Raynaud phenomenon, and superficial thrombophlebitis.

Pathology	Cardiovascular System	Peripheral vascular disease
Subject	System	Topic

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Etiology	• Segmental small & medium-sized vessel vasculitis Likely triggered by tobacco smoke

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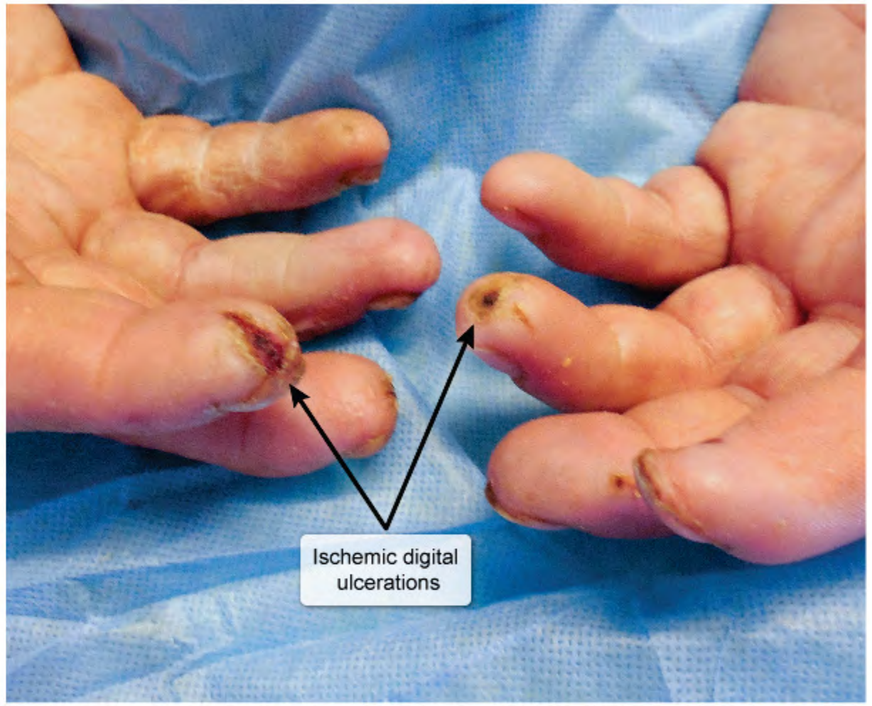
Thromboangiitis obliterans (Buerger disease)



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A 35-year-old previously healthy man is brought to the emergency department after being involved in a motor vehicle collision. He was an unrestrained driver and hit his chest against the steering wheel during the collision. When the paramedics reached him, he reported chest discomfort but was hemodynamically stable. Shortly after arrival at the emergency department, the patient experiences worsening respiratory distress and becomes hypotensive. On physical examination, the patient appears anxious and is tachycardic and tachypneic. The trachea is midline; the jugular veins are distended. The anterior chest wall is bruised and tender to palpation. Vesicular breath sounds are present bilaterally. The abdomen is soft and nontender. Which of the following is the most likely cause of this patient's clinical deterioration?

- A. Aortic rupture
- B. Cardiac tamponade
- C. Diaphragmatic rupture
- D. Hemothorax
- E. Tension pneumothorax

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- ✗ A. Aortic rupture (11%)
- ✓ B. Cardiac tamponade (74%)
- C. Diaphragmatic rupture (0%)
- D. Hemothorax (10%)
- E. Tension pneumothorax (3%)

Incorrect
Correct answer
B

74%
Answered correctly

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Explanation

Cardiac tamponade	
Etiology	• Blood in pericardial space (eg, LV rupture, cardiac surgery) • Pericardial effusion (eg, malignancy, infection, uremia)
Clinical signs	• Beck triad: hypotension, JVD, ↓ heart sounds • Pulsus paradoxus: SBP ↓10 mm Hg during inspiration

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Explanation

Cardiac tamponade	
Etiology	• Blood in pericardial space (eg, LV rupture, cardiac surgery) • Pericardial effusion (eg, malignancy, infection, uremia)
Clinical signs	• Beck triad: hypotension, JVD, ↓ heart sounds • Pulsus paradoxus: SBP ↓10 mm Hg during inspiration
Diagnosis	• ECG: low-voltage QRS, electrical alternans • Chest x-ray: enlarged cardiac silhouette*, clear lungs • Echocardiogram: right atrial & ventricular collapse, IVC plethora
*Subacute but not acute tamponade. **IVC** = inferior vena cava; **JVD** = jugular venous distension; **LV** = left ventricular; **SBP** = systolic blood pressure.	

This patient experienced significant **blunt chest trauma** when his chest struck the steering wheel, evidenced by anterior chest wall bruising and tenderness on examination. Such trauma can lead to several life-threatening cardiopulmonary injuries; this patient with progressive hypotension, tachycardia, and jugular venous distension most likely has **acute cardiac tamponade**.

Blunt cardiac injury can cause **rapid accumulation of blood** within the pericardium. Because the pericardium is relatively noncompliant and does not have time to adapt, even a small amount of pericardial fluid (eg, 100-200 mL) can dramatically **increase intrapericardial pressure** and lead to tamponade. The increased pericardial pressure compresses the relatively low-pressure, right-sided heart chambers and restricts diastolic filling of the right side of the heart. This elevates right atrial pressure, which is transmitted backward to the vena cava, the central venous system, and the **jugular veins** (causing **distension**).

With decreased right-sided filling, less blood is pumped through the pulmonary circulation to the left atrium and left ventricle, resulting in **decreased cardiac output**. This results in systemic **hypotension**, despite compensatory tachycardia. Impaired peripheral perfusion leads to unmet oxygen demand in the tissues with resulting dyspnea,

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relatively noncompliant and does not have time to adapt, even a small amount of pericardial fluid (eg, 100-200 mL) can dramatically **increase intrapericardial pressure** and lead to tamponade. The increased pericardial pressure compresses the relatively low-pressure, right-sided heart chambers and restricts diastolic filling of the right side of the heart. This elevates right atrial pressure, which is transmitted backward to the vena cava, the central venous system, and the **jugular veins** (causing **distension**).

With decreased right-sided filling, less blood is pumped through the pulmonary circulation to the left atrium and left ventricle, resulting in **decreased cardiac output**. This results in systemic **hypotension**, despite compensatory tachycardia. Impaired peripheral perfusion leads to unmet oxygen demand in the tissues with resulting dyspnea, increased respiratory drive, and tachypnea.

(Choice A) Blunt deceleration during chest trauma can cause tearing of the aorta (ie, aortic rupture). However, aortic rupture usually causes massive internal hemorrhage with rapidly developing hypovolemic shock (eg, flat jugular veins). Most affected patients do not survive transport to the hospital.

(Choice C) Diaphragmatic rupture can occur with blunt trauma, typically from a sudden increase in intraabdominal pressure. It may result in intrathoracic herniation of abdominal organs and respiratory symptoms (eg, dyspnea, tachypnea), but it often causes abdominal tenderness and does not typically cause significant bleeding or hypotension.

(Choice D) Blunt chest trauma could cause a massive hemothorax with hypotension and tachycardia (ie, hypovolemic shock) as well as tachypnea due to the mass effect on the lung. However, flat jugular veins and absent basilar breath sounds are expected.

(Choice E) Tension pneumothorax can result from blunt chest trauma that injures the visceral pleura or tracheobronchial tree. It can cause tachypnea and obstructive shock (eg, hypotension, tachycardia, jugular venous distension); however, unilaterally absent breath sounds are expected, and tracheal deviation is often present.

Educational objective:
Blunt chest trauma can lead to several life-threatening cardiopulmonary injuries. Progressive hypotension with tachycardia, jugular venous distension, and bilateral vesicular breath sounds suggests acute cardiac tamponade.

Pathology Cardiovascular System Cardiac tamponade

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A 68-year-old man comes to the office due to increased abdominal distension. The patient was discharged from the hospital last week after an acute pulmonary embolism. He also has a history of emphysema caused by extensive tobacco use. The patient has no other medical conditions and does not use alcohol or illicit drugs. Temperature is 37.6 C (99.7 F), blood pressure is 112/78 mm Hg, pulse is 85/min, and respirations are 18/min. BMI is 27 kg/m^2. Examination shows increased jugular venous pressure and lower extremity edema. The abdomen is distended, and a fluid wave is present. This patient's ascites most likely developed due to which of the following changes in portal capillary exchange parameters?

	Portal capillary oncotic pressure	Portal capillary hydrostatic pressure	Portal capillary permeability
A.	↓	↑	↑
B.	↓	↑	Normal
C.	↑	↓	Normal
D.	Normal	↑	↑
E.	Normal	↑	Normal
F.	Normal	Normal	↑

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	Portal capillary oncotic pressure	Portal capillary hydrostatic pressure	Portal capillary permeability	
✗ A.	↓	↑	↑	(23%)
B.	↓	↑	Normal	(24%)
C.	↑	↓	Normal	(1%)
D.	Normal	↑	↑	(12%)
✓ E.	Normal	↑	Normal	(36%)
F.	Normal	Normal	↑	(1%)

Incorrect
Correct answer
E

36%
Answered correctly

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Explanation

Ascites can develop in a variety of disease states (eg, cirrhosis, peritoneal metastasis). In this case, the presence of jugular venous distension and lower extremity edema shortly after pulmonary embolism (PE) are highly

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Explanation

Ascites can develop in a variety of disease states (eg, cirrhosis, peritoneal metastasis). In this case, the presence of jugular venous distension and lower extremity edema shortly after pulmonary embolism (PE) are highly suggestive of **right-sided heart failure (RHF)** (ie, cor pulmonale) as the cause of this patient's ascites.

Patients with chronic respiratory disorders (eg, **emphysema**, obstructive sleep apnea) often have **baseline pulmonary hypertension** due to hypoxia-induced pulmonary vasoconstriction and associated vascular remodelling, which increases the pressure that the right ventricle (RV) must work against to maintain forward flow. These patients are **predisposed to RHF**, which can develop subacutely from progressive pulmonary hypertension or **acutely** in the setting of a PE (due to abrupt obstruction of an already compromised pulmonary capillary bed).

Ascites due to RHF is associated with the following findings:

- **Increased hydrostatic pressure:** RV dysfunction results in the backup of blood within the venous circulation, leading to increased central venous pressure, which is transmitted to the portal capillaries (ie, hepatic sinusoids).

- **Normal oncotic pressure:** Although passive congestion from prolonged RHF eventually causes sinusoidal injury, leading to cirrhosis and hepatic synthetic dysfunction (ie hypoalbuminemia), this alteration occurs after a prolonged period (weeks). Albumin has a half-life of approximately 21 days; therefore, early in the disease process, oncotic pressure remains normal **(Choice B)**.

- **Normal capillary permeability:** Hepatic congestion is characterized by sinusoidal engorgement, but sinusoidal permeability, which is already high at baseline due to the discontinuous sinusoidal endothelium, remains essentially unchanged.

(Choices A, D, and F) Portal capillary permeability may be increased in the setting of hepatic malignancies due to disruption of portal capillary integrity; however, it is unaffected by RHF. Portal oncotic pressure remains normal early in the disease process.

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(Choices A, D, and F) Portal capillary permeability may be increased in the setting of hepatic malignancies due to disruption of portal capillary integrity; however, it is unaffected by RHF. Portal oncotic pressure remains normal early in the disease process.

(Choice C) Both increased portal capillary oncotic pressure and decreased portal capillary hydrostatic pressure would help resolve ascites rather than contribute to its formation.

Educational objective:
Right-sided heart failure promotes the formation of ascites due to increased central venous pressure, which is transmitted to the hepatic sinusoids, leading to increased capillary hydrostatic pressure. Capillary permeability is unaffected. Although chronic passive congestion may eventually result in hepatic synthetic dysfunction (hypoalbuminemia) with low oncotic pressures, the oncotic pressure will remain normal for several weeks at least.

Pathophysiology	Cardiovascular System	Ascites
Subject	System	Topic

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A 58-year-old man with dyspnea and chronic exertional angina is evaluated for coronary revascularization. He has a history of hypertension, type 2 diabetes mellitus, and hypercholesterolemia. His blood pressure is 130/80 mm Hg and pulse is 72/min and regular. Cardiopulmonary examination is normal with the exception of a fourth heart sound. Echocardiogram reveals hypokinesia of the anterior wall of the left ventricle and a left ventricular ejection fraction (LVEF) of 35%. The patient undergoes coronary artery bypass grafting. Repeat echocardiogram 10 days after the surgery shows that hypokinesia is no longer evident and LVEF has increased to 50%. Which of the following best explains the changes in the cardiac contractility and wall motion seen in this patient?

- A. Hibernating myocardium
- B. Ischemic preconditioning
- C. Post-infarction myocardial scarring
- D. Reperfusion injury
- E. Ventricular remodeling

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- ✓ A. Hibernating myocardium (42%)
- B. Ischemic preconditioning (19%)
- C. Post-infarction myocardial scarring (3%)
- D. Reperfusion injury (5%)
- E. Ventricular remodeling (28%)

Correct | 42% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

Myocardial hibernation refers to a state of **chronic myocardial ischemia** in which both myocardial metabolism and function are reduced to match a concomitant reduction in coronary blood flow (due to moderate/severe flow-limiting stenosis). This new equilibrium prevents myocardial necrosis. Chronically hibernating myocardium demonstrates decreased expression and disorganization of contractile and cytoskeletal proteins, altered adrenergic control, and reduced calcium responsiveness. These changes lead to **decreased contractility** and left ventricular (LV) systolic dysfunction. However, coronary revascularization and subsequent restoration of blood flow to hibernating myocardium improves contractility and LV function.

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Myocardial hibernation refers to a state of **chronic myocardial ischemia** in which both myocardial metabolism and function are reduced to match a concomitant reduction in coronary blood flow (due to moderate/severe flow-limiting stenosis). This new equilibrium prevents myocardial necrosis. Chronically hibernating myocardium demonstrates decreased expression and disorganization of contractile and cytoskeletal proteins, altered adrenergic control, and reduced calcium responsiveness. These changes lead to **decreased contractility** and left ventricular (LV) systolic dysfunction. However, coronary revascularization and subsequent restoration of blood flow to hibernating myocardium improves contractility and LV function.

(Choice B) Ischemic preconditioning is a phenomenon in which brief repetitive episodes of myocardial ischemia, followed by reperfusion, protect the myocardium from subsequent prolonged episodes of ischemia. In clinical terms, repetitive episodes of angina prior to myocardial infarction can delay cell death after complete coronary occlusion and therefore provide greater time for myocardial salvage with coronary revascularization.

(Choice C) Post-infarction myocardial scarring refers to the replacement of myocardium with collagen and fibrous tissue. This leads to complete loss of myocardial contractile function, and cardiac contractility would not be expected to improve with coronary revascularization.

(Choice D) Reperfusion injury refers to further myocardial cell damage due to restoration of blood flow after a sudden ischemic event. The clinical manifestations of reperfusion injury include arrhythmias, microvascular dysfunction with myocardial stunning, and myocyte injury and death.

(Choice E) Ventricular remodeling refers to a change in the structure (cardiac mass or dimensions) or function of the heart in response to cardiac injury or hemodynamic changes (pressure/volume overload). Remodeling can occur with long-term myocardial hibernation, but it would take weeks to months (not days) to recover following reperfusion therapy.

Educational objective:
Hibernating myocardium refers to the presence of left ventricular systolic dysfunction due to reduced coronary blood flow at rest that is partially or completely reversible by coronary revascularization.

References

- The hibernating myocardium in ischaemia and congestive heart failure

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A 44-year-old man comes to the office due to increasing shortness of breath. While climbing the stairs to his second-floor apartment, the patient must now stop halfway to catch his breath. He has a history of nonischemic cardiomyopathy and chronic heart failure with reduced ejection fraction. After evaluation, treatment with a new medication is begun that improves his symptoms by increasing urine output and decreasing peripheral vascular resistance. The medication works by inhibiting a metalloprotease to prolong the action of endogenous polypeptides. These polypeptides are most likely secreted by which of the following cell types?

- A. Adrenal zona glomerulosa cells
- B. Cardiomyocytes
- C. Hepatic stellate cells
- D. Myocardial Purkinje fibers
- E. Renal juxtaglomerular cells
- F. Vascular endothelial cells

Submit

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- ✗ A. Adrenal zona glomerulosa cells (5%)
- ✓ B. Cardiomyocytes (56%)
- C. Hepatic stellate cells (3%)
- D. Myocardial Purkinje fibers (1%)
- E. Renal juxtaglomerular cells (13%)
- F. Vascular endothelial cells (19%)

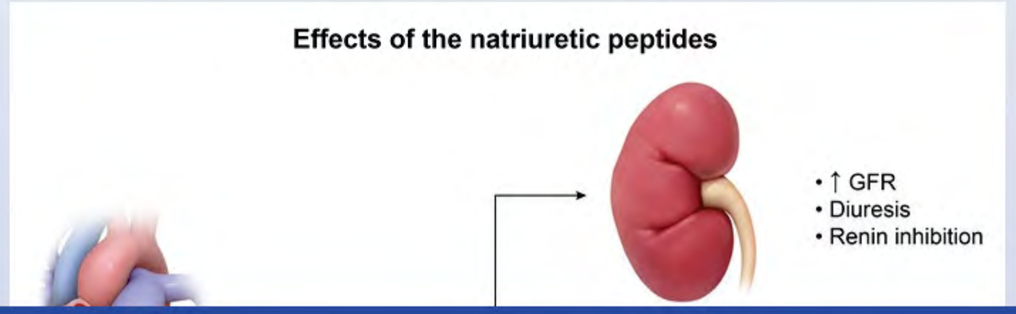
Incorrect
Correct answer
B

56%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation



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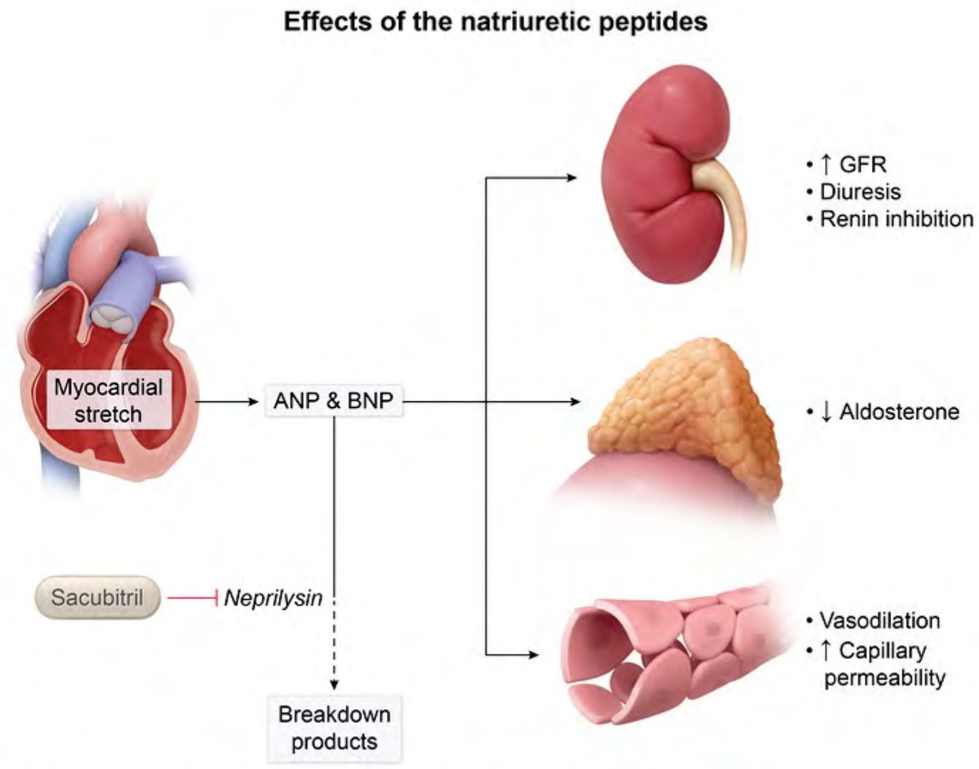

ANP = atrial natriuretic peptide; **BNP** = brain natriuretic peptide; **GFR** = glomerular filtration rate.
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ANP = atrial natriuretic peptide; **BNP** = brain natriuretic peptide; **GFR** = glomerular filtration rate.
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In response to myocardial stretching due to volume overload, atrial and ventricular **cardiomyocytes** secrete **atrial natriuretic peptide** (ANP) and **brain natriuretic peptide** (BNP). These peptides are subsequently broken down and inactivated by the metalloprotease **neprilysin**. This patient with **chronic heart failure** was most likely given a **neprilysin inhibitor** (eg, sacubitril), allowing for enhanced activity of ANP and BNP.

Once secreted, ANP and BNP bind to natriuretic peptide receptors to activate guanylate cyclase and form cyclic GMP, which stimulates **diuresis** and peripheral **vasodilation** to help alleviate volume overload. Several organs and tissues are affected as follows:

- **Kidneys**: The afferent arterioles are dilated and the efferent arterioles are constricted, raising the glomerular filtration rate and increasing urinary excretion of sodium and water. Renin secretion from the juxtaglomerular cells is inhibited **(Choice E)**, counteracting renin-angiotensin-aldosterone-system (RAAS) activity.
- **Adrenal glands**: Secretion of aldosterone from the zona glomerulosa cells is inhibited **(Choice A)**, further counteracting RAAS activity and bolstering the increase in sodium and water excretion.
- **Blood vessels**: Arteriolar and venular smooth muscle relaxes, producing vasodilation. There is also increased capillary permeability, leading to fluid extravasation into the interstitium and a decrease in circulating blood volume.

(Choice C) Hepatic stellate cells (perisinusoidal cells of Ito) may help regulate hepatic blood flow via release of local mediators; however, these cells do not play a significant role in heart failure.

(Choice D) Myocardial Purkinje fibers facilitate the organized transport of electrical impulses from the sinoatrial node through the atria, atrioventricular node, and ventricles; however, these fibers are not known to have endocrine function.

(Choice F) Vascular endothelial cells regulate systemic blood flow via release of vasodilators, such as nitric oxide and prostacyclin, and vasoconstrictors, such as endothelin and thromboxane A2. However, these local mediators are not a significant target of therapy for chronic heart failure.

Educational objective:
Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are secreted by atrial and ventricular

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Once secreted, ANP and BNP bind to natriuretic peptide receptors to activate guanylate cyclase and form cyclic GMP, which stimulates **diuresis** and peripheral **vasodilation** to help alleviate volume overload. Several organs and tissues are affected as follows:

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Educational objective:
Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are secreted by atrial and ventricular cardiomyocytes in response to myocardial stretching induced by hypervolemia. These natriuretic peptides inhibit the renin-angiotensin-aldosterone system and stimulate peripheral vasodilation and increased urinary excretion of sodium and water. Neprilysin inhibitors (eg, sacubitril) prevent the degradation of ANP and BNP, enhancing their beneficial effects in heart failure.

References

- The natriuretic peptide system in the pathophysiology of heart failure: from molecular basis to treatment.

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A 32-year-old man dies suddenly in his sleep. He had been experiencing easy fatigability for the past 6 months and mentioned that he was feeling tired "'all the time"' and needed to take naps during the day. He did not drink alcohol or use illicit drugs. He smoked a pack of cigarettes per day for 10 years. His father died from a "heart problem" at age 40. On autopsy, the heart appears grossly enlarged. A layered mural thrombus is seen in the left ventricular apex. Coronary atherosclerosis is present, with 20% narrowing of the mid-left anterior descending artery and 25% narrowing of the left circumflex artery. Which of the following is the most likely cause of death of this patient?

- A. Antiphospholipid antibody syndrome
- B. Dilated cardiomyopathy
- C. Hypertrophic cardiomyopathy
- D. Ischemic heart disease
- E. Restrictive cardiomyopathy

Submit

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- ✗ A. Antiphospholipid antibody syndrome (8%)
- ✓ B. Dilated cardiomyopathy (44%)
- C. Hypertrophic cardiomyopathy (30%)
- D. Ischemic heart disease (12%)
- E. Restrictive cardiomyopathy (3%)

Incorrect
Correct answer
B

44%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation

This patient most likely died due to **dilated cardiomyopathy** (DCM). A primary insult to the myocardium leads to impaired myocardial contractile function with dilation of one or both ventricles. The structural changes to the ventricular myocardium places these patients at risk of **sudden cardiac death** from ventricular arrhythmia (ie, ventricular tachycardia, ventricular fibrillation). In addition, the global hypokinesis of the left ventricle leads to stagnation of blood flow and the possible development of LV mural thrombus and subsequent systemic embolization.

Causes of DCM include infection (eg, viral myocarditis, Chagas disease), chemical toxicity (eg, alcohol), and

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Causes of DCM include infection (eg, viral myocarditis, Chagas disease), chemical toxicity (eg, alcohol), and inherited disease. The premature death of this patient's father from a heart problem suggests **familial DCM**, which is typically transmitted in an **autosomal dominant** inheritance pattern. Truncating mutations affecting the ***TTN* gene**, which encodes for the cardiac sarcomere protein **titin**, are the most common cause of familial DCM.

(Choice A) Antiphospholipid antibody syndrome is an autoimmune disorder characterized by venous and/or arterial thrombosis. Cardiac manifestations can include valvular thickening, nonbacterial vegetations, and increased risk of coronary atherosclerosis; however, LV cavity enlargement and mural thrombus are not typical.

(Choice C) Hypertrophic cardiomyopathy is an autosomal dominant genetic disorder caused by mutation in genes encoding for one of several cardiac sarcomere proteins (eg, beta-myosin heavy chain, myosin-binding protein C). It is a common cause of sudden cardiac death in young individuals, usually during exertion. Autopsy typically shows asymmetric septal hypertrophy of the left ventricle.

(Choice D) Ischemic heart disease can lead to myocardial dysfunction and LV enlargement that mimics DCM; however, this patient's coronary arteries had only minor atherosclerotic narrowing (ie, 20% and 25%) that would not have caused clinically significant myocardial ischemia.

(Choice E) Restrictive cardiomyopathy is typically the early cardiac manifestation of an infiltrative process (eg, amyloidosis, sarcoidosis) and is characterized by nondilated ventricles with enlargement of both atria.

Educational objective:
Dilated cardiomyopathy results from primary myocardial dysfunction leading to eccentric remodeling of the left ventricle. Patients can develop left ventricular mural thrombus and are at risk for sudden cardiac death due to ventricular arrhythmia. Familial dilated cardiomyopathy is typically inherited in an autosomal dominant pattern, and

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ventricular myocardium places these patients at risk of **sudden cardiac death** from ventricular arrhythmia (ie, ventricular tachycardia, ventricular fibrillation). In addition, the global hypokinesis of the left ventricle leads to stagnation of blood flow and the possible development of LV mural thrombus and subsequent systemic embolization.

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Educational objective:
Dilated cardiomyopathy results from primary myocardial dysfunction leading to eccentric remodeling of the left ventricle. Patients can develop left ventricular mural thrombus and are at risk for sudden cardiac death due to ventricular arrhythmia. Familial dilated cardiomyopathy is typically inherited in an autosomal dominant pattern, and most commonly results from truncating mutations of the *TTN* gene that codes for the sarcomere protein titin.

References

- Classification of the cardiomyopathies: a position statement from the European Society of Cardiology Working

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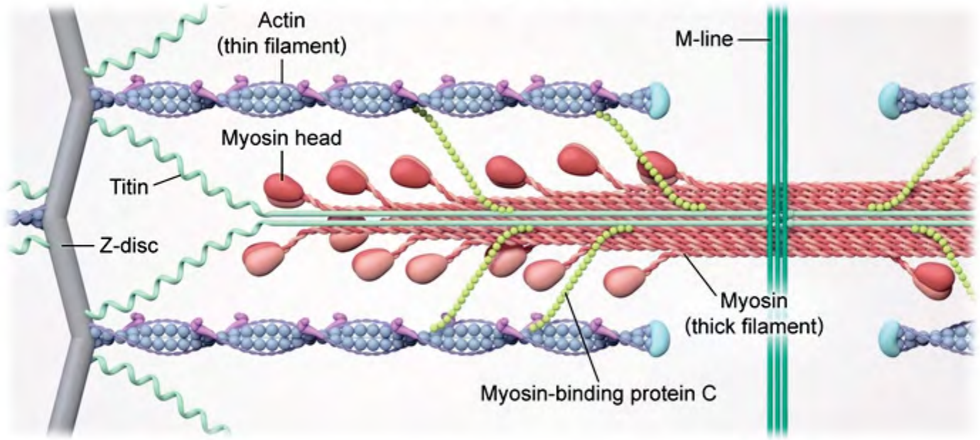
Cardiac sarcomere protein structure



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This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 71-year-old man comes to the office due to a 2-month history of progressive exertional dyspnea. The patient is unable to walk half a block without resting and can no longer climb stairs without stopping every few steps. He also has had difficulty sleeping due to repeatedly waking up at night feeling like he is suffocating. Physical examination shows bilateral lower extremity edema and distended neck veins. Cardiac auscultation findings over the apex are given below. This patient's auscultatory findings most likely reflect which of the following?

Play Media

- A. Aortic valve sclerosis
- B. Bulging of the interventricular septum
- C. Dynamic left ventricular outflow obstruction
- D. Elevated pulmonary arterial pressure
- E. Increased left ventricular end-systolic volume

Submit

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Play Media

- A. Aortic valve sclerosis (15%)
- B. Bulging of the interventricular septum (6%)
- C. Dynamic left ventricular outflow obstruction (10%)
- D. Elevated pulmonary arterial pressure (28%)
- ✓ E. Increased left ventricular end-systolic volume (38%)

Correct

38% Answered correctly

01 min, 47 secs Time Spent

2023 Version

Explanation

Extra (gallop) heart sounds		
	Features	Associated pathology*

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Show all

Extra (gallop) heart sounds		
	Features	**Associated pathology***
S3	• Heard just after S2 • Caused by reverberant sound as blood fills an enlarged LV cavity during passive diastolic filling	• Heart failure with reduced EF • High-output states (eg, thyrotoxicosis) • Mitral or aortic regurgitation
S4	• Heard just before S1 • Caused by blood striking a stiff LV wall during atrial contraction	• Concentric LV hypertrophy • Restrictive cardiomyopathy • Acute myocardial infarction
*S3 can be normal in children, young adults & during pregnancy. S4 can be normal in the elderly (eg, age >70). **EF** = ejection fraction; **LV** = left ventricular.		

Cardiac auscultation in this patient reveals a **low-frequency** sound occurring **just after S2**, known as an **S3**. Although an S3 can be normal in young individuals and pregnant patients, in those age >40 it is typically a sign of left ventricular (LV) **volume overload or failure** (causing exertional dyspnea and paroxysmal nocturnal dyspnea in this patient).

In early diastole, the ventricles relax and the atrioventricular valves open, allowing blood to rush in and fill the ventricles. An S3 is thought to represent reverberant sound created by blood entering an enlarged and overfilled ventricular cavity during **rapid passive filling**. As a result, S3 is typically heard in pathologic settings causing high ventricular filling pressures and volume overload, particularly aortic or mitral regurgitation and heart failure with reduced ejection fraction (eg, dilated cardiomyopathy).

(Choice A) Aortic valve sclerosis may produce a midsystolic ejection murmur but would not produce an extra heart sound. Progression to severe aortic stenosis may eventually produce an S4 due to concentric LV hypertrophy and stiffening of the ventricular wall

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(Choice B) Cardiac tamponade causes the restricted right ventricle to bulge into the left ventricle during inspiration. This phenomenon does not generate an auscultatory sound but does cause a >10 mm Hg decrease in systolic blood pressure during inspiration (pulsus paradoxus).

(Choice C) Hypertrophic cardiomyopathy can cause dynamic LV outflow obstruction, producing a crescendo-decrescendo ejection systolic murmur. The outflow obstruction can also cause systolic anterior motion of the mitral valve, resulting in mitral regurgitation (holosystolic murmur).

(Choice D) Elevated pulmonary arterial pressure (pulmonary hypertension) can present with exertional dyspnea and fatigue and eventual right ventricular failure (elevated jugular venous pressure, peripheral edema). However, auscultation reveals increased intensity of the pulmonic closure sound (P2), and affected patients typically lack nocturnal dyspnea due to intact LV function.

Educational objective:
An S3 is a low-frequency sound that occurs just after S2 and is typically associated with ventricular volume overload. It is commonly heard in patients with mitral or aortic regurgitation or heart failure with reduced ejection fraction (eg, dilated cardiomyopathy).

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A 19-year-old man comes to the office to establish medical care. He is transitioning care from his pediatrician. The patient states that he recently changed his glasses for myopia; he otherwise has no symptoms. He takes no medications. Vital signs are within normal limits. On physical examination, the patient is tall with long upper extremities and fingers. The face appears narrow with down-slanted palpebral fissures, flattened malar bones, and a small jaw. The lungs are clear on auscultation. A late-systolic murmur is present at the cardiac apex. The abdomen is soft and nontender with no organomegaly. Which of the following is the most likely cause of this patient's murmur?

- A. Aortic root dilation
- B. Aortic valve cusp fusion
- C. Endocardial fibrous deposition
- D. Myxomatous mitral degeneration
- E. Papillary muscle dysfunction

Submit

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- ✗ A. Aortic root dilation (25%)
- B. Aortic valve cusp fusion (6%)
- C. Endocardial fibrous deposition (2%)
- ✓ D. Myxomatous mitral degeneration (53%)
- E. Papillary muscle dysfunction (12%)

Incorrect
Correct answer
D

53%
Answered correctly

01 min, 16 secs
Time Spent

2023
Version

Explanation

This patient has clinical features of **Marfan syndrome**, an autosomal dominant disorder caused by **mutations** in the *FBN1* gene encoding **fibrillin-1**, a main component of extracellular matrix microfibrils. Fibrillin-1 provides support to elastic fibers and helps maintain connective tissue integrity. It also regulates extracellular matrix remodelling by binding to and sequestering **transforming growth factor-beta** (TGF-beta).

In Marfan syndrome, defective fibrillin-1 is unable to bind TGF-beta. The resulting **overexpression** of free, active TGF-beta leads to increased production of matrix metalloproteinases, which cleave elastic fibers and other components of the extracellular matrix, **reducing tissue integrity**. Within the mitral valve, this process results in

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In Marfan syndrome, defective fibrillin-1 is unable to bind TGF-beta. The resulting **overexpression** of free, active TGF-beta leads to increased production of matrix metalloproteinases, which cleave elastic fibers and other components of the extracellular matrix, **reducing tissue integrity**. Within the mitral valve, this process results in fragmentation of elastic fibers and decreased collagen density with pooling of glycosaminoglycans (**myxomatous mitral degeneration**).

The elongated, floppy mitral valve leaflets **prolapse** into the atrium, which causes a midsystolic click on auscultation and allows for mitral valve regurgitation. This regurgitant flow is characterized by a **mid-to-late systolic apical murmur**, as seen in this patient; severe regurgitation may produce a holosystolic murmur.

(Choice A) Although aortic root dilation is common with Marfan syndrome, the associated aortic regurgitation causes a decrescendo diastolic murmur.

(Choice B) Aortic valve cusp fusion (ie, bicuspid aortic valve) is commonly associated with Turner syndrome, not Marfan syndrome. Auscultatory findings are attributed to aortic stenosis (systolic crescendo-decrescendo murmur at the right upper sternal border) and/or aortic regurgitation (diastolic murmur along the left sternal border).

(Choice C) Endocardial fibrous deposition occurs with carcinoid heart disease. In contrast to this case, presentation includes right-sided heart failure (eg, edema, hepatomegaly), and common murmurs include tricuspid regurgitation (ie, holosystolic murmur at the left lower sternal border) and pulmonic regurgitation (ie, diastolic decrescendo murmur).

(Choice E) Papillary muscle dysfunction, which is typically the result of myocardial ischemia/infarction, can cause mitral regurgitation (ie, holosystolic murmur). Marfan syndrome is not associated with papillary muscle dysfunction.

Educational objective:
Marfan syndrome is a connective tissue disorder in which defective fibrillin-1 decreases tissue integrity and

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support to elastic fibers and helps maintain connective tissue integrity. It also regulates extracellular matrix remodelling by binding to and sequestering **transforming growth factor-beta** (TGF-beta).

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Educational objective:
Marfan syndrome is a connective tissue disorder in which defective fibrillin-1 decreases tissue integrity and increases expression of transforming growth factor-beta. Cardiac effects include myxomatous degeneration and prolapse of the mitral valve with associated regurgitation, which manifests as a midsystolic click and mid-to-late systolic murmur.

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Correct answer
D

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Answered correctly

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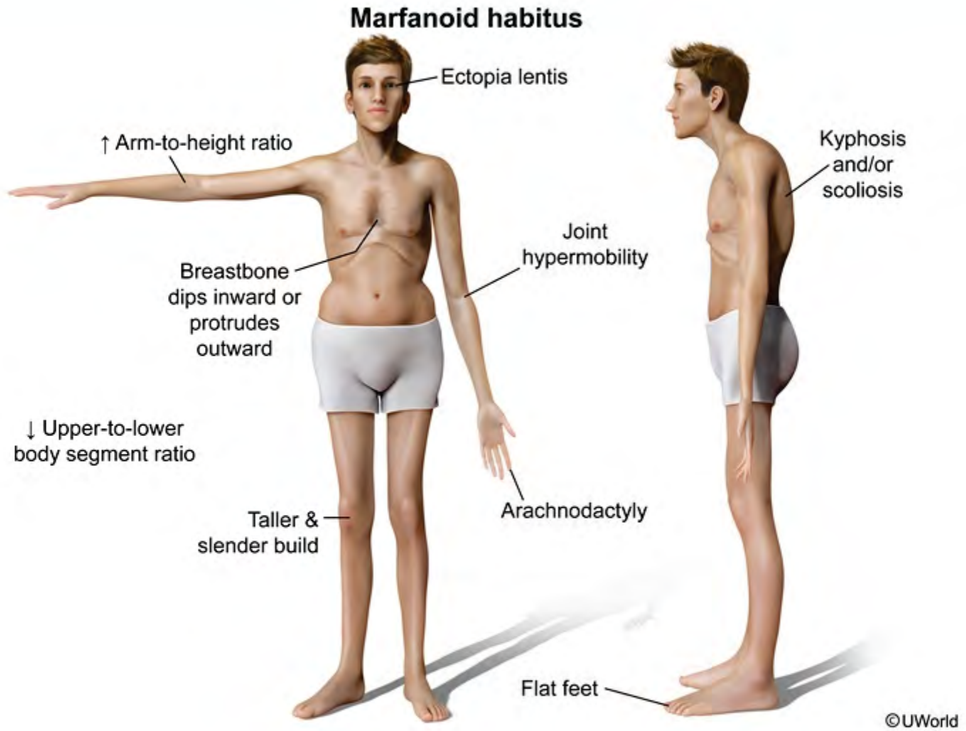

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regurgitation (ie, holosystolic murmur at the left lower sternal border) and pulmonic regurgitation (ie, diastolic decrescendo murmur).

(Choice E) Papillary muscle dysfunction, which is typically the result of myocardial ischemia/infarction, can cause

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Correct answer
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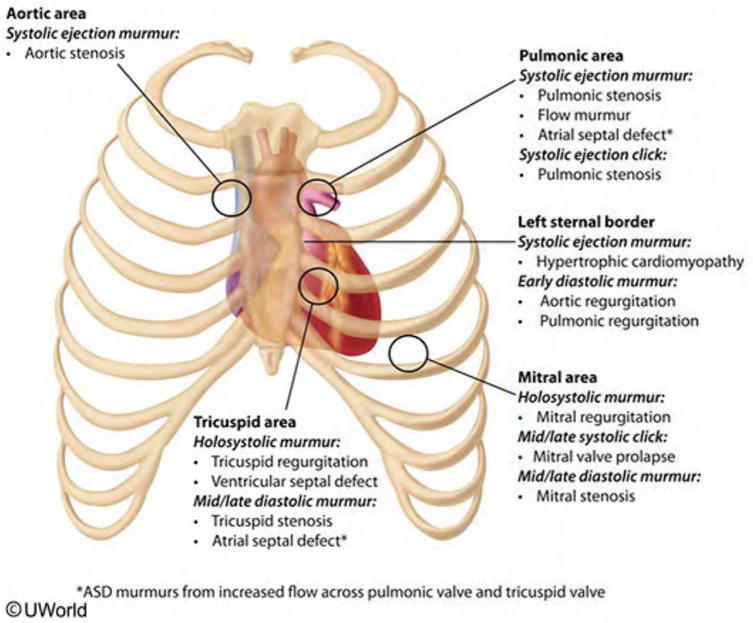

*ASD murmurs from increased flow across pulmonic valve and tricuspid valve

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(Choice E) Papillary muscle dysfunction, which is typically the result of myocardial ischemia/infarction, can cause

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support to elastic fibers and helps maintain connective tissue integrity. It also regulates extracellular matrix remodelling by binding to and sequestering **transforming growth factor-beta** (TGF-beta).

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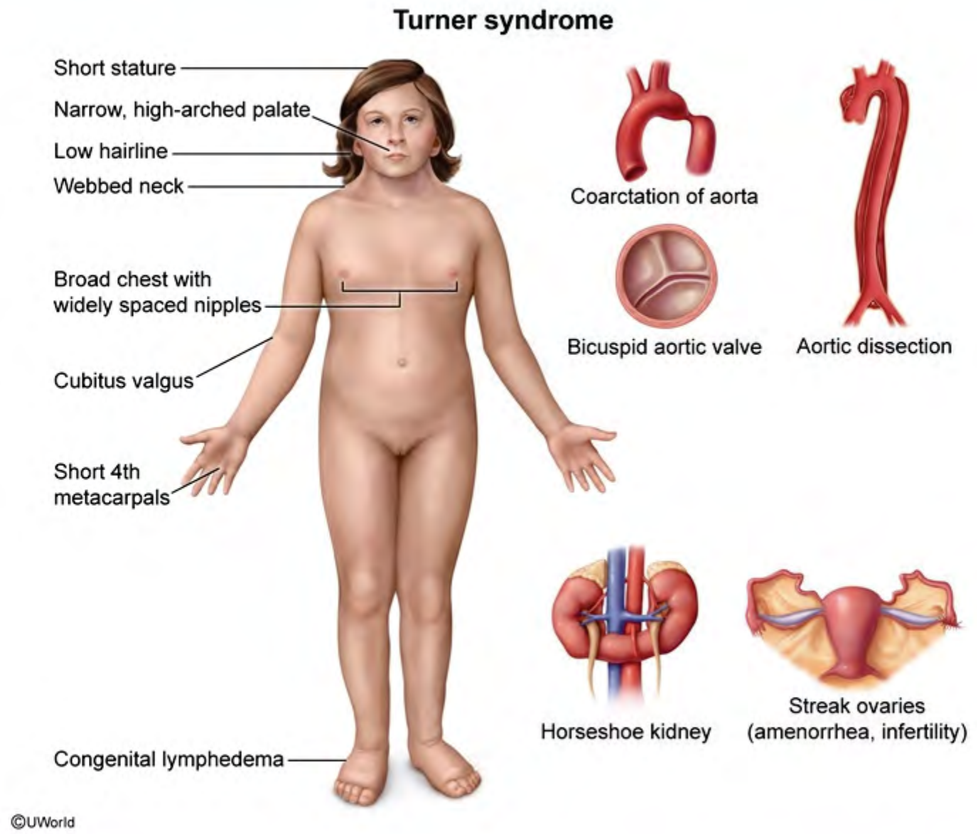

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A 64-year-old man has had difficulty walking for 6 months, experiencing muscle cramps in his right thigh after walking a block on level ground. The cramps subside quickly with rest. He has also had decreased sexual performance. Medical history is significant for hypercholesterolemia and myocardial infarction. The patient smokes a pack of cigarettes daily but does not use alcohol or illicit drugs. He is afebrile. Blood pressure is 128/78 mm Hg, pulse is 76/min, and respirations are 16/min. Cardiac auscultation reveals normal S1 and S2, and equal vesicular breath sounds are heard over the bilateral lungs. The abdomen is soft and without masses. There is no peripheral edema, muscle tenderness, or muscle weakness. Which of the following is the most likely cause of this patient's symptoms?

- A. Granulomatous inflammation of the arterial media
- B. Homogenous acellular thickening of the arteriolar walls
- C. Lipid-filled arterial intimal plaques
- D. Medial band–like arterial calcification
- E. Onion-like concentric thickening of the arteriolar walls

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- A. Granulomatous inflammation of the arterial media (4%)
- ✗ B. Homogenous acellular thickening of the arteriolar walls (6%)
- ✓ C. Lipid-filled arterial intimal plaques (74%)
- D. Medial band–like arterial calcification (6%)
- E. Onion-like concentric thickening of the arteriolar walls (9%)

Incorrect
Correct answer
C

74%
Answered correctly

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Explanation

This patient's muscle pain, which occurs with exercise and remits with rest, is consistent with **intermittent claudication**; smoking is an important risk factor. Claudication is usually due to atherosclerosis of the large arteries, specifically resulting from fixed stenoses caused by lipid-filled intimal plaques that bulge into the arterial lumen (**atheromas**). These stenoses prevent a sufficient increase in blood flow to muscles during exercise, leading to ischemic muscle pain. The pain is rapidly **relieved by rest**, as residual blood flow is adequate to meet the metabolic demands of resting, but not exercising, muscle.

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Explanation

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Thigh claudication is suggestive of occlusive disease of the ipsilateral **external iliac** artery or its more distal branches (ie, common femoral, superficial femoral, profunda femoris arteries). Accompanying **impotence** and/or **gluteal claudication** suggests more proximal **aortoiliac occlusion** (so-called Leriche syndrome), which, in addition to affecting the external iliac artery, also diminishes blood flow to the internal pudendal and gluteal branches of the internal iliac artery.

(Choice A) Granulomatous inflammation of the arterial media occurs in temporal (giant cell) arteritis, the most common form of systemic vasculitis in adults. This condition typically affects patients age >50 and causes headache, facial pain, jaw claudication, and visual loss.

(Choice B) Hyaline arteriolosclerosis involves homogeneous deposition of hyaline material in the intima and media of small arteries and arterioles, typically in patients with hypertension or diabetes mellitus (both absent in this patient). The underlying structure of the vessel wall is maintained, but the intima is thickened and the arterial lumen narrowed.

(Choice D) Medial band–like calcifications occur in medial calcific sclerosis (Mönckeberg arteriosclerosis). The condition is characterized by calcified deposits in muscular arteries and typically affects individuals age >50. Although visible radiologically and often palpable on physical examination, these calcifications are clinically asymptomatic because they do not narrow the vessel lumen.

(Choice E) Onion-like concentric thickening of arteriolar walls due to layers of smooth muscle cells and reduplicated basement membrane is seen in hyperplastic arteriolosclerosis, which can occur in severe chronic hypertension (eg, blood pressure >180/120 mm Hg). Although arterioles in all tissues may be affected, those in the

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the metabolic demands of resting, but not exercising, muscle.

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Educational objective:
Intermittent claudication describes muscle pain that is reproducibly caused by exercise and relieved by rest; it occurs due to atherosclerotic stenoses (lipid-filled intimal plaques) in the large arteries that prevent sufficient blood flow to exercising muscle. The lower extremities are most commonly affected; however, proximal lesions (ie, aortoiliac occlusion) can cause gluteal claudication and/or impotence.

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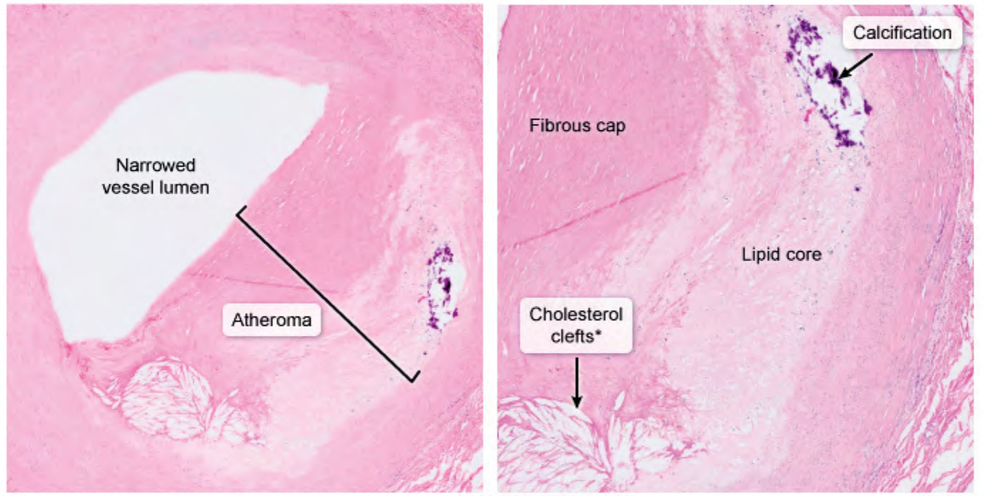
Atherosclerosis



*Cholesterol dissolves during tissue processing, leaving empty needle-shaped clefts.

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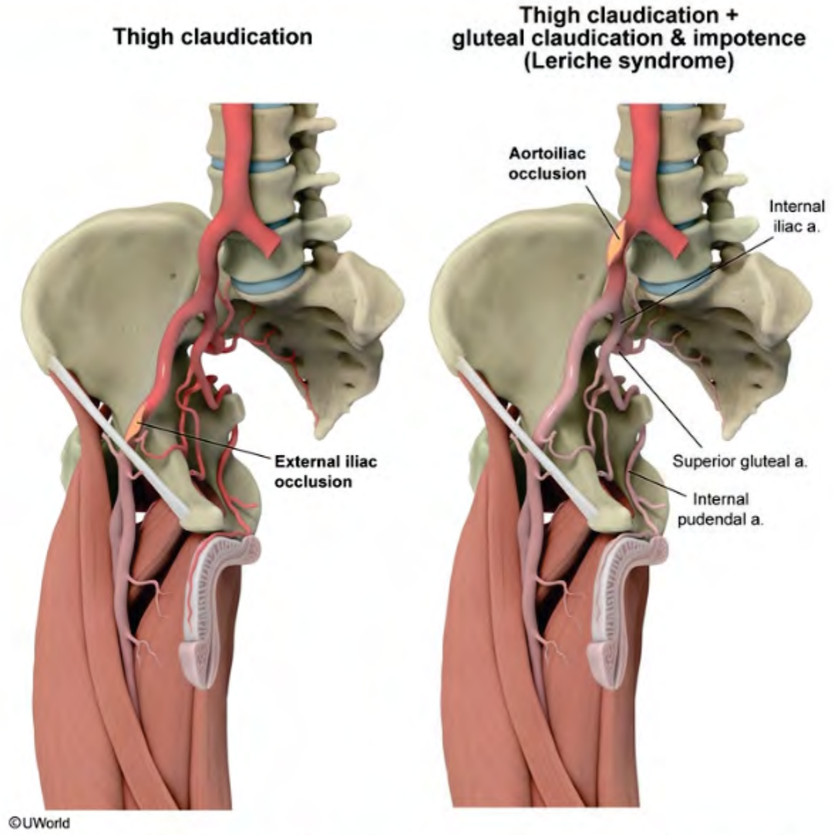

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condition is characterized by calcified deposits in muscular arteries and typically affects individuals age >50.

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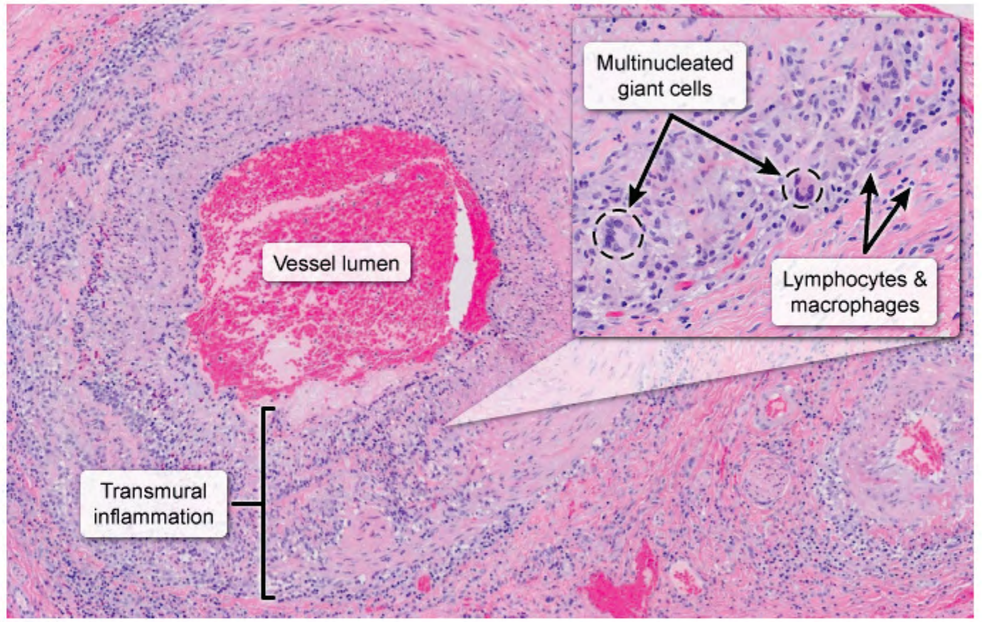
(Choice E) Onion-like concentric thickening of arteriolar walls due to layers of smooth muscle cells and

Giant cell arteritis



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claudication; smoking is an important risk factor. Claudication is usually due to atherosclerosis of the large arteries, specifically resulting from fixed stenoses caused by lipid-filled intimal plaques that bulge into the arterial lumen
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kidneys, retinas, and intestines are most common; skeletal muscle is less likely to be symptomatically involved.

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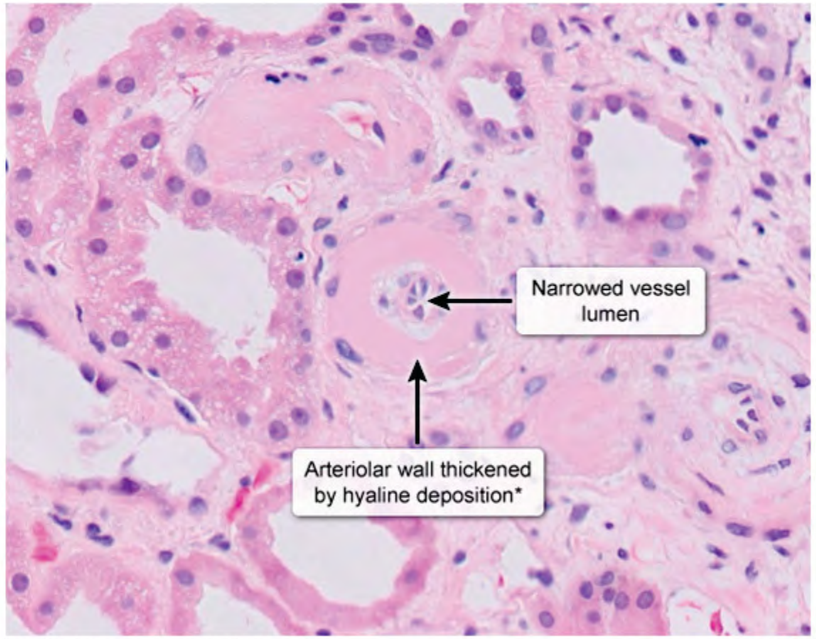
Hyaline arteriolosclerosis



*Homogenous, glassy, eosinophilic material.

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the metabolic demands of resting, but not exercising, muscle.

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occurs due to atherosclerotic stenoses (lipid-filled intimal plaques) in the large arteries that prevent sufficient blood

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Vascular calcifications

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gluteal claudication suggests more proximal **aortoiliac occlusion** (so-called Leriche syndrome), which, in

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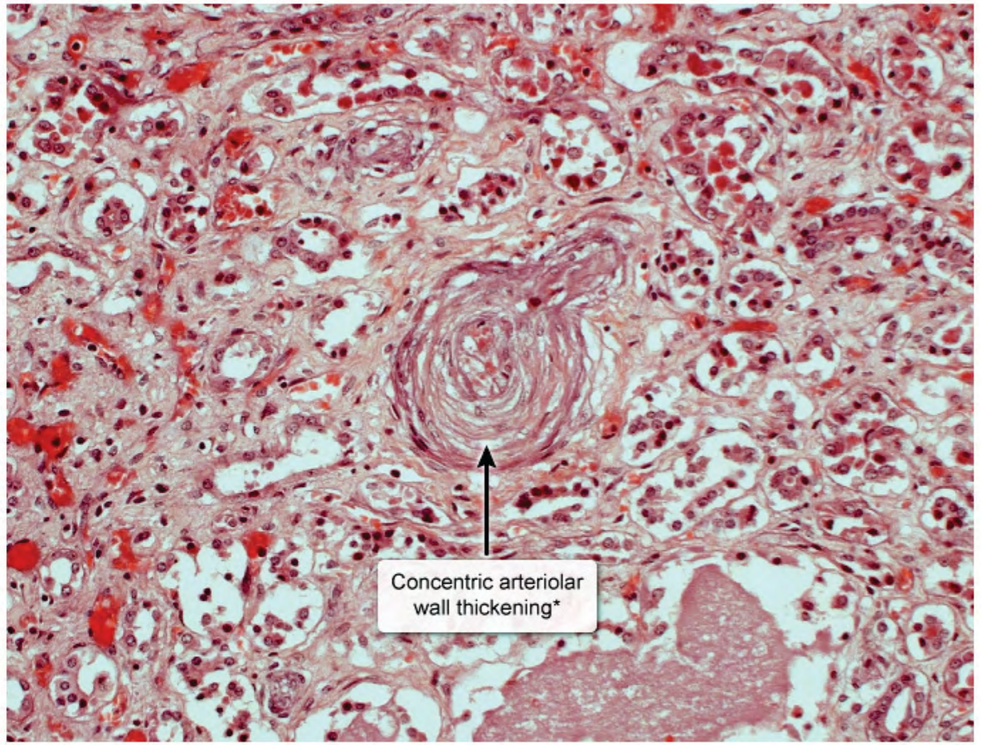
Hyperplastic arteriolosclerosis



*"Onion skin" appearance.

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Pathology | Cardiovascular System | Peripheral vascular disease

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A 72-year-old man is brought to the hospital due to severe substernal chest pain and shortness of breath for the last several hours. The pain started suddenly when he was outside working in the garden. Medical history includes diet-controlled diabetes mellitus and hypertension. Physical examination reveals bilateral crackles and a third heart sound. ECG shows ST elevation in multiple leads. The patient is diagnosed with an acute myocardial infarction complicated by acute severe heart failure. He is started on dobutamine infusion and prepared for coronary catheterization. Which of the following is most likely to be increased due to the medication used in this patient?

- A. Cardiac diastolic filling time
- B. Myocardial oxygen consumption
- C. Peripheral vascular resistance
- D. Pulmonary capillary wedge pressure
- E. Right ventricular end diastolic pressure

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- ✗ A. Cardiac diastolic filling time (23%)
- ✓ B. Myocardial oxygen consumption (53%)
- C. Peripheral vascular resistance (8%)
- D. Pulmonary capillary wedge pressure (5%)
- E. Right ventricular end diastolic pressure (8%)

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Correct answer
B

53%
Answered correctly

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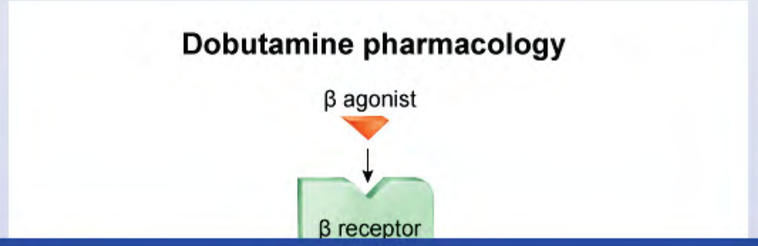
Explanation



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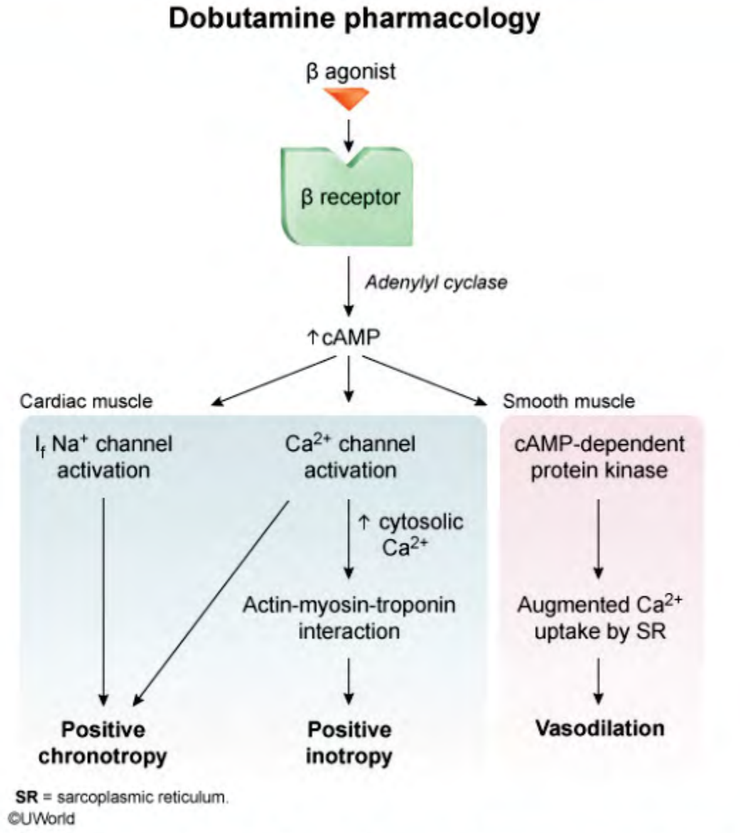

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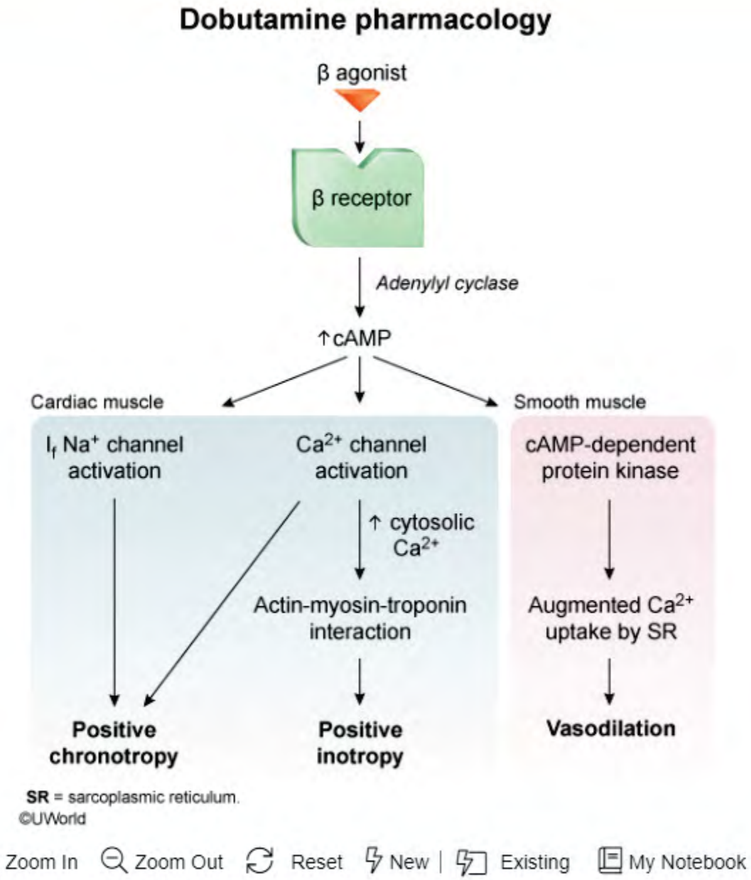

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decrease in cardiac diastolic filling time.

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SR = sarcoplasmic reticulum.
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Dobutamine is a beta adrenergic agonist with predominant activity on **beta-1 receptors**, weaker activity on beta-2 receptors, and minimal activity on alpha-1 receptors. It is used for management of refractory heart failure associated with severe left ventricular systolic dysfunction and **cardiogenic shock**. Stimulation of beta adrenergic receptors results in increased production of cAMP in target cells and causes the following effects:

- **Positive inotropy and chronotropy**: Increased cardiac contractility (potent effect) and heart rate (weaker effect), leading to increased cardiac output (improves end-organ perfusion) and decreased left ventricular filling pressures (improves pulmonary congestion/edema)
- Mild **vasodilation**: Decreased systemic vascular resistance that often causes a slight reduction in blood pressure; this avoids the increase in afterload seen with other vasopressor/inotropic agents (eg, norepinephrine) but limits its usefulness in severely hypotensive patients

The strong inotropic effect of dobutamine significantly **increases myocardial oxygen consumption**, which can trigger or exacerbate myocardial ischemia. As such, dobutamine should not be used routinely in patients with decompensated heart failure. However, in patients with cardiogenic shock, this drawback is often **outweighed** by improvement in cardiac output and end-organ perfusion.

(Choice A) Dobutamine is a weak chronotropic agent that causes an increase in heart rate. This leads to a decrease in cardiac diastolic filling time.

(Choice C) In the vasculature, the beta-2 agonist effects (vasodilation) of dobutamine are somewhat stronger than its alpha-1 agonist activity (vasoconstriction), resulting in overall mild vasodilation. The net hemodynamic effects are a decrease in systemic vascular resistance with or without a slight reduction in blood pressure.

(Choices D and E) Dobutamine causes decreased pulmonary capillary wedge pressure and left and right ventricular filling pressures due to the increased cardiac contractility and output.

Educational objective:

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Educational objective:
Dobutamine is a beta adrenergic agonist with predominant activity on beta-1 receptors. It causes an increase in heart rate and cardiac contractility, leading to an increase in myocardial oxygen consumption.

References

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A 68-year-old man is brought to the emergency department with chest pain. For the last year, the patient has had exertional chest pain that has progressively worsened. He was shoveling snow this morning when the pain became unbearable. He has smoked a pack of cigarettes daily for 40 years. Blood pressure is 140/100 mm Hg and pulse is 90/min. Examination showed mild diaphoresis. The lungs are clear to auscultation. ECG shows ST- and T-wave changes suggestive of ischemia. Cardiac enzymes are elevated. Emergent coronary angiography is performed, which demonstrates significant atherosclerotic involvement of the left anterior descending and circumflex arteries. Development of these vascular lesions most likely involved growth factors released from which of the following sources?

- A. B lymphocytes
- B. Eosinophils
- C. Erythrocytes
- D. Mast cells
- E. Neutrophils
- F. Platelets

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- ✗ A. B lymphocytes (2%)
- B. Eosinophils (1%)
- C. Erythrocytes (2%)
- D. Mast cells (7%)
- E. Neutrophils (22%)
- ✓ F. Platelets (64%)

Incorrect
Correct answer
F

64%
Answered correctly

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Explanation

Formation of atheroma

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A 68-year-old man is brought to the emergency department with chest pain. For the last year, the patient has had exertic
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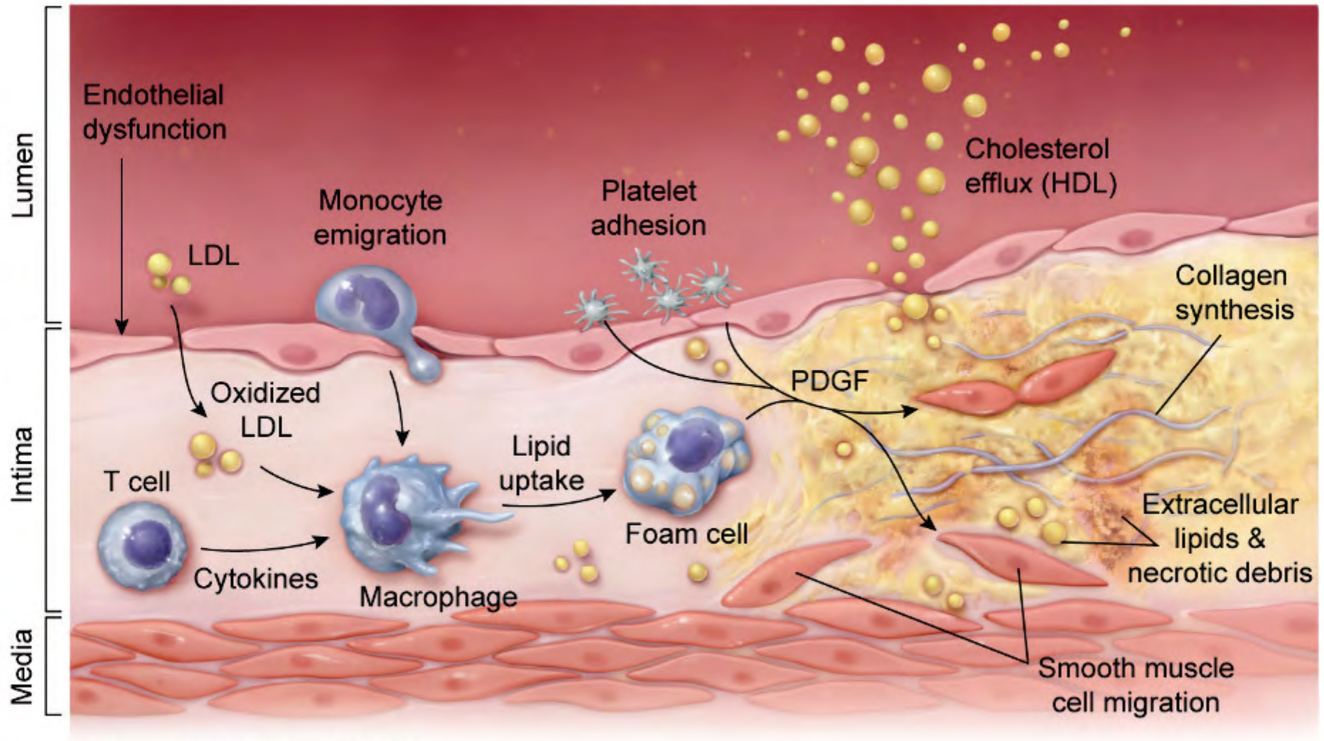
Formation of atheroma



PDGF = platelet-derived growth factor.

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The development of atherosclerotic plaque, or atheroma, involves a multistep process:

- Initially, factors including hyperlipidemia, hypertension, hyperglycemia, and smoking trigger **endothelial injury and/or dysfunction**. This leads to increased vascular permeability, enhanced leukocyte adhesion, and a higher propensity for thrombosis.
- Lipoproteins (ie, LDL and oxidized LDL) enter the arterial wall intima and begin to accumulate. Monocytes adhere to the endothelial wall and enter into the intima as well; these cells transform into macrophages and engulf lipid particles to become foam cells. **Platelets** adhere to the abnormal endothelium and become activated.
- Growth factors, namely **platelet-derived growth factor** (PDGF), are released from platelets, **activated macrophages**, and endothelial cells. This triggers **smooth muscle cell** (SMC) recruitment from the media and **proliferation** in the intima.

Over time, progressive SMC proliferation and accumulation of necrotic debris (due to macrophage/foam cell and SMC death) lead to growth of the atheroma. SMCs encourage plaque stability by synthesizing collagen, whereas activated inflammatory cells break down collagen and contribute to plaque instability. HDL likely extracts lipids from the intima back into the bloodstream and helps slow atheroma development.

(Choice A) B lymphocytes are not known to play a significant role in atheroma development. However, T lymphocytes in the arterial wall intima likely play a role in activating macrophages, endothelial cells, and SMCs by releasing inflammatory cytokines (eg, interferon-γ).

(Choices B and D) Eosinophils and mast cells are important in IgE-mediated (Type I) hypersensitivity reactions; mast cells release histamine in the early phase, and eosinophils release leukotrienes and major basic protein in the late phase of the reaction. However, these cells are not significantly involved in atheroma development.

(Choices C and E) Neither erythrocytes nor neutrophils release growth factors or play a significant role in atheroma development.

Educational objective:

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higher propensity for thrombosis.

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Educational objective:
During the development of atherosclerotic plaque (atheroma), activated macrophages, platelets, and endothelial cells release growth factors (eg, platelet-derived growth factor) that stimulate recruitment of smooth muscle cells from the arterial wall media and their subsequent proliferation in the intima.

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A 59-year-old African American male presents to the emergency room with crushing chest pain, sweating, and lightheadedness. His blood pressure is 90/60 mm Hg and his heart rate is 48 beats per minute. Electrocardiogram (ECG) shows sinus bradycardia and ST segment elevation in leads II, III, and aVF. Occlusion of which of the following coronary arteries is most likely responsible for this patient's symptoms?

- A. Left main coronary artery
- B. Left anterior descending artery
- C. Left circumflex artery
- D. Right coronary artery
- E. Intramural arteries

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A 59-year-old African American male presents to the emergency room with crushing chest pain, sweating, and lightheadedness. His blood pressure is 90/60 mm Hg and his heart rate is 48 beats per minute. Electrocardiogram (ECG) shows sinus bradycardia and ST segment elevation in leads II, III, and aVF. Occlusion of which of the following coronary arteries is most likely responsible for this patient's symptoms?

- A. Left main coronary artery (2%)
- ✗ B. Left anterior descending artery (35%)
- C. Left circumflex artery (5%)
- ✓ D. Right coronary artery (55%)
- E. Intramural arteries (1%)

Incorrect
Correct answer
D

55%
Answered correctly

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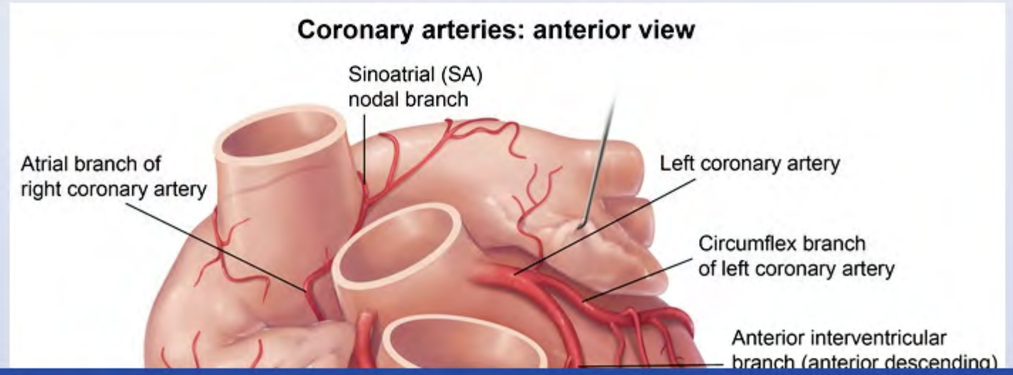
Explanation



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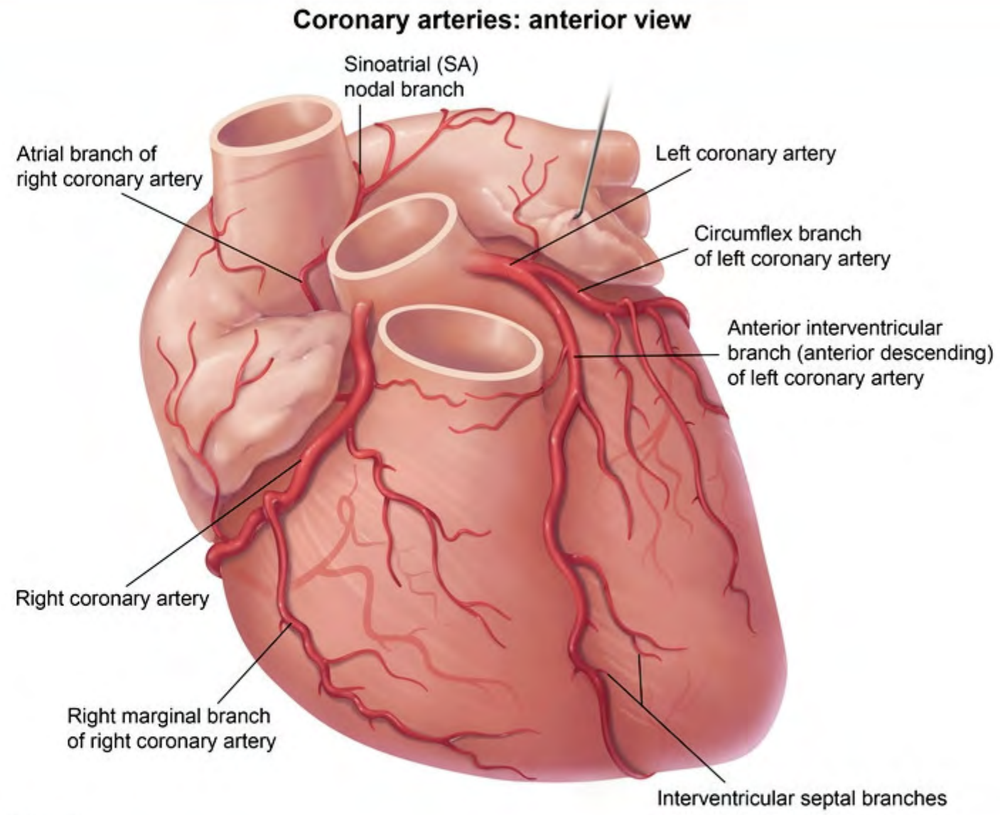

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Anterior interventricular branch (anterior descending)

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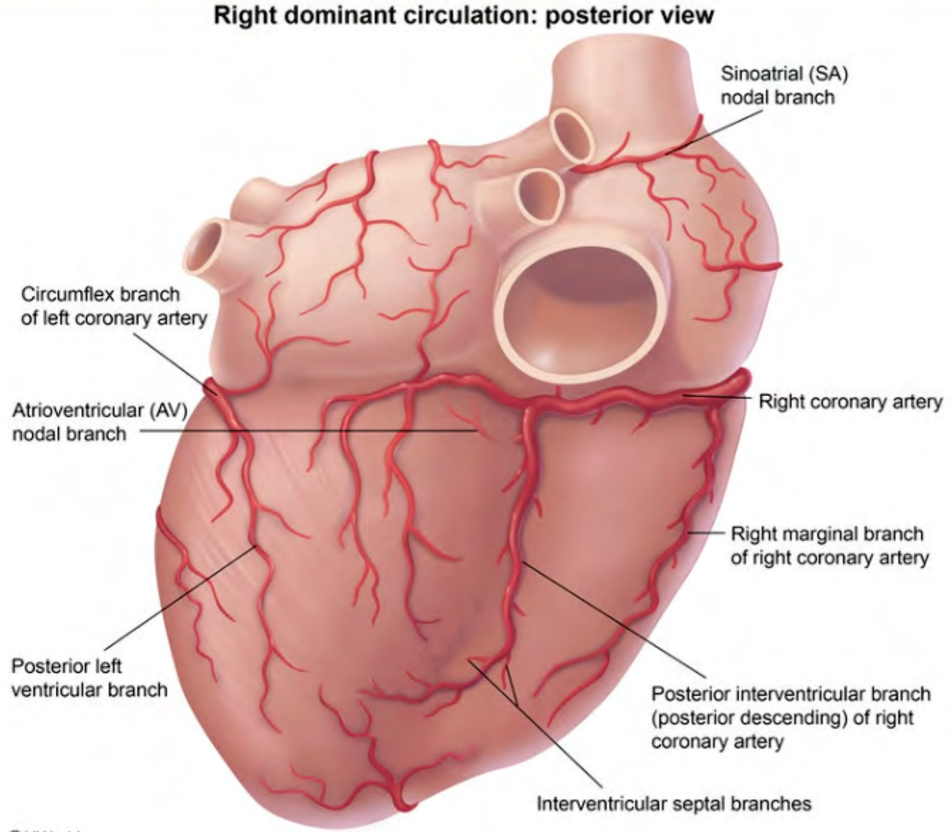

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the epicardial coronary arteries, they are rarely affected by atherosclerosis. Moreover, if occlusion of intramural

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Interventricular septal branches

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Click here for posterior view.

This patient presents with symptoms and ECG findings consistent with transmural ischemic injury to the inferior wall of the heart. In 90% of individuals, the posteroinferior wall of the left ventricle is supplied by the posterior descending branch of the right coronary artery (RCA). The patient's bradycardia and resultant hypotension suggests that there may have been ischemic injury to the sinus node as well. (The sinus node normally receives its arterial blood supply from the RCA.)

(Choices A, B, and C) The left main coronary artery gives rise to the left anterior descending (LAD) and the left circumflex (LCX) coronary arteries. The LAD supplies the interventricular septum and the anterior wall of the left ventricle. Transmural ischemia of the septum would produce ST elevation mainly in leads V1 and V2. Infranodal (Mobitz type II) second-degree or third-degree heart block is a possible result as well; but sinus bradycardia would not occur with this type of injury. Transmural ischemia of the anterior left ventricular wall would produce ST elevations mainly in leads V3 and V4. An occlusion of the proximal LAD would cause anteroseptal transmural ischemia, with ST elevations in leads V1–V4.

The LCX supplies the lateral wall of the left ventricle. Transmural ischemia secondary to LCX occlusion would produce ST elevations mainly in leads V5 and V6, and possibly also in leads I and aVL. An occlusion of the left main coronary artery would therefore be expected to produce widespread transmural ischemia of the interventricular septum and left ventricle, with ST elevations in all of the chest (V) leads, and possibly also in leads I and aVL.

(Choice E) Intramural arteries in the substance of the myocardium are unlikely sites of occlusion because, unlike the epicardial coronary arteries, they are rarely affected by atherosclerosis. Moreover, if occlusion of intramural arteries did occur, subendocardial ischemia (with associated ST depression) would be more likely than transmural ischemia (with ST elevation).

Educational Objective:

In 90% of individuals, occlusion of the right coronary artery can result in transmural ischemia of the inferior wall of

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its arterial blood supply from the RCA.)

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Educational Objective:
In 90% of individuals, occlusion of the right coronary artery can result in transmural ischemia of the inferior wall of the left ventricle, producing ST elevation in leads II, III, and aVF as well as possible sinus node dysfunction. Occlusion of the proximal LAD would be expected to result in anteroseptal transmural ischemia, with ST elevations in leads V1–V4. Occlusion of the LCX would produce transmural ischemia of the lateral wall of the left ventricle, with ST elevations mainly in V5 and V6, and possibly also in I and aVL.

Pathology	Cardiovascular System	Coronary blood flow
Subject	System	Topic

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A 63-year-old man comes to the emergency department with 2 hours of chest pain and shortness of breath. The pain started as lower sternal discomfort that he attributed to indigestion, but then spread all over the anterior chest. The patient has never had similar pain before. Medical history includes hypertension, hyperlipidemia, and diet-controlled type 2 diabetes mellitus. The patient is an ex-smoker with a 20-pack-year history. On cardiac auscultation, a systolic murmur is heard. Basilar crackles are present on both sides. After initial evaluation, the patient is taken to the cardiac catheterization laboratory and is found to have a coronary artery occlusion, which is successfully revascularized. The next morning, the patient appears comfortable and wants to go home. The lungs are clear on auscultation, and no heart murmur is present. The murmur heard during initial presentation is most likely explained by a pathologic process involving which of the following structures?

- A. Aortic root
- B. Aortic valve leaflets
- C. Interventricular septum
- D. Mitral valve chordae
- E. Papillary muscle

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- ✗ A. Aortic root (7%)
- B. Aortic valve leaflets (17%)
- C. Interventricular septum (7%)
- D. Mitral valve chordae (22%)
- ✓ E. Papillary muscle (46%)

Incorrect
Correct answer
E

46%
Answered correctly

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successfully revascularized. The next morning, the patient appears comfortable and wants to go home. The lungs are cle
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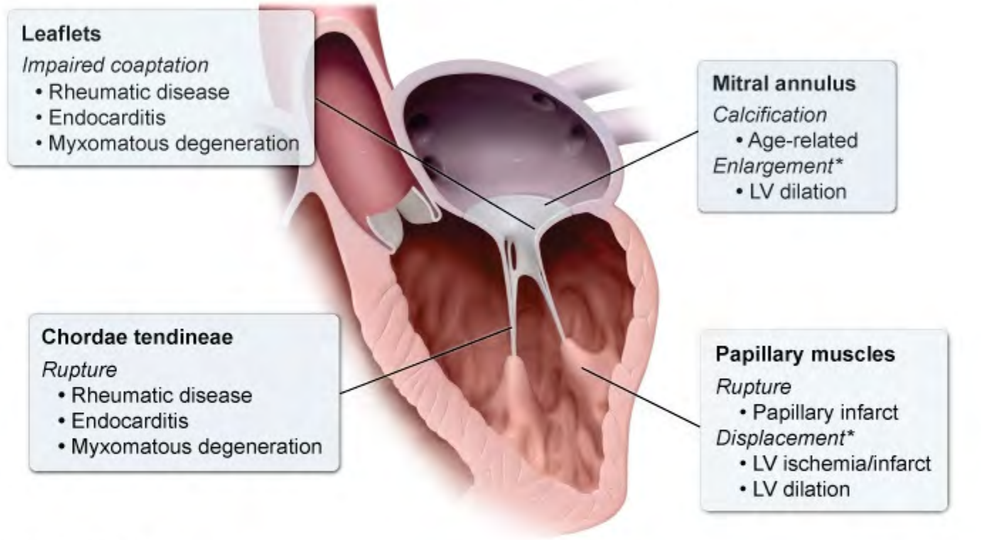
Causes of mitral regurgitation



LV = left ventricular.
*Secondary cause of mitral regurgitation.

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Chordae tendineae

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LV = left ventricular.
*Secondary cause of mitral regurgitation.

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This patient with **myocardial infarction** developed a **new systolic murmur** that resolved following revascularization, which is consistent with **mitral regurgitation** (MR) due to papillary muscle dysfunction. Myocardial infarction can cause **ischemia** of the **papillary muscle** and the adjacent LV wall on which it is mounted. This results in **hypokinesis and outward displacement** of the papillary muscle, creating increased tension on the attached chordae tendineae and preventing complete closure of the corresponding mitral valve cusp. Timely restoration of adequate blood supply with coronary revascularization restores papillary muscle and LV wall motion, often leading to resolution of the MR.

In contrast to papillary muscle dysfunction, papillary muscle rupture is a mechanical complication of MI that occurs acutely or within 3-5 days and leads to acute MR; it does not resolve with coronary revascularization and typically requires surgical repair.

(Choices A and B) Aortic root and aortic valve leaflet function are not directly affected by myocardial ischemia and would not explain a systolic murmur during acute MI.

(Choice C) Rupture of the interventricular septum can occur acutely or within 3-5 days of acute MI. Patients have sudden-onset hypotension, dyspnea, pulmonary edema, and a harsh, holosystolic murmur at the left sternal border with palpable thrill. The condition does not resolve with coronary revascularization and typically requires surgical repair.

(Choice D) Mitral valve chordae tendineae are not directly affected by myocardial ischemia; however, rupture resulting in acute MR can occur in patients with myxomatous mitral valve disease (mitral valve prolapse), rheumatic fever, or endocarditis.

Educational objective:
Myocardial infarction that causes ischemia of the papillary muscle or nearby left ventricular wall can result in acute mitral regurgitation with development of a new systolic murmur. Timely restoration of blood flow can improve

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This patient with **myocardial infarction** developed a **new systolic murmur** that resolved following revascularization, which is consistent with **mitral regurgitation** (MR) due to papillary muscle dysfunction. Myocardial infarction can cause **ischemia** of the **papillary muscle** and the adjacent LV wall on which it is mounted. This results in **hypokinesis and outward displacement** of the papillary muscle, creating increased tension on the attached chordae tendineae and preventing complete closure of the corresponding mitral valve cusp. Timely restoration of adequate blood supply with coronary revascularization restores papillary muscle and LV wall motion, often leading to resolution of the MR.

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Educational objective:
Myocardial infarction that causes ischemia of the papillary muscle or nearby left ventricular wall can result in acute mitral regurgitation with development of a new systolic murmur. Timely restoration of blood flow can improve papillary muscle dysfunction and lead to resolution of the regurgitation.

References

- Acute valvular regurgitation.

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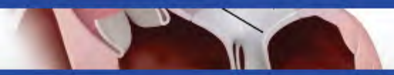

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Mechanical complications of acute myocardial infarction

Complication	Time course	Clinical findings
Papillary muscle rupture/dysfunction*	Acute or within 3-5 days	• Severe pulmonary edema, respiratory distress • New early systolic murmur (acute MR) • Hypotension/cardiogenic shock
Interventricular septum rupture	Acute or within 3-5 days	• Chest pain • New holosystolic murmur • Hypotension/cardiogenic shock • Step up in O_2 level from RA to RV
Free wall rupture**	Within 5 days or up to 2 weeks	• Chest pain • Distant heart sounds • Shock, rapid progression to cardiac arrest
Left ventricular aneurysm**	Up to several months	• Heart failure • Angina, ventricular arrhythmias

*Usually due to right coronary artery occlusion.
**Usually due to left anterior descending artery occlusion.
MR = mitral regurgitation; **RA** = right atrium; **RV** = right ventricle.

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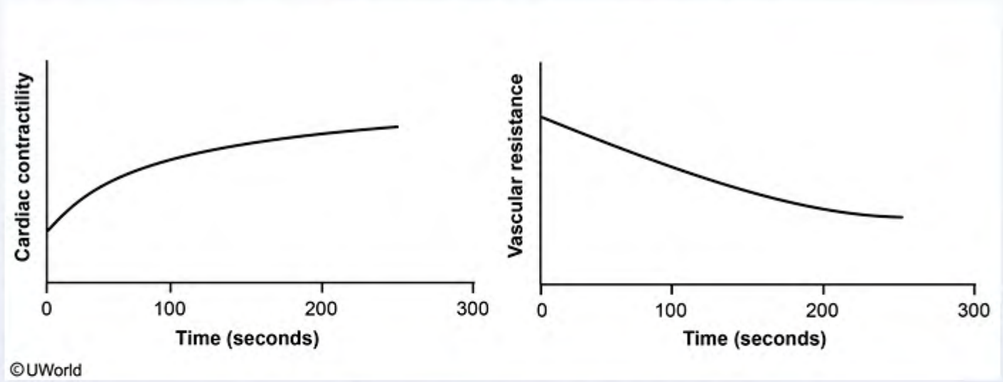
A 66-year-old man is evaluated for recurrent syncope. He has had 3 episodes of dizziness and palpitations followed by brief loss of consciousness over the last 6 months. He has no chest pain or dyspnea. The patient has a history of hypertension and hyperlipidemia. There is no family history of sudden death. Initial evaluation shows normal ECG and echocardiogram. A cardiac electrophysiologic study is performed. During the study, intravenous infusion of a medication is administered and produces the responses shown below.



Which of the following medications was most likely administered to this patient?

- (●) A. Acetylcholine
- ( ) B. Adenosine
- ( ) C. Clonidine
- ( ) D. Esmolol
- ( ) E. Isoproterenol
- ( ) F. Norepinephrine
- ( ) G. Phenylephrine

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A 66-year-old man is evaluated for recurrent syncope. He has had 3 episodes of dizziness and palpitations
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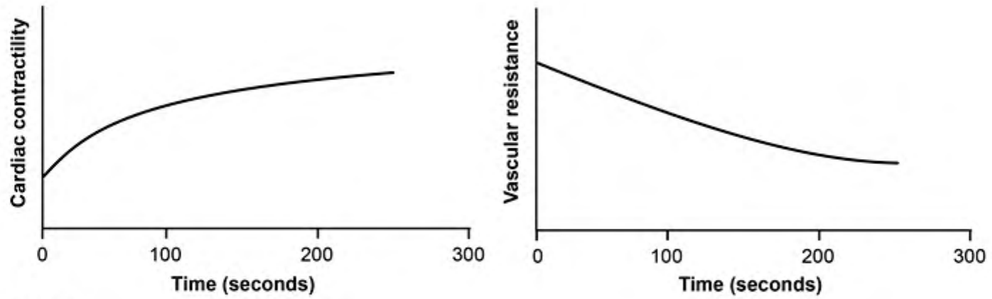

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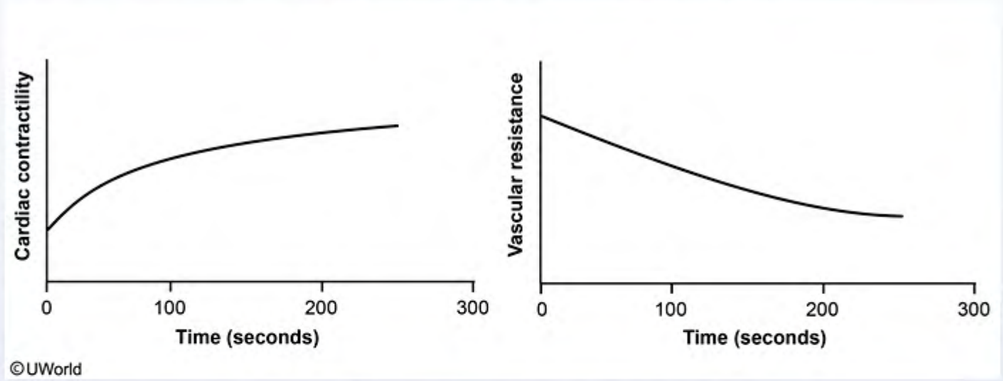

Which of the following medications was most likely administered to this patient?

- ✗ A. Acetylcholine (1%)
- B. Adenosine (11%)
- C. Clonidine (5%)
- D. Esmolol (7%)
- ✓ E. Isoproterenol (55%)
- F. Norepinephrine (12%)
- G. Phenylephrine (5%)

Incorrect
Correct answer

55%
Answered correctly

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Explanation

Effects of adrenergic drugs			
Receptor	**Target organ**	**Effect of stimulation**	**Examples**
α-1	• Peripheral vasculature • Bladder • Eye	• ↑ Blood pressure (especially systolic) • Contraction of internal urethral sphincter • Mydriasis (contraction of pupillary dilator muscle)	• Epinephrine • Norepinephrine • Phenylephrine
β-1	• Heart	• ↑ Heart rate, contractility & conductance	• Epinephrine • Dopamine • Dobutamine • Isoproterenol
β-2	• Peripheral vasculature (smooth muscle) • Bronchi • Uterus	• Vasodilation, ↓ blood pressure (especially diastolic) • Bronchodilation • Relaxation (tocolysis)	• Isoproterenol • Terbutaline

The effects seen in the graphs above are most similar to those elicited by **isoproterenol**, a nonselective β adrenergic agonist. Isoproterenol **increases cardiac contractility** (positive inotropic effect) by acting on myocardial β-1 adrenergic receptors. It also binds to β-2 adrenergic receptors and causes vasodilation by relaxation of vascular smooth muscle, leading to **decreased vascular resistance** and mean arterial blood pressure. Isoproterenol has negligible effects on α-1 adrenergic receptors.

(Choice A) Acetylcholine binds to M_2 muscarinic receptors in the heart and M_3 receptors in the vasculature and causes decreased heart rate and conduction velocity and vasodilation (indirectly mediated by endothelial nitric oxide release). Acetylcholine has no significant effect on ventricular contractility.

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(Choice B) Adenosine acts on A1 receptors on cardiac cells and activates potassium channels, increasing potassium conductance. This leads to transient conduction delay through the atrioventricular node. Adenosine can also cause peripheral vasodilation, but it attenuates the adrenergic-mediated increase in myocardial contractility.

(Choice C) Clonidine stimulates α_2 adrenoceptors in the brainstem, resulting in reduced sympathetic outflow from the central nervous system, leading to a decrease in peripheral vascular resistance, heart rate, and blood pressure. It has no effect on cardiac contractility.

(Choice D) Esmolol is a selective β-1 adrenergic blocking agent with negative inotropic and chronotropic effects. It would cause a decrease in cardiac contractility.

(Choice F) Norepinephrine acts on α-1 receptors, causing vasoconstriction and an increase in systemic vascular resistance. Norepinephrine also acts as a weak agonist at β-1 receptors, with a modest increase in myocardial contractility.

(Choice G) Phenylephrine is a sympathomimetic drug with pure α adrenergic agonist activity. Infusion of this drug causes vasoconstriction with an increase in systemic vascular resistance.

Educational objective:
Isoproterenol is a β-1 and β-2 adrenergic receptor agonist that causes increased myocardial contractility and decreased systemic vascular resistance.

Pharmacology Cardiovascular System Sympathomimetic agents

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A 52-year-old woman comes to the office due to fatigue for the last 2 weeks. The patient was previously healthy, but over the past 3 months, she has had significant weight loss without changing her diet. She also reports increased sweating, hand tremor, and decreased sleep. Examination shows diffuse, nontender enlargement of the thyroid gland. The eyeballs are protuberant. The remainder of the examination is notable for 1+ pitting edema in the ankles. Which of the following parameters is most likely to be decreased in this patient?

- A. Cardiac contractility
- B. Myocardial oxygen consumption
- C. Pulmonary artery pressure
- D. Pulse pressure
- E. Systemic vascular resistance
- F. Systolic blood pressure

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A 52-year-old woman comes to the office due to fatigue for the last 2 weeks. The patient was previously healthy, but over the past 3 months, she has had significant weight loss without changing her diet. She also reports increased sweating, hand tremor, and decreased sleep. Examination shows diffuse, nontender enlargement of the thyroid gland. The eyeballs are protuberant. The remainder of the examination is notable for 1+ pitting edema in the ankles. Which of the following parameters is most likely to be decreased in this patient?

- ✗ A. Cardiac contractility (4%)
- B. Myocardial oxygen consumption (4%)
- C. Pulmonary artery pressure (17%)
- D. Pulse pressure (31%)
- ✓ E. Systemic vascular resistance (39%)
- F. Systolic blood pressure (1%)

Incorrect
Correct answer
E

39%
Answered correctly

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Explanation

Cardiovascular effects of hyperthyroidism	
Increased rate	• Tachycardia/palpitations • Atrial fibrillation
Increased contractility	• ↑ Ejection fraction & cardiac output • ↑ Myocardial oxygen demand & angina • ↑ Pulmonary artery pressure
Peripheral vasodilation	• ↓ Systemic vascular resistance

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Cardiovascular effects of hyperthyroidism	
Increased rate	• Tachycardia/palpitations • Atrial fibrillation
Increased contractility	• ↑ Ejection fraction & cardiac output • ↑ Myocardial oxygen demand & angina • ↑ Pulmonary artery pressure
Peripheral vasodilation	• ↓ Systemic vascular resistance
Additional effects	• ↓ Diastolic pressure • ↑ Systolic pressure • ↑ Pulse pressure • High-output heart failure

This patient with an enlarged thyroid gland and multiple consistent symptoms (eg, fatigue, unintentional weight loss, sweating, tremor, insomnia) most likely has **hyperthyroidism**. Protuberance of the eyeballs (ie, exophthalmos) suggests Graves disease, specifically.

In adults, the major effect of thyroid hormone is control of cellular metabolism, including the rate of oxygen consumption and use of proteins, carbohydrates, and lipids. As a result, hyperthyroidism causes characteristic **cardiovascular effects**:

- Increased metabolic demand in the peripheral tissues, combined with a direct effect of thyroid hormone on vascular smooth muscle, causes **peripheral vasodilation,** which leads to **decreased systemic vascular resistance** (SVR) and reduced diastolic blood pressure.
- Thyroid hormone has a direct **sympathetic-like effect** on the myocardium, which stimulates increased heart rate and contractility **(Choice A)**. A reflexive response to decreased SVR also contributes to these changes. With increased contractility, stroke volume is increased, resulting in increased pulse pressure and systolic blood pressure **(Choices D and F)**. Ejection fraction and cardiac output are also increased.

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Over time, some patients develop **high-output heart failure** due to the hyperdynamic circulatory state. Restoration of a euthyroid state normalizes cardiovascular hemodynamics and typically resolves any heart failure that has developed.

(Choice B) Myocardial oxygen consumption increases in hyperthyroidism, caused by a direct effect of thyroid hormone and by hemodynamic changes that increase myocardial oxygen demand (ie, increased heart rate, contractility, and afterload [systolic blood pressure]).

(Choice C) Both diastolic and systolic pulmonary artery pressures increase in hyperthyroidism. This is likely because cardiac output increases, but the pulmonary circulation does not undergo the same vasodilation and reduction in resistance that occurs in the systemic circulation (pressure = cardiac output × resistance).

Educational objective:
Hyperthyroidism causes characteristic cardiovascular changes. Increased metabolic demand in the tissues and a direct vasodilatory effect of thyroid hormone lead to reduced systemic vascular resistance (and decreased diastolic blood pressure). A direct sympathetic-like effect of thyroid hormone on the myocardium leads to increased heart rate, contractility, and cardiac output. Increased stroke volume increases pulse pressure and systolic blood pressure.

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This patient with an enlarged thyroid gland and multiple consistent symptoms (eg, fatigue, unintentional weight loss, sweating, tremor, insomnia) most likely has **hyperthyroidism**. Protuberance of the eyeballs (ie, exophthalmos) suggests Graves disease, specifically.

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Educational objective:
Hyperthyroidism causes characteristic cardiovascular changes. Increased metabolic demand in the tissues and a direct vasodilatory effect of thyroid hormone lead to reduced systemic vascular resistance (and decreased diastolic blood pressure). A direct sympathetic-like effect of thyroid hormone on the myocardium leads to increased heart

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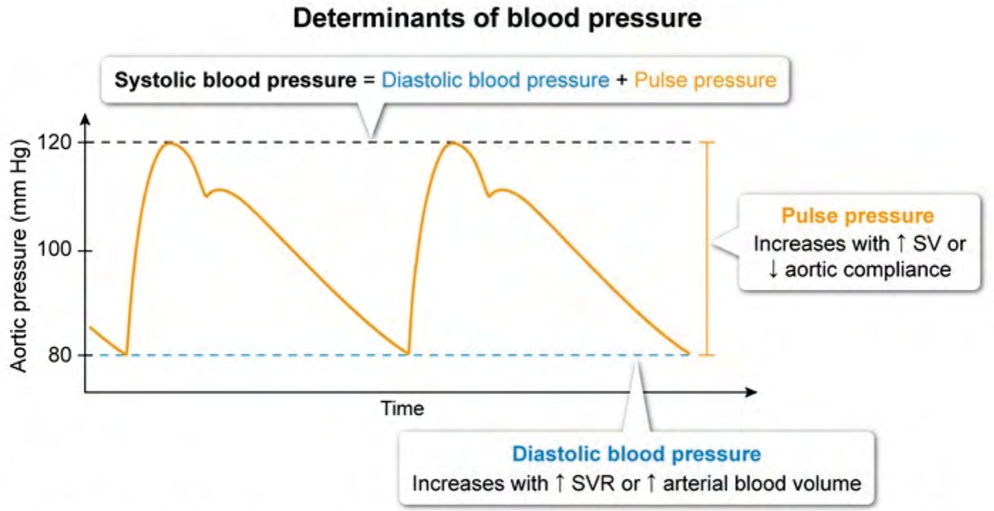
Educational objective:
Hyperthyroidism causes characteristic cardiovascular changes. Increased metabolic demand in the tissues and a direct vasodilatory effect of thyroid hormone lead to reduced systemic vascular resistance (and decreased diastolic blood pressure). A direct sympathetic-like effect of thyroid hormone on the myocardium leads to increased heart

Exhibit Display



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A 1-week-old boy is brought to the office for his first primary care visit following an uncomplicated vaginal delivery at 40 weeks gestation. The patient was born to a 30-year-old woman who took prenatal vitamins throughout the pregnancy. The nursery course was uncomplicated, and he was discharged at approximately 30 hours of life following observation of appropriate breastfeeding, voiding, and stooling. Weight, length, and head circumference are at the 50th percentile. Physical examination shows a grade II/VI harsh, holosystolic murmur best heard at the left-mid to left-lower sternal border. The rest of the examination is unremarkable. Review of medical records shows absence of the murmur at birth. This patient's findings are most likely due to which of the following embryologic events?

- A. Arrested growth of the septum secundum
- B. Failed fusion between the septum primum and endocardial cushions
- C. Failed obliteration of the vascular connection between the pulmonary artery and aorta
- D. Insufficient development of the interventricular septum
- E. Malalignment of the infundibular septum

Submit

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- ✗ B. Failed fusion between the septum primum and endocardial cushions
- C. Failed obliteration of the vascular connection between the pulmonary artery and aorta
- ✓ D. Insufficient development of the interventricular septum
- E. Malalignment of the infundibular septum

Incorrect
Correct answer
D

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Explanation

This patient's harsh, holosystolic murmur is characteristic of ventricular septal defect (VSD), occurring due to **inadequate development** of the **interventricular septum**. This septum is composed of muscular and membranous portions; failed fusion of any of its derivative parts results in VSD. The associated murmur **may not be audible immediately** after birth due to high pulmonary vascular resistance (PVR) that limits left-to-right shunting through the VSD; it typically develops over the ensuing days as PVR declines (as in this case).

- **Small VSDs** are typically asymptomatic due to the small volume of shunted blood, but turbulent flow through

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Explanation

This patient's harsh, holosystolic murmur is characteristic of ventricular septal defect (VSD), occurring due to **inadequate development** of the **interventricular septum**. This septum is composed of muscular and membranous portions; failed fusion of any of its derivative parts results in VSD. The associated murmur **may not be audible immediately** after birth due to high pulmonary vascular resistance (PVR) that limits left-to-right shunting through the VSD; it typically develops over the ensuing days as PVR declines (as in this case).

- **Small VSDs** are typically asymptomatic due to the small volume of shunted blood, but turbulent flow through this narrow connection causes a **harsh, holosystolic murmur** at the left-mid to left-lower sternal border. Unlike larger defects, small VSDs usually undergo spontaneous closure.
- **Moderate to large VSDs** lead to pulmonary overcirculation and left-sided volume overload that can cause **heart failure** (eg, diaphoresis, failure to thrive); larger shunts increase the risk. Because of relatively unrestricted flow compared to smaller defects, auscultation typically demonstrates a **softer or no murmur**. Chronic left-to-right shunting can also eventually lead to pulmonary hypertension with shunt reversal and resulting cyanosis (Eisenmenger syndrome).

(Choices A and B) Atrial septal defect (ASD) can occur due to failed fusion of the septum primum and endocardial cushions (primum ASD) or to arrested growth of the septum secundum (secundum ASD); it permits left-to-right shunting. The characteristic systolic ejection murmur best heard over the second intercostal space is caused by right-sided volume overload and increased flow across the pulmonic valve.

(Choice C) Patent ductus arteriosus (PDA) occurs due to failed obliteration of the vascular connection between the pulmonary artery and aorta and allows left-to-right shunting. Like VSD, a PDA murmur typically increases in intensity as PVR declines following birth; the *continuous* murmur is best heard at the left *upper* sternal border.

(Choice E) Malalignment of the infundibular septum can cause varying degrees of right ventricular outflow tract (RVOT) obstruction, as in tetralogy of Fallot. Patients typically have a large VSD and a murmur created by flow through the partially obstructed RVOT (crescendo-decrescendo murmur best heard along the left mid to upper sternal border).

Educational objective:

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shunting through the VSD, it typically develops over the ensuing days as PVR declines (as in this case).

- **Small VSDs** are typically asymptomatic due to the small volume of shunted blood, but turbulent flow through this narrow connection causes a **harsh, holosystolic murmur** at the left-mid to left-lower sternal border. Unlike larger defects, small VSDs usually undergo spontaneous closure.
- **Moderate to large VSDs** lead to pulmonary overcirculation and left-sided volume overload that can cause **heart failure** (eg, diaphoresis, failure to thrive); larger shunts increase the risk. Because of relatively unrestricted flow compared to smaller defects, auscultation typically demonstrates a **softer or no murmur**. Chronic left-to-right shunting can also eventually lead to pulmonary hypertension with shunt reversal and resulting cyanosis (Eisenmenger syndrome).

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Educational objective:
Ventricular septal defect occurs due to insufficient development of the interventricular septum and commonly presents with a holosystolic murmur at the left lower sternal border as pulmonary vascular resistance declines in the neonatal period. Presentation varies from asymptomatic murmur (small defect) to heart failure (large defect).

References

- Recent advances in managing septal defects: ventricular septal defects and atrioventricular septal defects.

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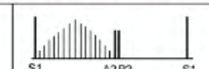
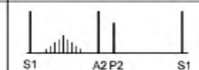
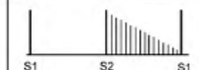
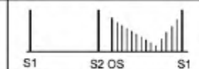
Cardiac murmurs

Systolic	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid–left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation*		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; **RUSB** = right upper sternal border.

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(Choice E) Malalignment of the infundibular septum can cause varying degrees of right ventricular outflow tract

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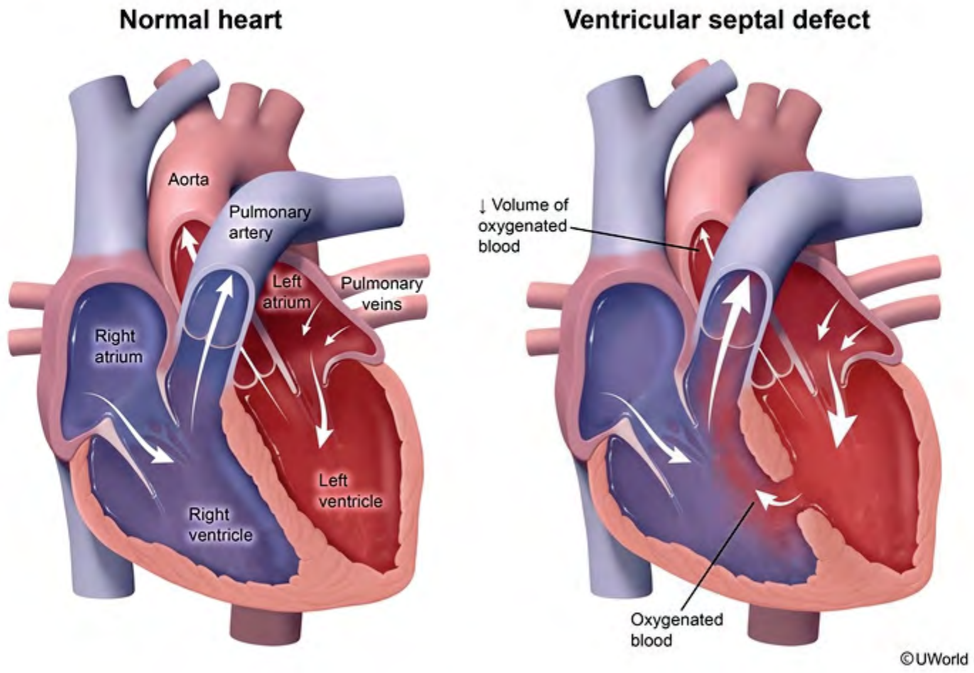

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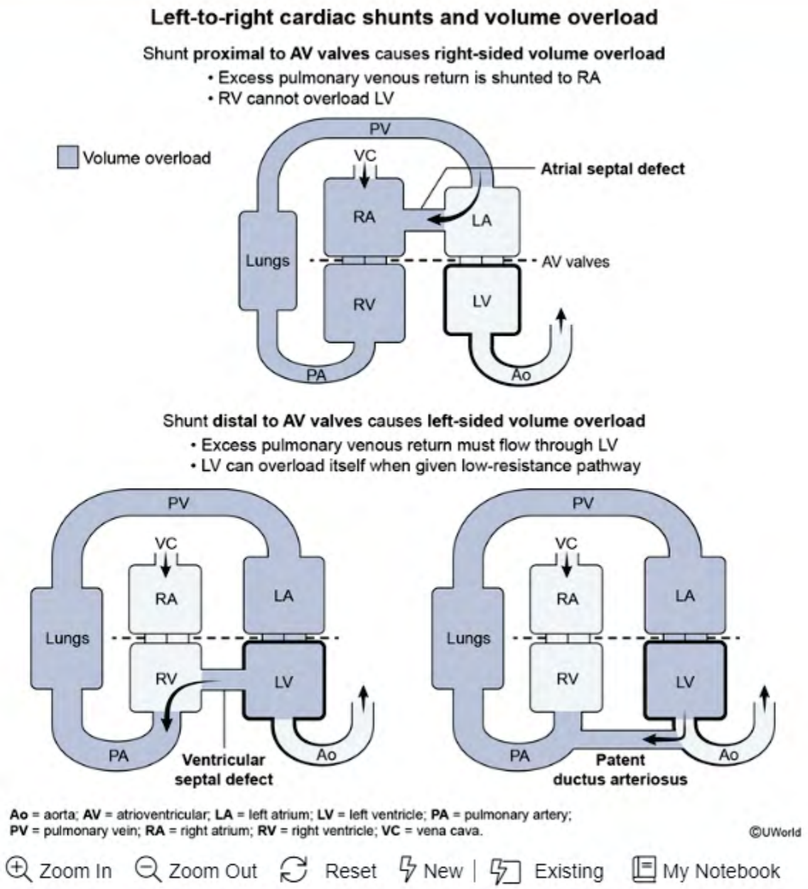

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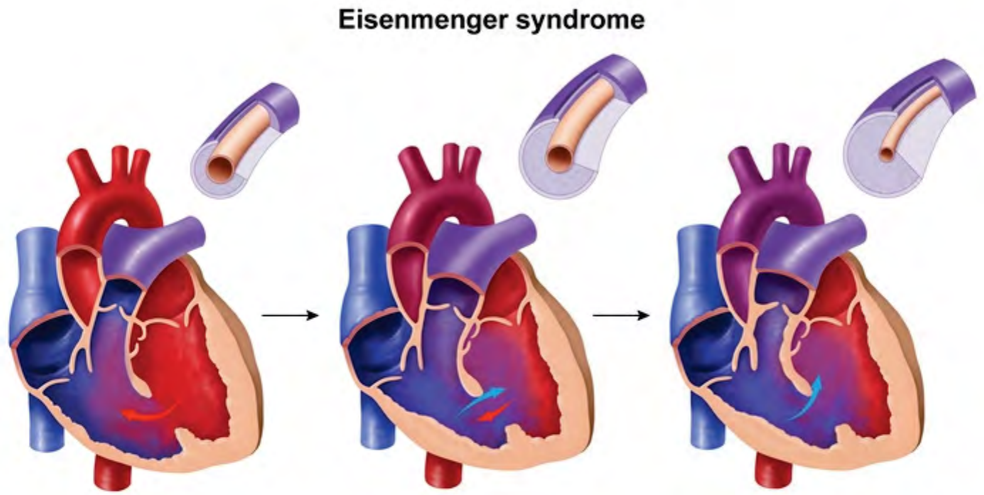

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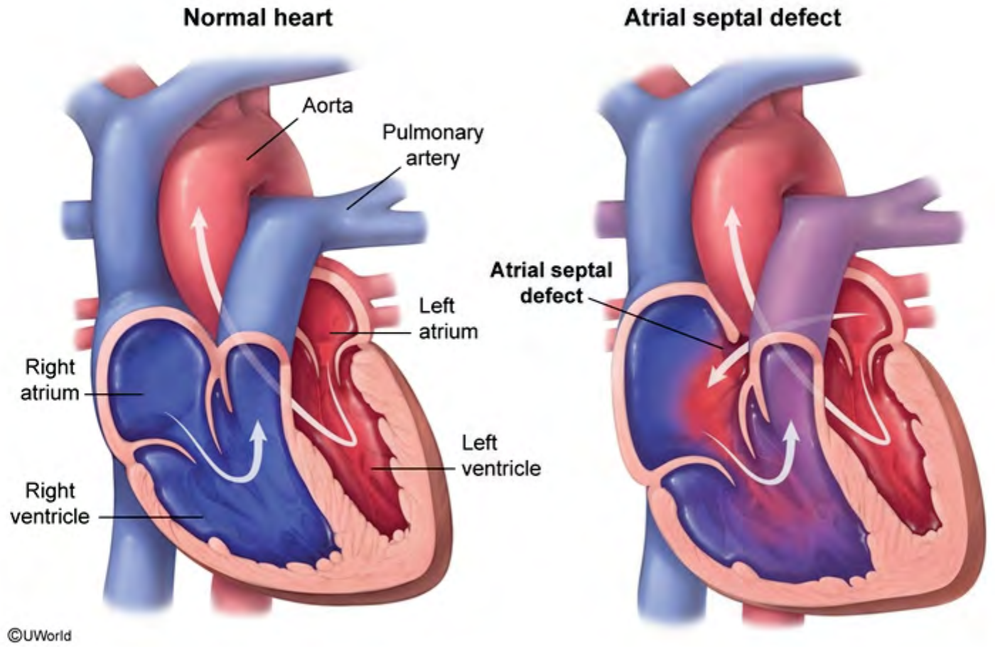

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Educational objective:

Ventricular septal defect occurs due to insufficient development of the interventricular septum and commonly presents with a holosystolic murmur at the left lower sternal border as pulmonary vascular resistance declines in the neonatal period. Presentation varies from asymptomatic murmur (small defect) to heart failure (large defect)

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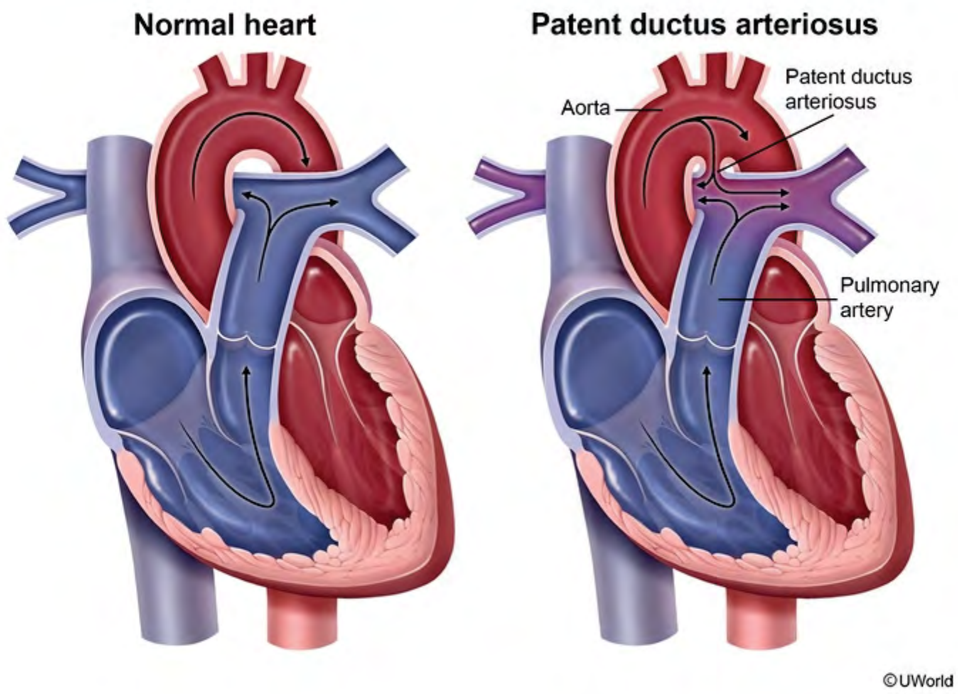

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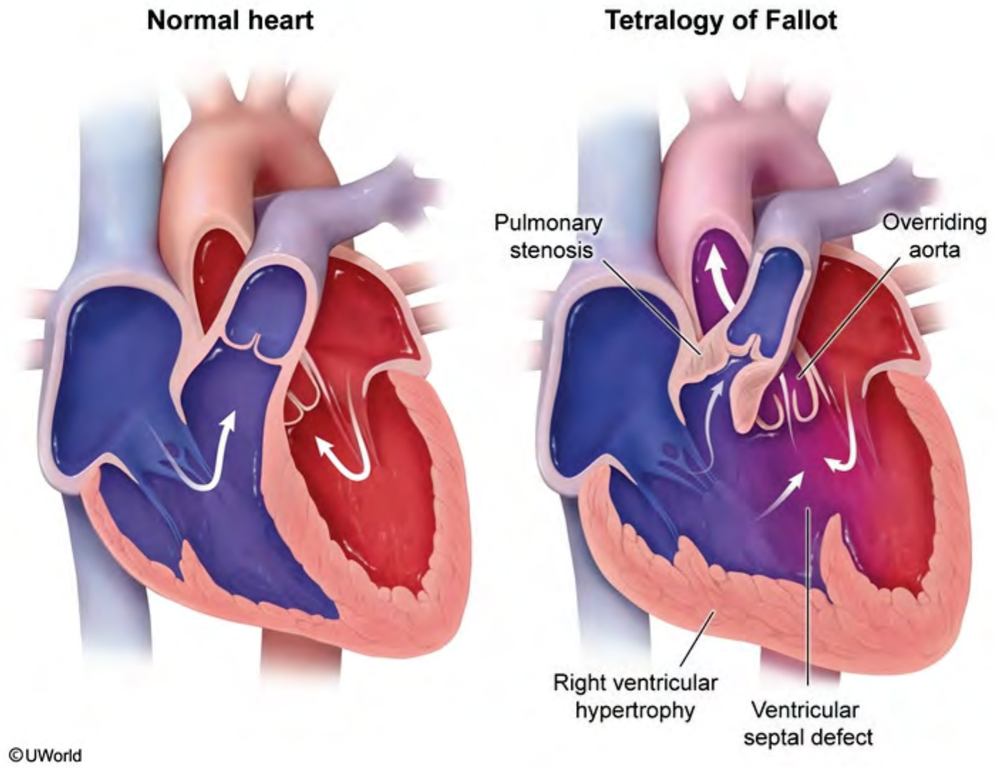

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A 65-year-old woman is brought to the hospital due to sudden-onset chest heaviness and shortness of breath that began an hour ago. Her medical history is significant for type 2 diabetes mellitus, obesity, and hypertension. Temperature is 36.7 C (98.1 F), blood pressure is 80/50 mm Hg, pulse is 110/min, and respirations are 20/min. Extremities are cold and clammy. Diaphoresis is present. ECG shows ST-segment elevation in the lateral leads. Which of the following hemodynamic changes are most likely present in this patient?

	Cardiac contractility	Systemic vascular resistance	Pulmonary artery systolic pressure
○ A.	Decreased	Decreased	Decreased
○ B.	Decreased	Increased	Decreased
○ C.	Decreased	Increased	Increased
○ D.	Increased	Decreased	Decreased
○ E.	Increased	Increased	Increased

Submit

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		Cardiac contractility	Systemic vascular resistance	Pulmonary artery systolic pressure	
✗	A.	Decreased	Decreased	Decreased	(10%)
	B.	Decreased	Increased	Decreased	(28%)
✓	C.	Decreased	Increased	Increased	(51%)
	D.	Increased	Decreased	Decreased	(5%)
	E.	Increased	Increased	Increased	(4%)

Incorrect
Correct answer
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51%
Answered correctly

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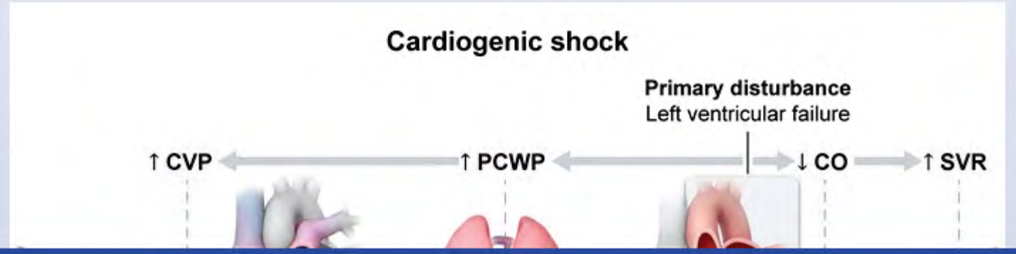
Explanation



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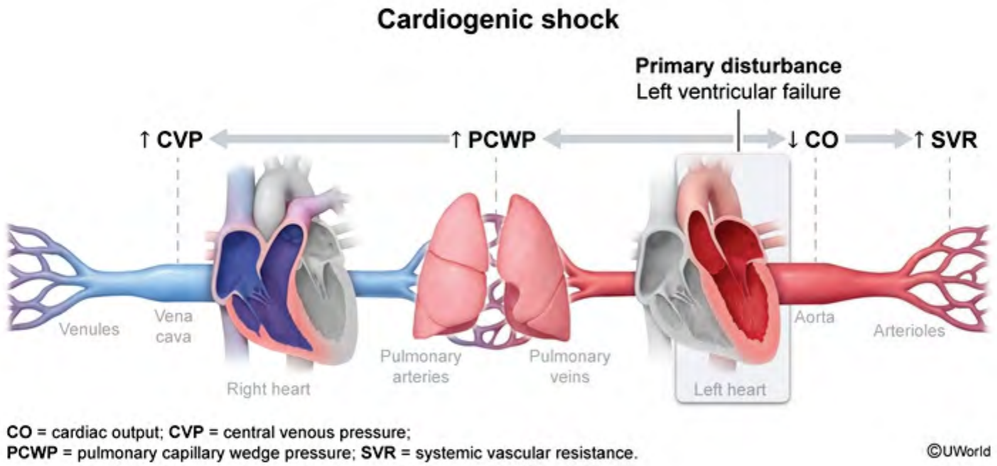

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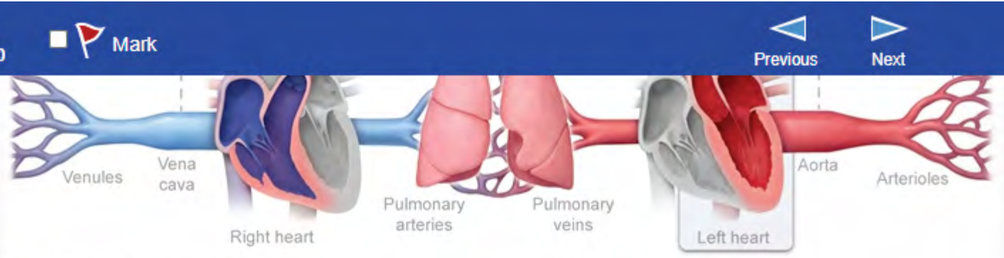

CO = cardiac output; **CVP** = central venous pressure;
PCWP = pulmonary capillary wedge pressure; **SVR** = systemic vascular resistance.

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This patient with acute-onset chest heaviness and shortness of breath and ECG showing ST-segment elevation has a **myocardial infarction** (MI). Localization to the lateral leads is consistent with involvement of the left ventricle. Hypotension and cold and clammy extremities (evidence of poor tissue perfusion) suggest **cardiogenic shock** due to MI-induced **acute left ventricular (LV) failure**.

A large MI can cause profoundly **decreased cardiac contractility**. The resulting LV systolic failure leads to markedly reduced cardiac output and **hypotension**. In response to the hypotension, the aortic and carotid baroreceptors stimulate peripheral vasoconstriction to **increase systemic vascular resistance** in an effort to maintain blood pressure. Heart rate is also increased (ie, tachycardia) to help increase cardiac output.

Failure to eject blood from the left ventricle also increases LV end-diastolic pressure, which is transmitted backward to **increase pulmonary capillary wedge pressure** (an estimate of left atrial pressure) and cause pulmonary edema. This increase in pulmonary venous pressure also necessitates **higher pulmonary arterial systolic pressure** to maintain forward blood movement, which may precipitate acute right ventricular failure.

Educational objective:
Myocardial infarction may cause acute left ventricular (LV) failure and cardiogenic shock. Decreased cardiac contractility leads to reduced cardiac output and hypotension. In response, systemic vascular resistance is increased (via peripheral vasoconstriction) to maintain blood pressure. The LV systolic failure increases LV end-diastolic pressure, which is transmitted backward to increase pulmonary capillary wedge pressure and pulmonary arterial pressure.

Physiology	Cardiovascular System	Cardiogenic shock
Subject	System	Topic

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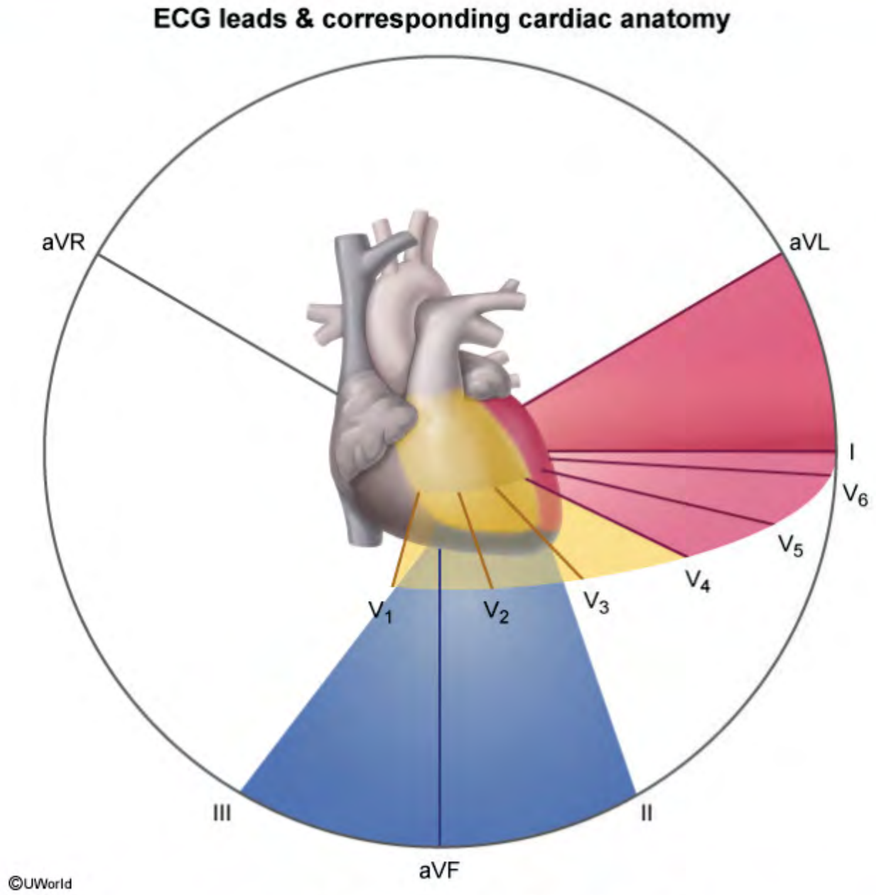

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A 60-year-old man comes to the office with a 6-month history of exertional chest pain that remits with rest. His other medical conditions include hypertension, diabetes mellitus, and hypercholesterolemia. An exercise stress test is positive for inducible ischemia. Cardiac catheterization shows 80% occlusion of the right coronary artery and 60% occlusion of the left coronary artery. The first step in the pathogenesis of this patient's coronary artery disease most likely involved which of the following cell types?

- A. Endothelial cells
- B. Interstitial fibroblasts
- C. Macrophages
- D. Mast cells
- E. Pericytes
- F. Platelets
- G. Smooth muscle cells

Submit

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- ✓ A. Endothelial cells (71%)
- B. Interstitial fibroblasts (1%)
- ✗ C. Macrophages (18%)
- D. Mast cells (0%)
- E. Pericytes (0%)
- F. Platelets (3%)
- G. Smooth muscle cells (4%)

Incorrect
Correct answer
A

71%
Answered correctly

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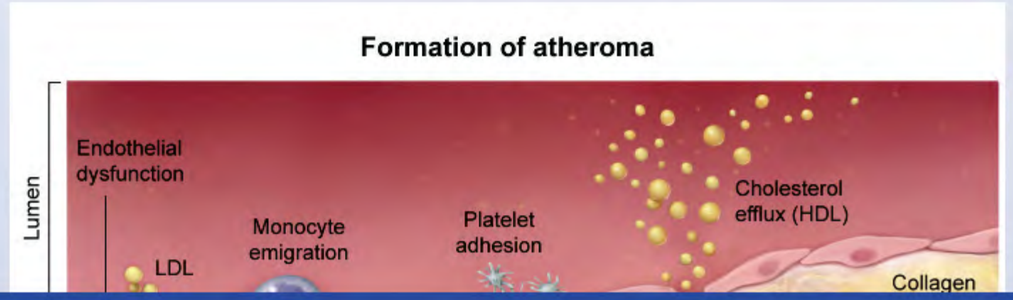
Explanation



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A 60-year-old man comes to the office with a 6-month history of exertional chest pain that remits with rest. His other r
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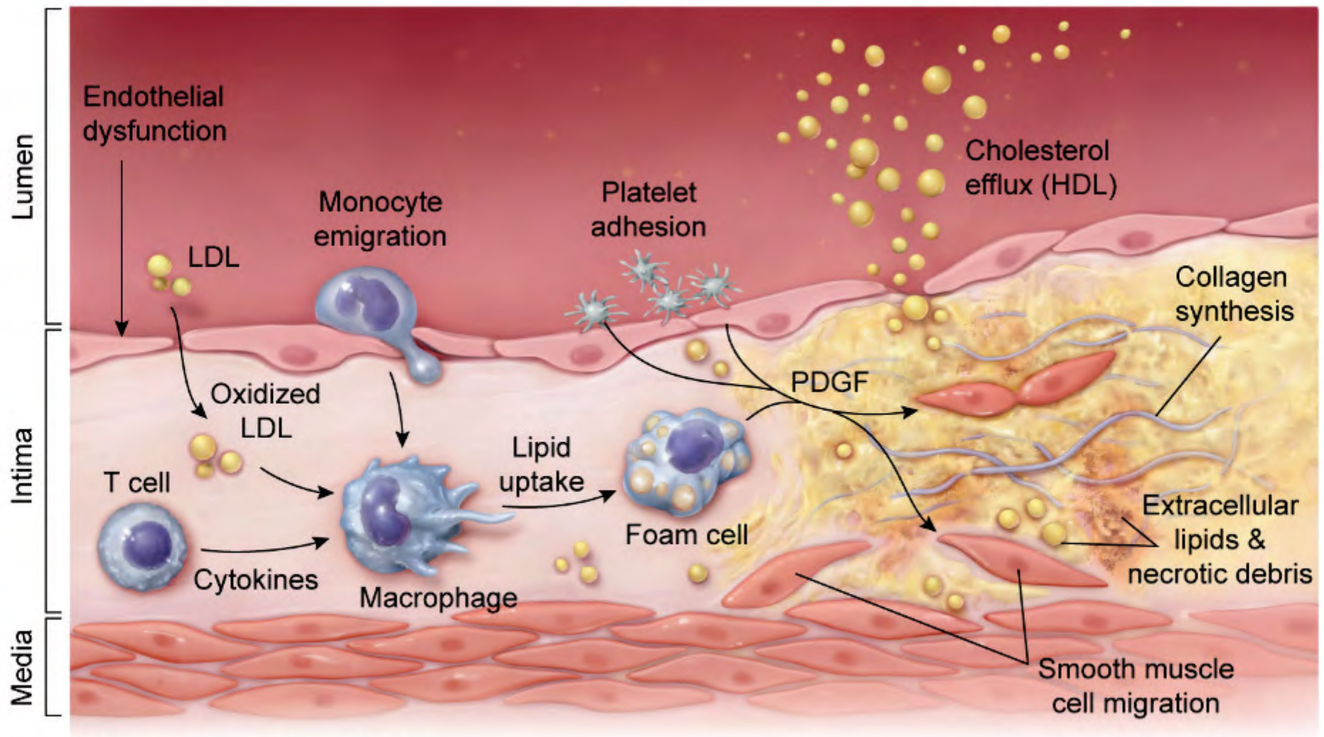
Formation of atheroma



PDGF = platelet-derived growth factor.

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PDGF = platelet-derived growth factor.

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This patient's presentation is consistent with coronary artery atherosclerosis. The pathogenesis of atherosclerosis likely begins with **endothelial cell injury**. Chronic endothelial cell injury may result from hypertension (and related hemodynamic factors), hyperlipidemia, smoking, and diabetes mellitus. Such injury leads to endothelial cell dysfunction and/or exposure of subendothelial collagen (endothelial cell denudation).

Endothelial cell dysfunction results in monocyte and lymphocyte adhesion and migration into the intima **(Choice C)**, while exposure of subendothelial collagen promotes platelet adhesion **(Choice F)**. Growth factors produced by monocytes and platelets stimulate medial smooth muscle cell (SMC) migration into the intima **(Choice G)**. At the same time, increased vascular permeability allows LDL cholesterol into the intima, where it is phagocytosed by the accumulating macrophages and SMCs to produce lipid-laden foam cells (**fatty streak**).

The continued release of cytokines and growth factors results in a chronic inflammatory state within the underlying intima. This promotes further deposition of LDL cholesterol within the intima and stimulates SMC proliferation with increased production of collagen and proteoglycans. Necrosis of foam cells results in release of toxic oxidized LDL into the extracellular matrix, perpetuating a cycle of injury. The lesion eventually organizes into a core of lipid debris surrounded by monocytes and lymphocytes covered by a fibrous cap with intermixed SMCs (**fibrofatty atheroma**).

(Choice B) Fibroblasts do not contribute significantly to atheroma formation. The fibrous tissue, including the fibrous cap, of atheromas is synthesized by SMCs that have migrated to the intimal layer in which the plaque forms.

(Choice D) Mast cells are not significantly involved in the pathogenesis of atherosclerosis.

(Choice E) Pericytes are pluripotent cells that surround the smallest blood vessels (especially postcapillary venules). Because atherosclerosis affects the large elastic arteries and large- and medium-sized muscular arteries, these cells do not play a significant role.

Educational objective:
Atherosclerosis is initiated by repetitive endothelial cell injury, which leads to a chronic inflammatory state in the underlying intima of large elastic arteries as well as in large- and medium-sized muscular arteries.

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hemodynamic factors), hyperlipidemia, smoking, and diabetes mellitus. Such injury leads to endothelial cell dysfunction and/or exposure of subendothelial collagen (endothelial cell denudation).

Endothelial cell dysfunction results in monocyte and lymphocyte adhesion and migration into the intima **(Choice C)**, while exposure of subendothelial collagen promotes platelet adhesion **(Choice F)**. Growth factors produced by monocytes and platelets stimulate medial smooth muscle cell (SMC) migration into the intima **(Choice G)**. At the same time, increased vascular permeability allows LDL cholesterol into the intima, where it is phagocytosed by the accumulating macrophages and SMCs to produce lipid-laden foam cells (**fatty streak**).

The continued release of cytokines and growth factors results in a chronic inflammatory state within the underlying intima. This promotes further deposition of LDL cholesterol within the intima and stimulates SMC proliferation with increased production of collagen and proteoglycans. Necrosis of foam cells results in release of toxic oxidized LDL into the extracellular matrix, perpetuating a cycle of injury. The lesion eventually organizes into a core of lipid debris surrounded by monocytes and lymphocytes covered by a fibrous cap with intermixed SMCs (**fibrofatty atheroma**).

(Choice B) Fibroblasts do not contribute significantly to atheroma formation. The fibrous tissue, including the fibrous cap, of atheromas is synthesized by SMCs that have migrated to the intimal layer in which the plaque forms.

(Choice D) Mast cells are not significantly involved in the pathogenesis of atherosclerosis.

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Educational objective:
Atherosclerosis is initiated by repetitive endothelial cell injury, which leads to a chronic inflammatory state in the underlying intima of large elastic arteries as well as in large- and medium-sized muscular arteries.

Pathology	Cardiovascular System	Atherosclerosis
Subject	System	Topic

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A 63-year-old man comes to the emergency department due to progressive dyspnea and fatigue. Over the last week, he has not been able to lie flat due to difficulty breathing and has had to sleep in a sitting position. He had an anterior myocardial infarction 6 months ago and has not been compliant with his medication regimen since that time. Medical history is also significant for hypertension. The patient is an ex-smoker with a 30-pack-year history. He does not drink alcohol. His father died of a heart attack at age 60. Blood pressure is 170/100 mm Hg, and pulse is 100/min and regular. Oxygen saturation is 90% on room air. He is afebrile. Auscultation reveals crackles at the lung bases, an S3 gallop, and a II/VI holosystolic murmur over the apex. The patient is admitted to the hospital, and after treatment with diuretics and vasodilators his condition improves significantly. Three days later there are no appreciable gallops or murmurs on cardiac examination. Which of the following best explains the murmur heard at the time of the initial examination?

- A. Heavily calcified mitral annulus
- B. Increased flow velocity through the aortic valve
- C. Ruptured chordae tendineae
- D. Secondary mitral regurgitation
- E. Thickened and deformed mitral valve cusps

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A 63-year-old man comes to the emergency department due to progressive dyspnea and fatigue. Over the last week, he has not been able to lie flat due to difficulty breathing and has had to sleep in a sitting position. He had an anterior myocardial infarction 6 months ago and has not been compliant with his medication regimen since that time. Medical history is also significant for hypertension. The patient is an ex-smoker with a 30-pack-year history. He does not drink alcohol. His father died of a heart attack at age 60. Blood pressure is 170/100 mm Hg, and pulse is 100/min and regular. Oxygen saturation is 90% on room air. He is afebrile. Auscultation reveals crackles at the lung bases, an S3 gallop, and a II/VI holosystolic murmur over the apex. The patient is admitted to the hospital, and after treatment with diuretics and vasodilators his condition improves significantly. Three days later there are no appreciable gallops or murmurs on cardiac examination. Which of the following best explains the murmur heard at the time of the initial examination?

- A. Heavily calcified mitral annulus (1%)
- ✗ B. Increased flow velocity through the aortic valve (12%)
- C. Ruptured chordae tendineae (7%)
- ✓ D. Secondary mitral regurgitation (75%)
- E. Thickened and deformed mitral valve cusps (3%)

Incorrect
Correct answer
D

75%
Answered correctly

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Explanation

Normal	Functional mitral valve regurgitation

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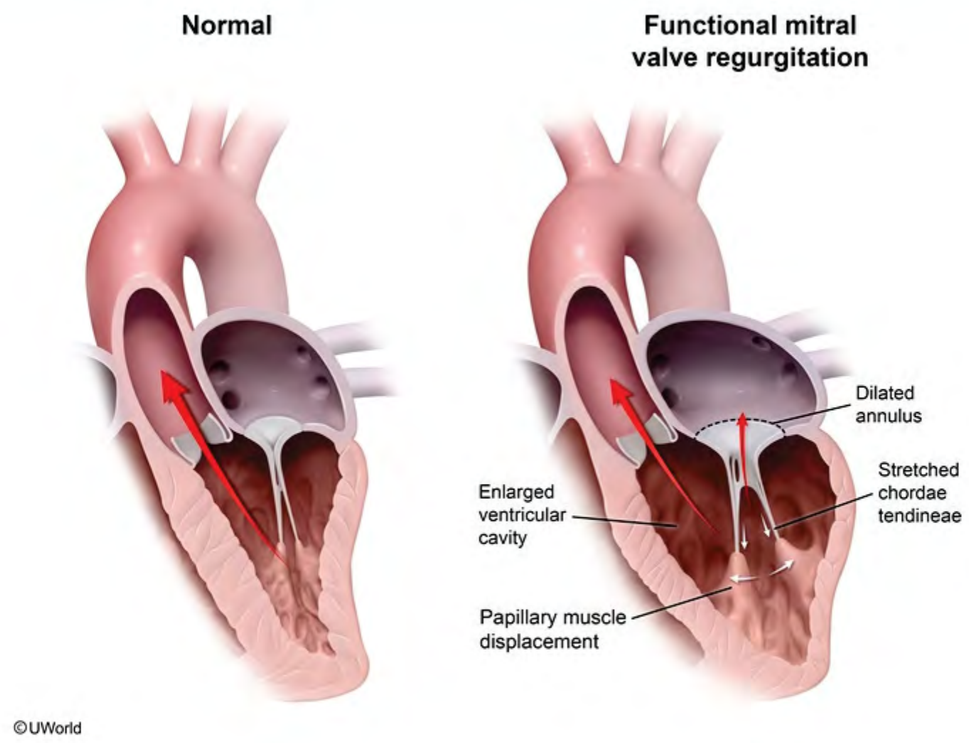

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valve regurgitation

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This patient's presentation with fatigue, progressive dyspnea, orthopnea, pulmonary crackles, and an S3 heart sound is consistent with **decompensated heart failure**. Appropriate treatment with diuretics and vasodilators resulted in symptomatic improvement and disappearance of the apical holosystolic murmur, which was most likely due to **secondary (functional) mitral valve regurgitation** (MR).

Whereas primary MR is caused by an intrinsic defect of the mitral valve apparatus (eg, cleft in a valve cusp, myxomatous degeneration of the chordae tendineae), secondary MR occurs due to other factors. Decompensated heart failure is a common cause of secondary MR because it leads to an increase in left ventricular end-diastolic volume (LVEDV), or preload, with **dilation of the mitral valve annulus** (the tissue on which the mitral valve cusps are mounted) and taut stretching of the chordae tendineae. The dilated annulus and **restricted movement of the chordae tendineae** can cause insufficient closure of an intrinsically normal mitral valve, resulting in MR. Systemic hypertension can also contribute to secondary MR by favoring relatively lower-resistance regurgitant flow.

Treatment with diuretics to reduce LVEDV and vasodilators to reduce blood pressure can lead to resolution of heart failure-induced MR.

(Choice A) Calcification of the mitral valve annulus may occur with aging, and can be accelerated by hypertension or advanced kidney disease. It is usually an incidental finding that does not affect mitral valve function.

(Choice B) Increased flow velocity through the aortic valve occurs with aortic valve stenosis. The resulting murmur is crescendo-decrescendo and best heard at the right second intercostal space (rather than holosystolic and best heard at the apex).

(Choice C) Chordae tendineae rupture is a potential cause of primary MR that can occur in the setting of bacterial endocarditis, connective tissue disease (eg, Marfan syndrome), or acute myocardial infarction. The resulting murmur does not resolve with diuretics and vasodilators; surgical repair is typically required.

(Choice E) Thickened and deformed mitral valve cusps can occur in rheumatic heart disease and are a potential cause of primary MR. Such a murmur does not resolve with treatment of decompensated heart failure. In addition,

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(Choice E) Thickened and deformed mitral valve cusps can occur in rheumatic heart disease and are a potential cause of primary MR. Such a murmur does not resolve with treatment of decompensated heart failure. In addition, mitral stenosis (evidenced by an opening snap and mid-diastolic rumble) is more likely than MR to occur with rheumatic heart disease.

Educational objective:
Decompensated heart failure is a common cause of secondary (functional) mitral valve regurgitation. Increased left ventricular end-diastolic volume causes dilation of the mitral valve annulus and restricted movement of the chordae tendineae with subsequent regurgitation. Treatment with diuretics and vasodilators can improve heart failure-induced MR.

References

- Functional mitral regurgitation.

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A 54-year-old man comes to the emergency department due to severe shortness of breath and palpitations. During initial assessment, pulse oximetry shows 83%. Diffuse, bilateral crackles are heard on lung auscultation. Chest x-ray is consistent with pulmonary edema. ECG shows atrial fibrillation with rapid ventricular response. The patient dies despite appropriate medical care. Autopsy is performed and gross examination of the heart is shown below.

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A 54-year-old man comes to the emergency department due to severe shortness of breath and palpitations.
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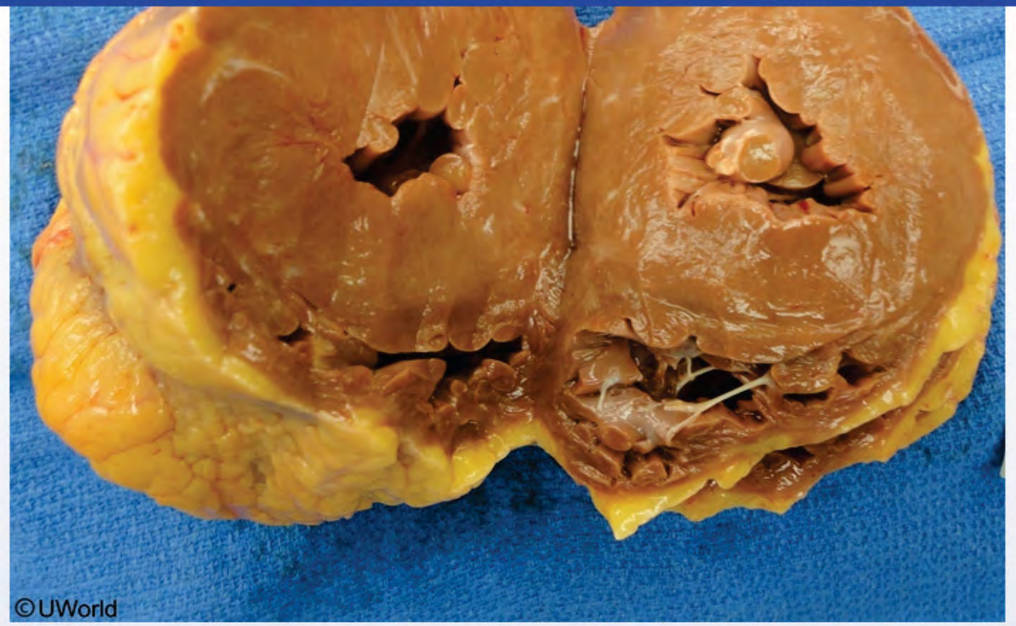

Histologic examination of the myocardial tissue reveals thick myocardial fibers with large, hyperchromatic nuclei as well as mild interstitial fibrosis. Which of the following is the most likely diagnosis?

- A. Cardiac amyloidosis
- B. Hypertensive heart disease
- C. Hypertrophic cardiomyopathy
- D. Mitral stenosis
- E. Pulmonary arterial hypertension

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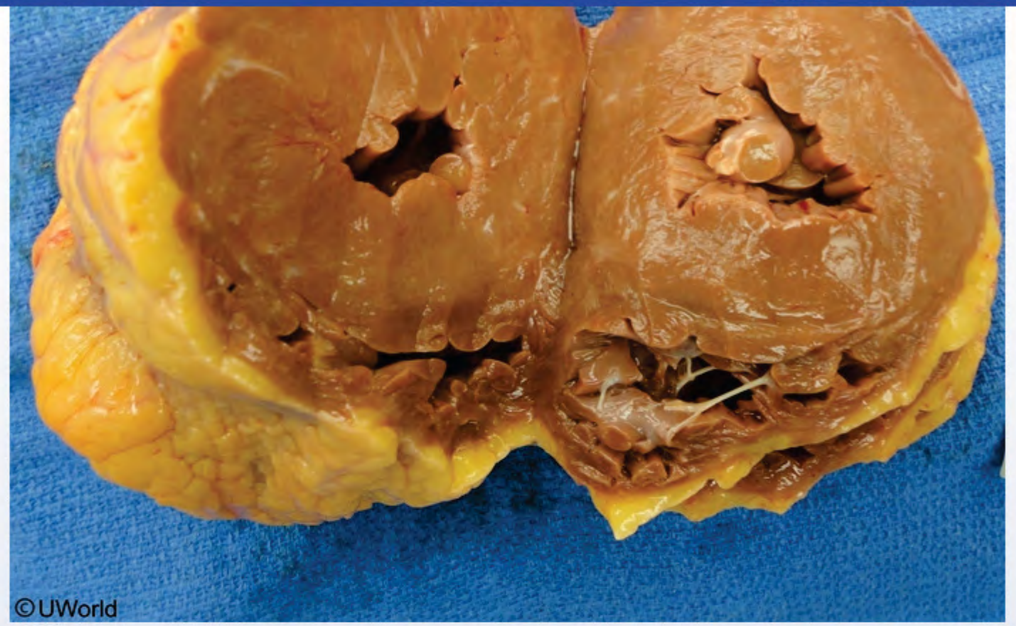

Histologic examination of the myocardial tissue reveals thick myocardial fibers with large, hyperchromatic nuclei as well as mild interstitial fibrosis. Which of the following is the most likely diagnosis?

- A. Cardiac amyloidosis (9%)
- ✓ B. Hypertensive heart disease (41%)
- ✗ C. Hypertrophic cardiomyopathy (42%)
- D. Mitral stenosis (2%)
- E. Pulmonary arterial hypertension (3%)

Incorrect
Correct answer
B

41%
Answered correctly

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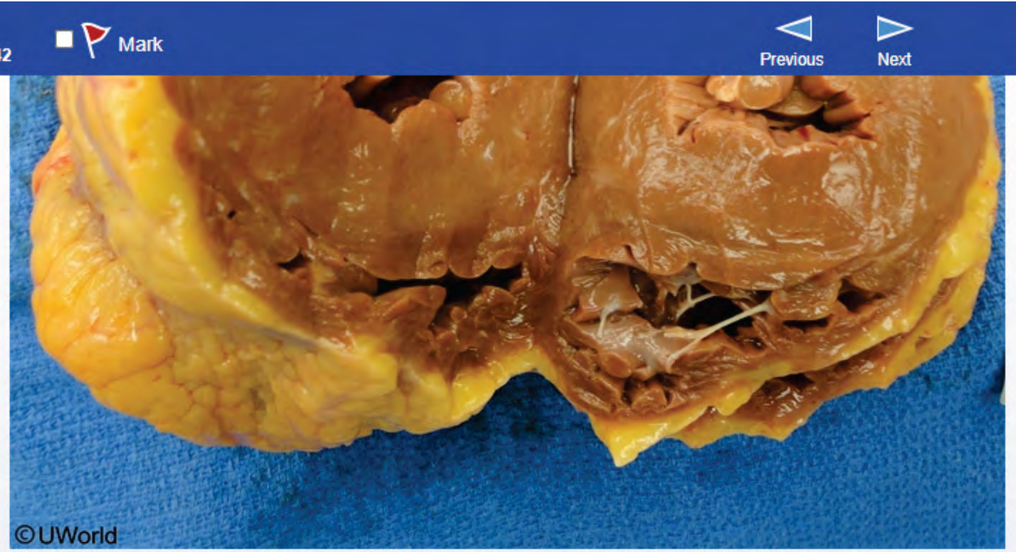

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Incorrect
Correct answer
B

41%
Answered correctly

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Explanation

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Concentric left ventricular hypertrophy

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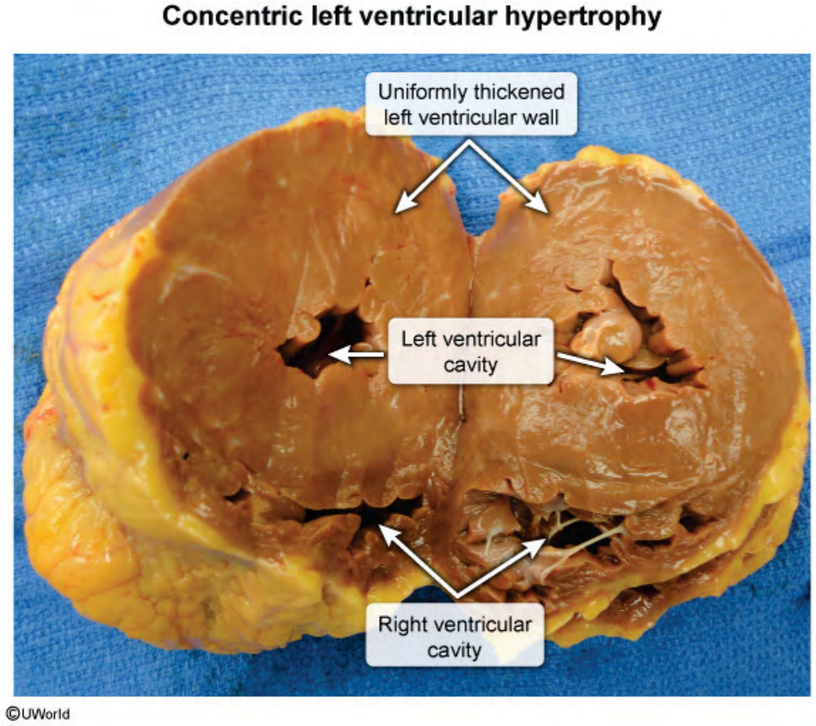

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back to the left atrium, causing atrial dilation and altered conduction that predisposes to **atrial fibrillation**. Loss of

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This individual developed acute decompensated heart failure (eg, shortness of breath, pulmonary edema) and likely died due to subsequent cardiogenic shock. Autopsy findings reveal **uniformly thickened** left ventricular (LV) walls and a small LV cavity, which are consistent with concentric LV hypertrophy.

Concentric LV hypertrophy most commonly results from prolonged **systemic hypertension** as myocardial fibers are added in parallel to increase contractile strength and handle the increased pressure load. As concentric hypertrophy worsens, it can manifest clinically as **hypertensive heart disease**, consisting of **impaired diastolic filling** and heart failure with preserved ejection fraction. The elevated diastolic filling pressures are transmitted back to the left atrium, causing atrial dilation and altered conduction that predisposes to **atrial fibrillation**. Loss of atrial contraction can lead to a dramatic **reduction in left ventricular filling** in patients with diastolic heart failure and can precipitate **acute decompensation** (as likely occurred in this patient).

On histopathology, hypertensive heart disease demonstrates transverse thickening of cardiomyocytes with prominent hyperchromatic nuclei. The increased myocardial mass may be inadequately supplied with oxygen, contributing to cellular necrosis and interstitial fibrosis.

(Choice A) Cardiac amyloidosis involves deposition of abnormal proteins in the myocardium and can lead to restrictive cardiomyopathy, which is characterized by impaired diastolic filling due to stiffening, rather than thickening, of the ventricular walls. Histopathology in cardiac amyloidosis reveals collections of pink amorphous material abutting normal cardiomyocytes.

(Choice C) Hypertrophic cardiomyopathy involves localized, rather than uniform, thickening of the LV walls, predominantly affecting the interventricular septum. As in hypertensive heart disease, histopathology can reveal cardiomyocyte thickening, hyperchromatic nuclei, and interstitial fibrosis; however, cardiomyocyte disarray (loss of organized cellular alignment) is also expected.

(Choice D) Mitral stenosis causes impaired diastolic filling and can lead to decompensated heart failure and atrial fibrillation; however, it does not increase the pressure load on the left ventricle and does not cause concentric hypertrophy.

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(Choice D) Mitral stenosis causes impaired diastolic filling and can lead to decompensated heart failure and atrial fibrillation; however, it does not increase the pressure load on the left ventricle and does not cause concentric hypertrophy.

(Choice E) Pulmonary arterial hypertension results from primary changes in the pulmonary arteries that increase pulmonary arterial resistance. The elevated pressure load on the right ventricle can lead to concentric right ventricular hypertrophy and right-sided heart failure. Pulmonary edema is not present in right-sided heart failure.

Educational objective:
Concentric left ventricular (LV) hypertrophy involves uniform thickening of the LV walls with reduction in LV cavity size and most commonly results from prolonged systemic hypertension. It can progress to hypertensive heart disease with impaired diastolic filling and heart failure with preserved ejection fraction. Histopathology demonstrates transverse thickening of cardiomyocytes with prominent hyperchromatic nuclei and interstitial fibrosis.

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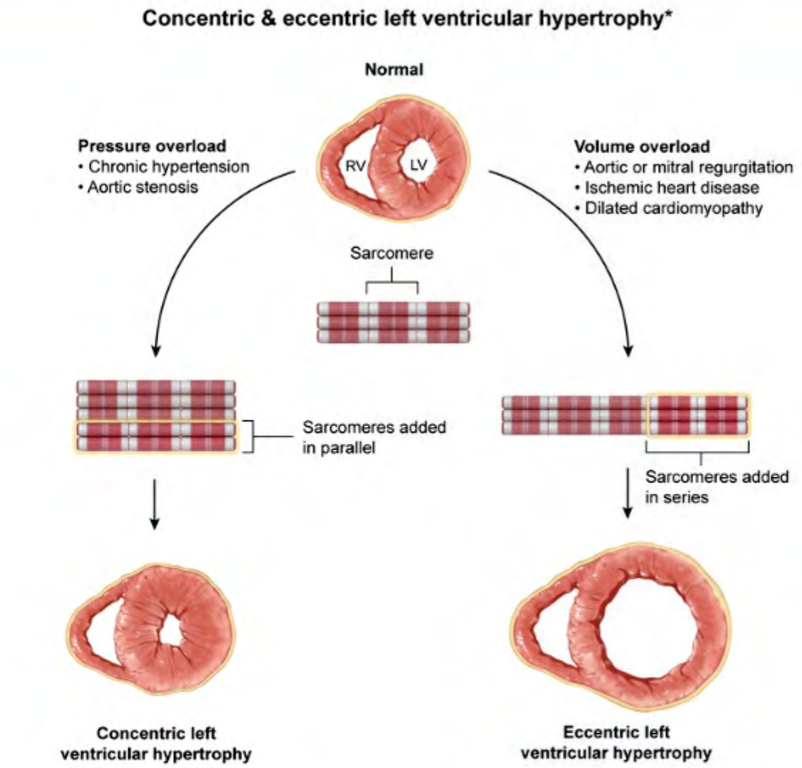

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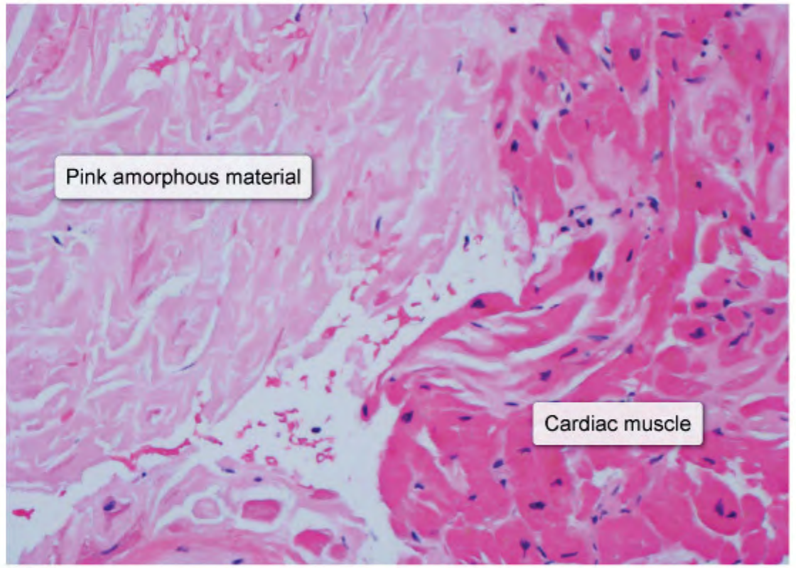

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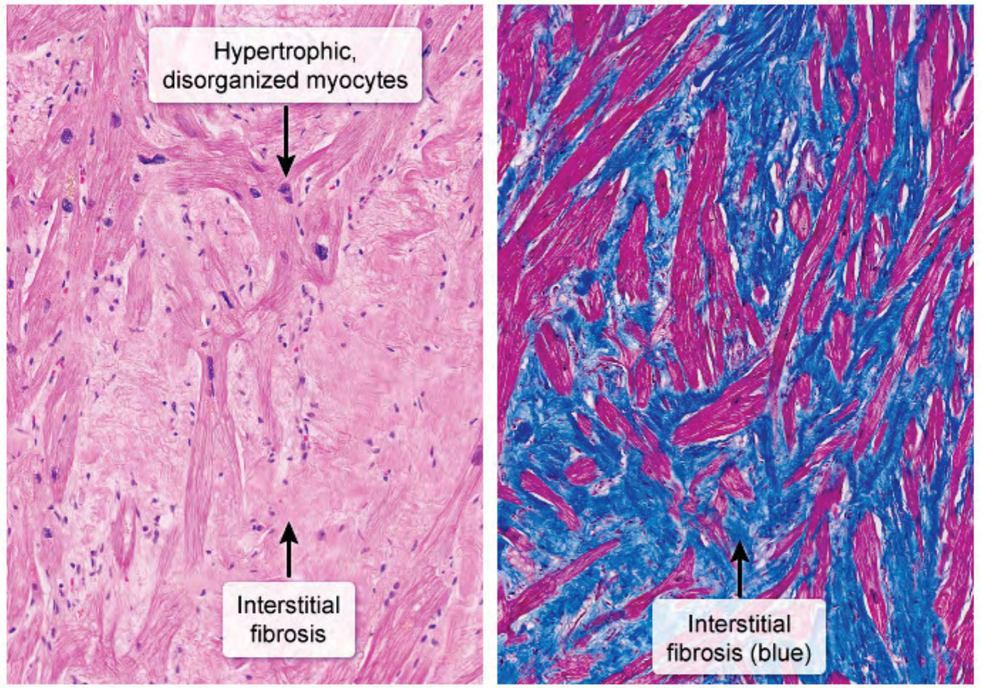
Hypertrophic cardiomyopathy



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Hematoxylin & eosin stain

Trichrome stain

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A 78-year-old woman is admitted to the hospital due to altered mental status. Medical history is significant for pyelonephritis, type 2 diabetes mellitus, coronary artery disease, and heart failure with reduced ejection fraction. Temperature is 36.2 C (97.2 F), blood pressure is 80/50 mm Hg, pulse is 120/min, and respirations are 24/min. Oxygen saturation is 90% on room air. Examination shows bilateral crackles. After she is started on appropriate treatment, including initiation of norepinephrine infusion, her blood pressure increases and her heart rate slightly decreases. This patient's change in heart rate is most likely due to norepinephrine's effect on which of the following receptors?

- A. Alpha-1 receptors in blood vessels
- B. Alpha-2 receptors in the medulla
- C. Beta-1 receptors in the heart
- D. Beta-2 receptors in blood vessels
- E. Dopamine receptors in blood vessels

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- ✓ A. Alpha-1 receptors in blood vessels (71%)
- ✗ B. Alpha-2 receptors in the medulla (7%)
- C. Beta-1 receptors in the heart (17%)
- D. Beta-2 receptors in blood vessels (3%)
- E. Dopamine receptors in blood vessels (0%)

Incorrect
Correct answer
A

71%
Answered correctly

03 secs
Time Spent

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Explanation

Cardiovascular effects of adrenergic agonists

- α_1: peripheral vasoconstriction, ↑ SVR
- β_1: ↑ heart rate & contractility, ↑ cardiac output
- β_2: peripheral vasodilation, ↓ SVR
- D_1: renal arteriolar vasodilation

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Cardiovascular effects of adrenergic agonists

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- β_1: ↑ heart rate & contractility, ↑ cardiac output
- β_2: peripheral vasodilation, ↓ SVR
- D_1: renal arteriolar vasodilation

Agonist	Receptor binding	Blood pressure	Heart rate
Dobutamine	$\beta_1 > \beta_2$	–/↓	↑
Dopamine	$D_1 > \beta_1 > \alpha_1$ (low-dose)	–/↓	↑
	$\alpha_1 > \beta_1 > D_1$ (high-dose)	↑	–/↓*
Epinephrine	$\beta_1 > \beta_2 > \alpha_1$ (low-dose)	–/↓	↑
	$\alpha_1 > \beta_1 > \beta_2$ (high-dose)	↑	–/↓*
Isoproterenol	$\beta_1 = \beta_2$	–/↓	↑
Norepinephrine	$\alpha_1 > \beta_1 > \beta_2$	↑	–/↓*
Phenylephrine	α_1	↑	↓*

*Reflex-mediated decrease.

SVR = systemic vascular resistance.

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Norepinephrine has both vasopressor and inotropic effects and is commonly used in patients with shock. It directly and **potently stimulates alpha-1 receptors** to trigger peripheral vasoconstriction, causing **increased systemic vascular resistance** (SVR) and increased blood pressure. Norepinephrine also directly stimulates beta-1 receptors to facilitate increased heart rate, myocardial contractility, and cardiac output **(Choice C)**.

In addition to the direct effects of norepinephrine, the alpha-1–mediated increase in SVR triggers indirect effects via the **baroreceptor reflex**. Increased blood pressure stimulates increased firing of the aortic and carotid baroreceptors. In response, the CNS increases parasympathetic output and decreases sympathetic output, decreasing heart rate and contractility. These reflexive effects on the heart **oppose the effects of direct beta-1–receptor stimulation**. Because of the marked increase in SVR with norepinephrine, the reflexive decrease in heart rate usually completely offsets or overrides the directly-mediated increase in heart rate, resulting in an **unchanged or slightly decreased heart rate**.

A similar offsetting effect occurs with high-dose dopamine and high-dose epinephrine due to baroreceptor-mediated feedback from predominant alpha-1 receptor stimulation.

(Choices B and E) Stimulation of alpha-2 receptors in the CNS (eg, with clonidine) decreases sympathetic output, causing peripheral vasodilation, decreased heart rate, and reduced blood pressure. Stimulation of dopamine-1 receptors (eg, with dopamine) causes renal arteriolar vasodilation and increases renal blood flow. Norepinephrine has no significant effect on either dopamine-1 or central alpha-2 receptors (does not cross blood brain barrier).

(Choice D) Although norepinephrine also causes mild, direct stimulation of vasodilatory beta-2 receptors, its peripheral vasoconstrictive effect (mediated by alpha-1 receptors) predominates, making it a very potent vasopressor.

Educational objective:
Norepinephrine potently stimulates alpha-1 receptors to cause peripheral vasoconstriction, increasing systemic vascular resistance and blood pressure. This triggers a baroreceptor–mediated reflexive decrease in heart rate that completely offsets or overrides the direct beta-1–mediated increase in heart rate caused by norepinephrine, resulting in overall unchanged or slightly decreased heart rate.

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1 receptors to facilitate increased heart rate, myocardial contractility, and cardiac output **(Choice C)**.

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Educational objective:
Norepinephrine potently stimulates alpha-1 receptors to cause peripheral vasoconstriction, increasing systemic vascular resistance and blood pressure. This triggers a baroreceptor–mediated reflexive decrease in heart rate that completely offsets or overrides the direct beta-1–mediated increase in heart rate caused by norepinephrine, resulting in overall unchanged or slightly decreased heart rate.

Pharmacology	Cardiovascular System	Sepsis
Subject	System	Topic

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Phenylephrine	α_1	↑	↓*

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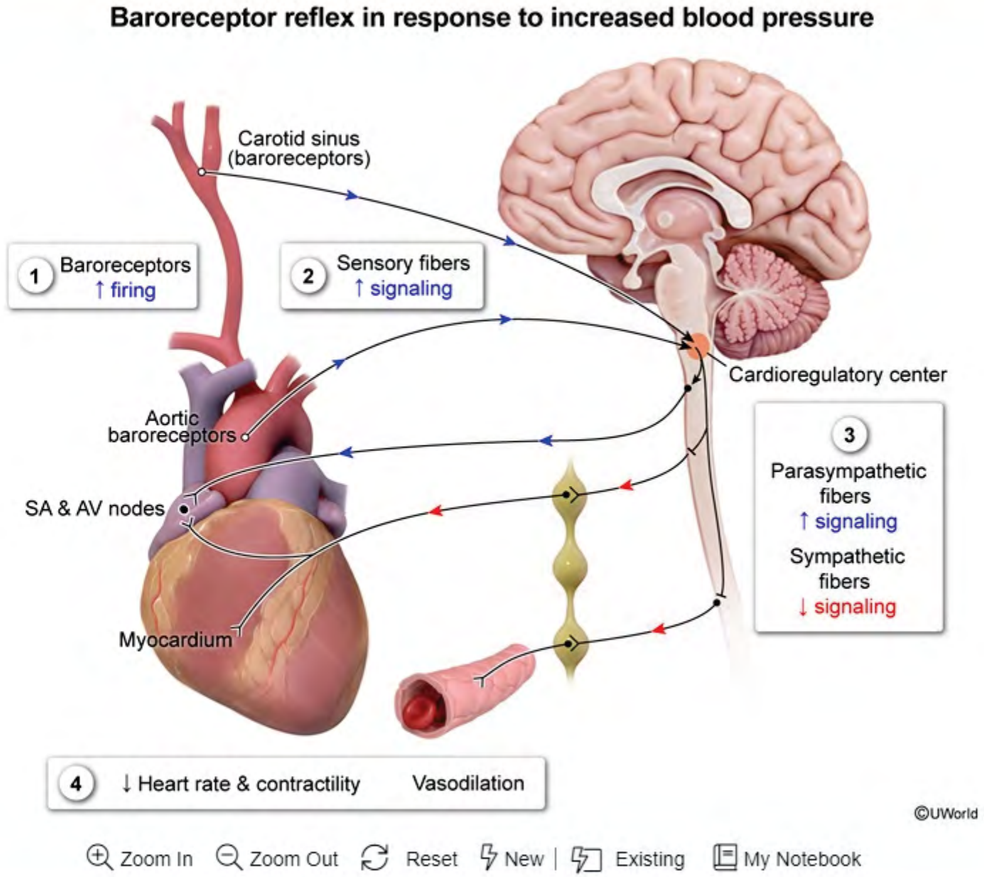

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Educational objective:

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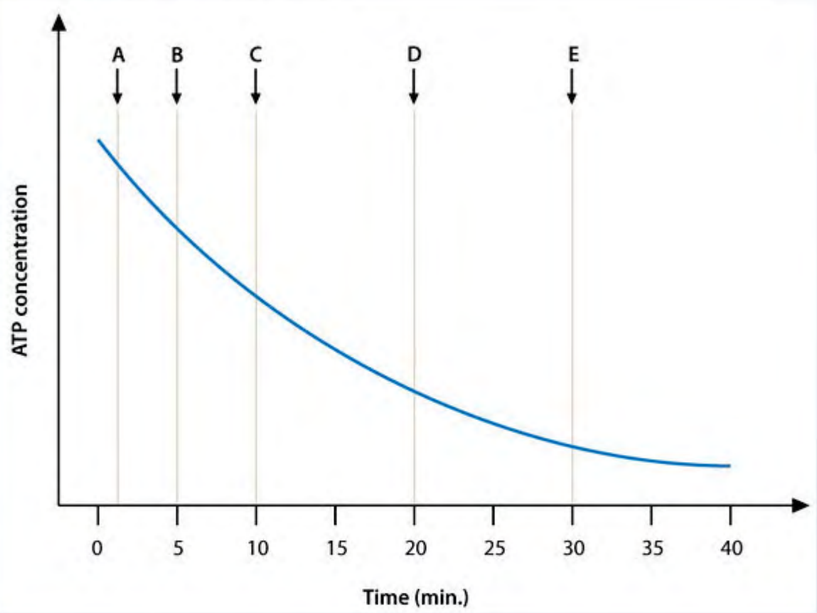
Cellular physiologists are investigating the mechanisms of contraction cessation in cardiomyocytes in response to ischemic injury. In an animal experiment, the left anterior descending coronary artery is ligated at time 0, and changes in ATP levels within the affected myocardial cells are recorded. At which point on the graph does the affected portion of the myocardium stop contracting?



- A. A
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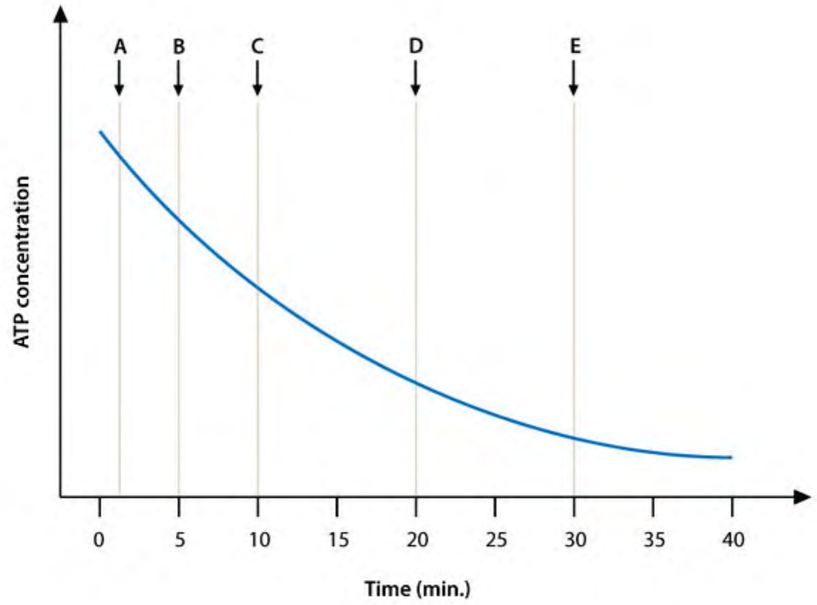

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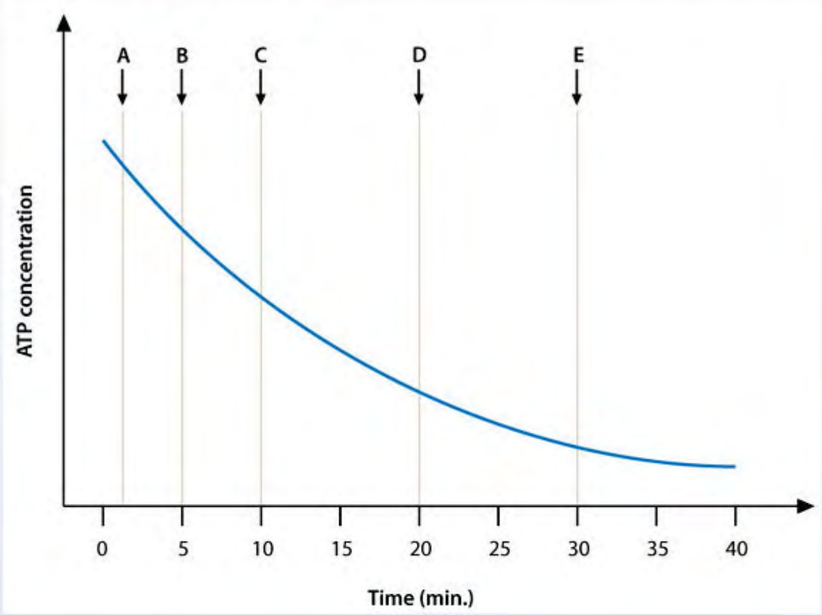

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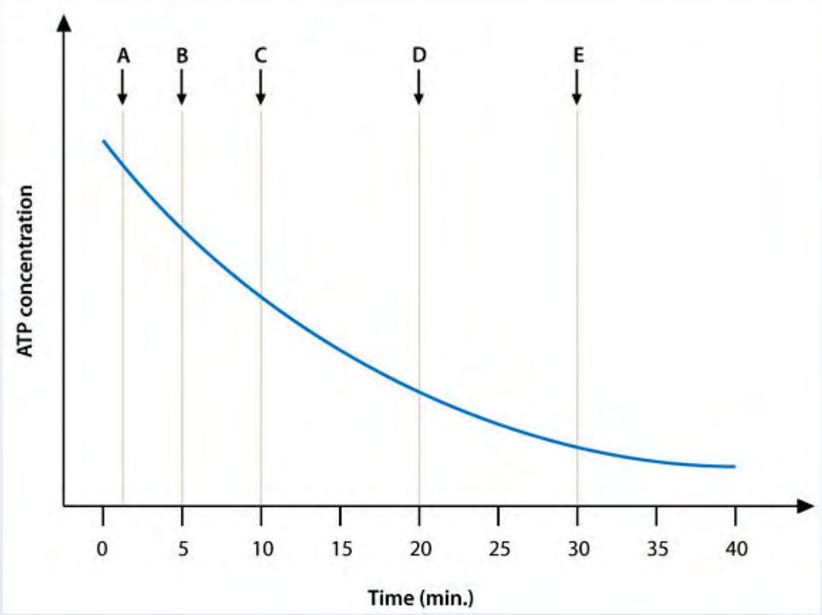

- ✓ ○ A. A (25%)
- ○ B. B (11%)
- ✗ ◉ C. C (13%)
- ○ D. D (25%)
- ○ E. E (24%)

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Correct answer
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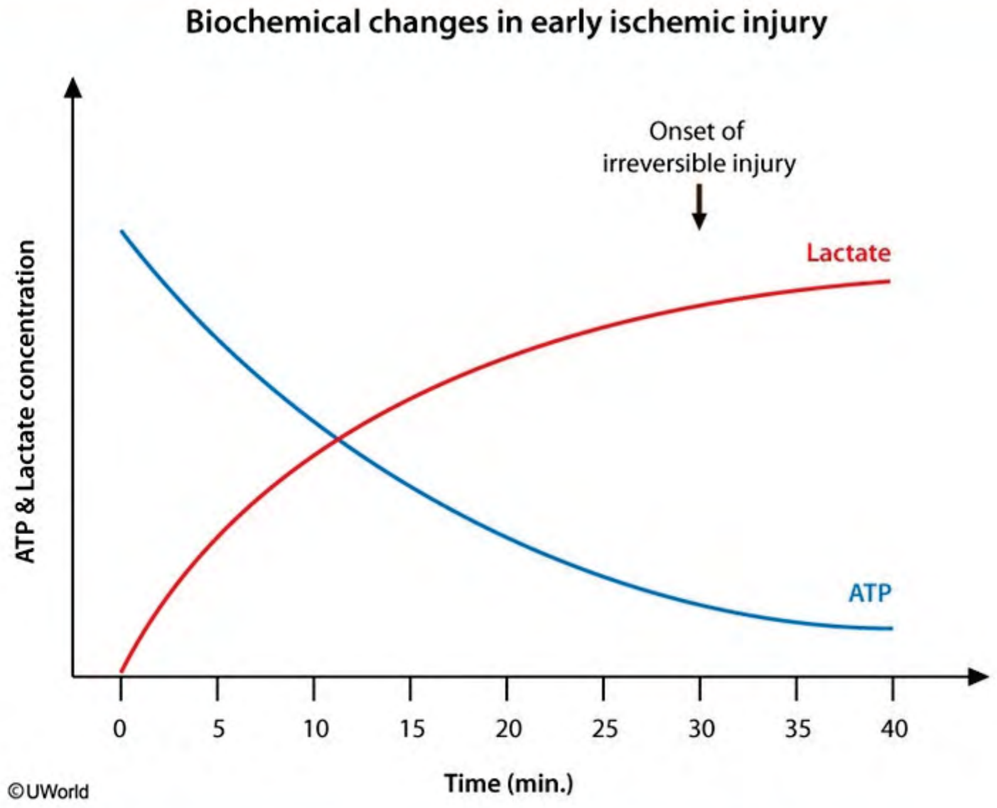

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including lactate. Although total ATP levels remain relatively normal during the first few minutes of ischemia, ATP is

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The major early biochemical consequence of total myocardial ischemia is cessation of aerobic glycolysis and initiation of anaerobic glycolysis. This transition occurs within seconds and results in inadequate production of high-energy phosphates (eg, ATP and creatine phosphate) and the accumulation of deleterious metabolites, including lactate. Although total ATP levels remain relatively normal during the first few minutes of ischemia, ATP is rapidly depleted from areas of the cell with high metabolic demand, such as the cytosol surrounding the contraction fibers and electrolyte transport pumps. The depletion of ATP in critical cellular areas and the accumulation of toxins result in loss of contractility within about 60 seconds of total myocardial ischemia **(Choice A)**.

As ischemia persists, ATP levels within the affected cardiac myocytes continue to fall while lactate levels continue to rise. When ischemia lasts less than 30 minutes, loss of contractile function is reversible. However, on restoration of blood flow, full myocardial contractility is not immediately restored. Instead, there is prolonged dysfunction of the myocardium (myocardial stunning), with contractility gradually returning to normal over the next several hours to days. Increasing durations of ischemia prolong the time that the myocardium remains stunned.

Under hypoxic conditions, ATP within cardiomyocytes is degraded to ADP, AMP, and eventually adenosine. Adenosine is able to cross the cell membrane and function as a vasodilator when coronary blood flow is insufficient to meet myocardial demand. However, persistent ischemia can lead to depletion of massive amounts of adenosine. After 30 minutes of total myocardial ischemia, about half of the cellular adenine stores are lost. At this point, failure of cellular homeostasis is inevitable and ischemic injury becomes irreversible **(Choice E)**.

Educational objective:
Loss of cardiomyocyte contractility occurs within 60 seconds after the onset of total ischemia. When ischemia lasts less than 30 minutes, restoration of blood flow leads to reversible contractile dysfunction (myocardial stunning), with contractility gradually returning to normal over the next several hours to days. However, after about 30 minutes of total ischemia, ischemic injury becomes irreversible.

Pathophysiology	Cardiovascular System	Myocardial infarction
Subject	System	Topic

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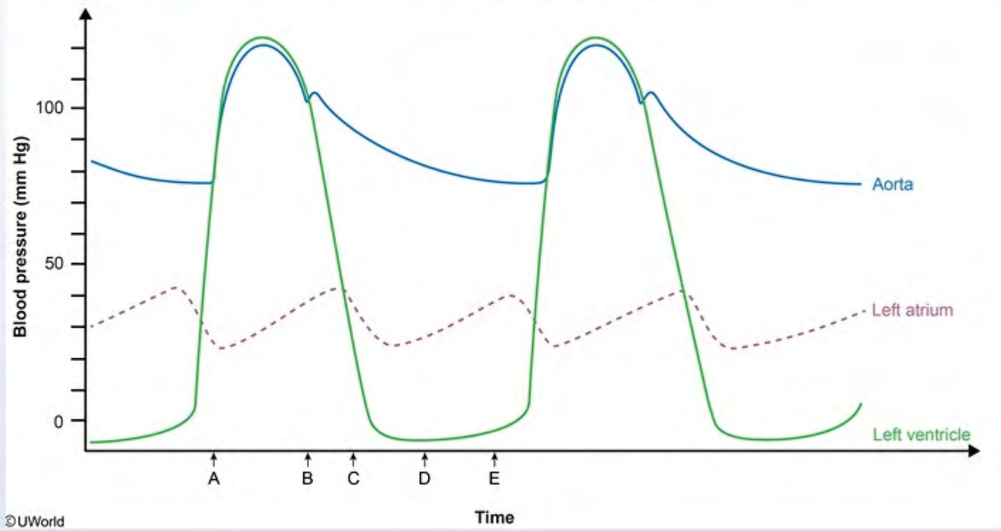
A 32-year-old woman comes to the office due to progressive exertional dyspnea and easy fatigability. She immigrated to the United States from India several years ago and reports a history of rheumatic fever during her childhood. Cardiac auscultation reveals a diastolic high-frequency sound and a rumbling diastolic murmur heard best in the left fifth intercostal space at the midclavicular line. Cardiac catheterization is performed for hemodynamic evaluation, and the results are represented below. Timing of the high-frequency sound heard during cardiac auscultation of this patient best corresponds to which of the following letters?



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- B. B
- C. C
- D. D
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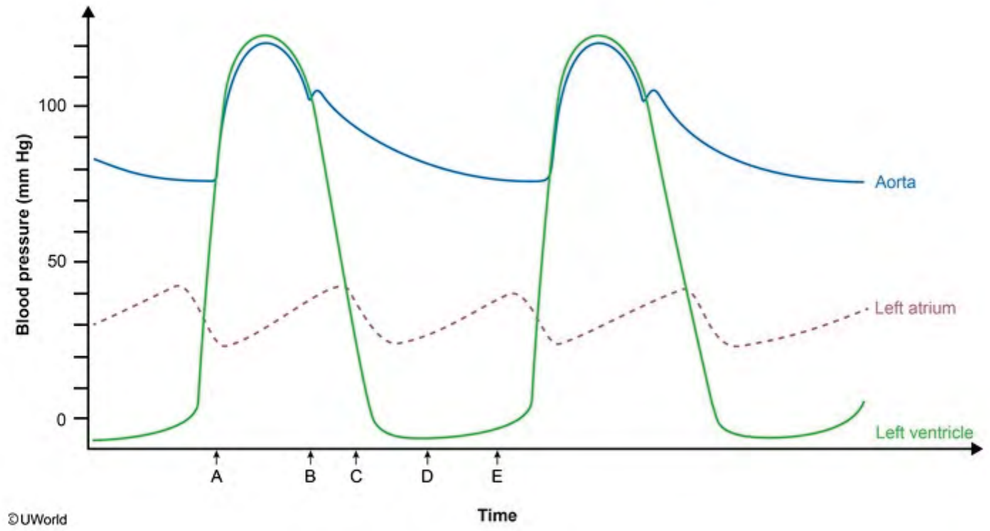

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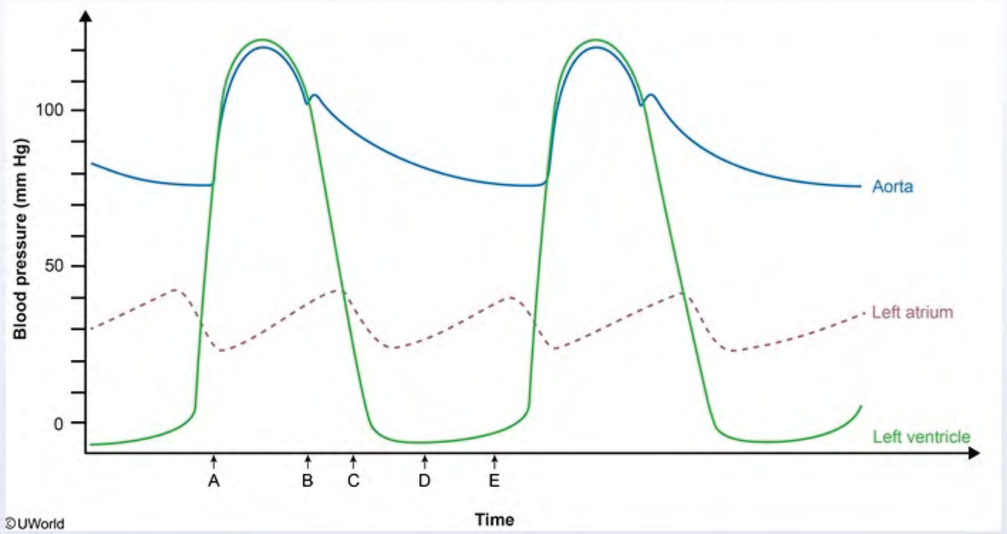

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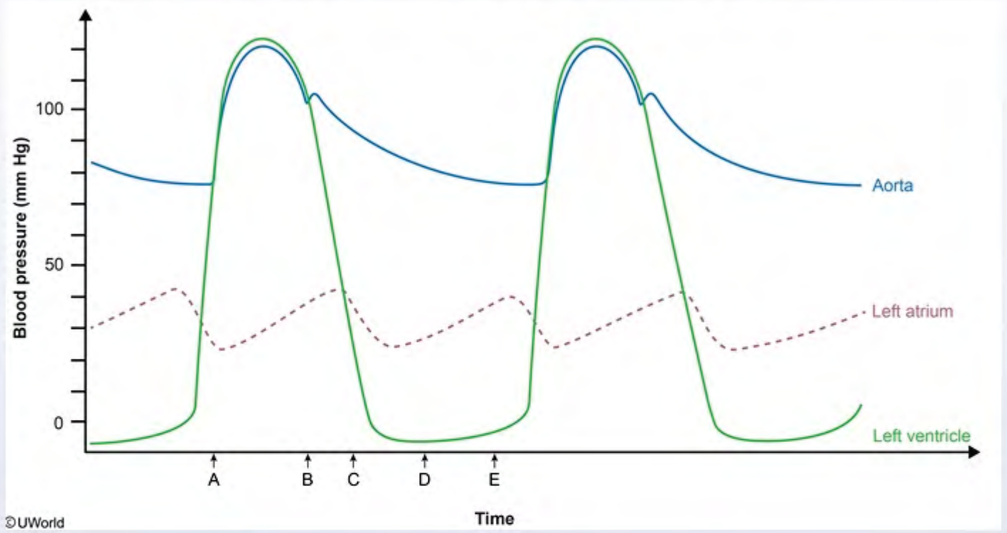

- A. A (11%)
- ✗ B. B (7%)
- ✓ C. C (43%)
- D. D (24%)
- E. E (13%)

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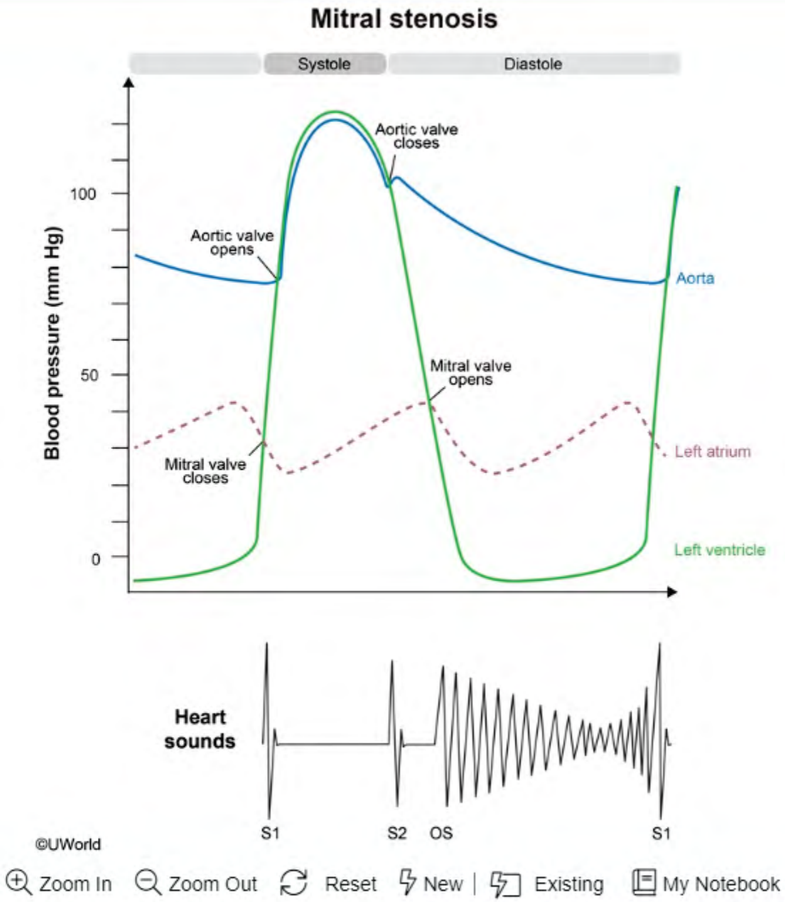

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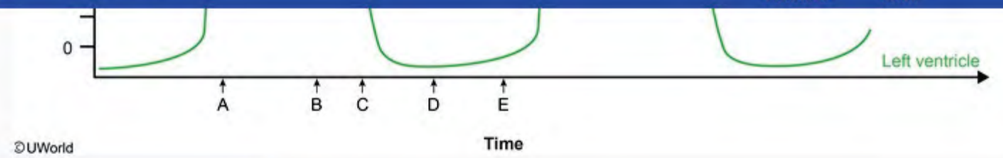

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- ✗ B. B (7%)
- ✓ C. C (43%)
- D. D (24%)
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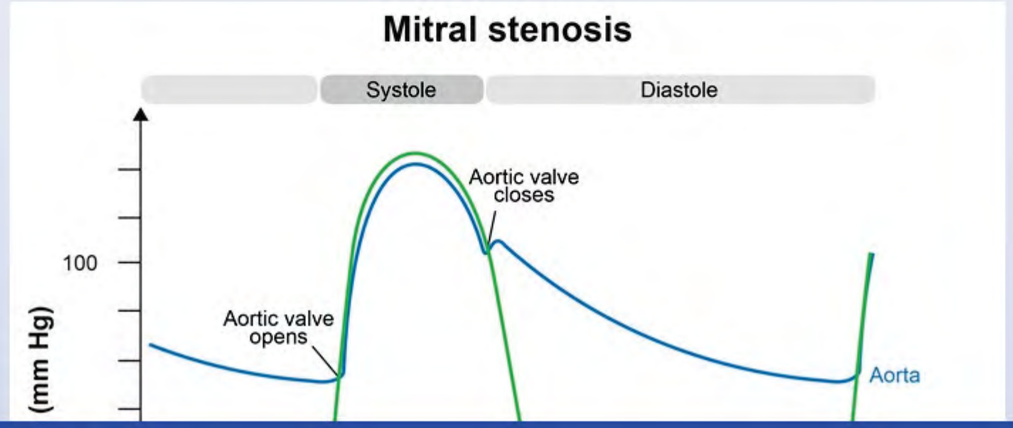

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This patient with a history of rheumatic fever now presenting with exertional dyspnea and a diastolic murmur at the cardiac apex most likely has **mitral stenosis**. In comparison to normal tracings, hemodynamic tracings in patients with mitral stenosis typically reveal an elevated pressure gradient between the left atrial and left ventricular end-diastolic pressure.

Classic physical examination findings include a loud first heart sound (S1), an early diastolic high-frequency **opening snap** after the second heart sound (S2), and a low-pitched **diastolic rumble**. These findings are best heard at the **cardiac apex** using the bell of the stethoscope with the patient lying on the left side in held expiration.

Left ventricular pressure falls rapidly during isovolumetric relaxation. The opening snap is caused by the sudden opening of the mitral valve leaflets when the left ventricular diastolic pressure falls below the left atrial pressure at the beginning of diastole. As mitral stenosis becomes more severe, the opening snap occurs earlier after S2.

(Choice A) This point corresponds to the opening of the aortic valve at the end of isovolumetric ventricular contraction in early systole when the left ventricular pressure exceeds the central aortic pressure. This correlates with the timing of an aortic ejection murmur that may be heard in patients with aortic stenosis (eg, due to a calcified bicuspid aortic valve).

(Choice B) This point corresponds to the closure of the aortic valve at the end of systole, producing the aortic component of S2. The aortic valve closes as soon as left ventricular pressure drops below the aortic pressure.

(Choice D) This is a random point in diastole when the mitral valve is already maximally open and there is passive filling of the left ventricular cavity (diastasis). In patients with mitral stenosis, the elevated pressure gradient between the left atrium and left ventricle during diastole causes the mid-diastolic rumble.

(Choice E) This point roughly corresponds to the onset of atrial contraction during late ventricular diastole. In patients in sinus rhythm, atrial contraction causes an increase in the intensity of the diastolic murmur just before S1, a phenomenon termed "presystolic accentuation."

Educational objective:
Cardiac auscultation in patients with mitral stenosis reveals a loud first heart sound, an early diastolic opening snap

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diastolic pressure.

Classic physical examination findings include a loud first heart sound (S1), an early diastolic high-frequency **opening snap** after the second heart sound (S2), and a low-pitched **diastolic rumble**. These findings are best heard at the **cardiac apex** using the bell of the stethoscope with the patient lying on the left side in held expiration.

Left ventricular pressure falls rapidly during isovolumetric relaxation. The opening snap is caused by the sudden opening of the mitral valve leaflets when the left ventricular diastolic pressure falls below the left atrial pressure at the beginning of diastole. As mitral stenosis becomes more severe, the opening snap occurs earlier after S2.

(Choice A) This point corresponds to the opening of the aortic valve at the end of isovolumetric ventricular contraction in early systole when the left ventricular pressure exceeds the central aortic pressure. This correlates with the timing of an aortic ejection murmur that may be heard in patients with aortic stenosis (eg, due to a calcified bicuspid aortic valve).

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(Choice D) This is a random point in diastole when the mitral valve is already maximally open and there is passive filling of the left ventricular cavity (diastasis). In patients with mitral stenosis, the elevated pressure gradient between the left atrium and left ventricle during diastole causes the mid-diastolic rumble.

(Choice E) This point roughly corresponds to the onset of atrial contraction during late ventricular diastole. In patients in sinus rhythm, atrial contraction causes an increase in the intensity of the diastolic murmur just before S1, a phenomenon termed "presystolic accentuation."

Educational objective:
Cardiac auscultation in patients with mitral stenosis reveals a loud first heart sound, an early diastolic opening snap after the second heart sound, and a low-pitched diastolic rumble best heard at the cardiac apex. The opening snap is caused by the sudden opening of the mitral valve leaflets when the left ventricular pressure falls below the left atrial pressure at the beginning of diastole.

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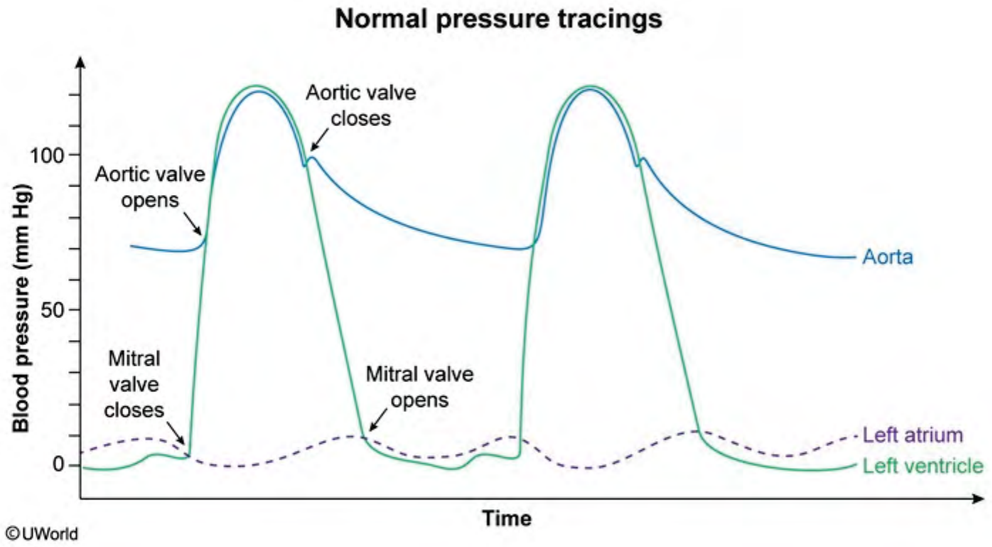

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A 39-year-old man comes to the office due to increasing fatigue for the past 2 weeks. The patient reports no prior medical conditions, although he recently had a self-limiting upper respiratory illness. He does not use tobacco, alcohol, or illicit drugs and has not traveled recently. Temperature is 37.2 C (99 F), blood pressure is 116/66 mm Hg, pulse is 100/min, and respirations are 20/min. Physical examination reveals lung crackles. Echocardiography shows no valvular abnormalities and the following parameters:

Left ventricular end-diastolic volume	125 mL
Left ventricular end-systolic volume	75 mL

Based on the observed findings, which of the following is most accurate about this patient?

- A. Cardiac output is 4 L/min
- B. Cardiac output is 6 L/min
- C. Left ventricular ejection fraction is 40%
- D. Left ventricular ejection fraction is 60%
- E. Stroke volume is 40 mL
- F. Stroke volume is 60 mL

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Left ventricular end-diastolic volume	125 mL
Left ventricular end-systolic volume	75 mL

Based on the observed findings, which of the following is most accurate about this patient?

- ✗ A. Cardiac output is 4 L/min (1%)
- B. Cardiac output is 6 L/min (1%)
- ✓ C. Left ventricular ejection fraction is 40% (77%)
- D. Left ventricular ejection fraction is 60% (16%)
- E. Stroke volume is 40 mL (2%)
- F. Stroke volume is 60 mL (0%)

Incorrect
Correct answer
C

77%
Answered correctly

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Explanation

Stroke volume & ejection fraction

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F. Stroke volume is 60 mL (0%)

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Stroke volume & ejection fraction

Stroke volume (SV) = End-diastolic volume (EDV) − End-systolic volume

$$\text{Ejection fraction} = \frac{\text{SV}}{\text{EDV}}$$

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Left ventricular **stroke volume** is the absolute volume of blood ejected from the left ventricle with each contraction. It is calculated by subtracting the volume of blood in the left ventricle at the end of contraction, the **left ventricular end-systolic volume**, from the volume of blood in the left ventricle at the beginning of contraction, the **left ventricular end-diastolic volume** (LVEDV). This patient's stroke volume is 50 mL (125 mL − 75 mL), which is lower than the expected normal value of approximately 70 mL.

Ejection fraction, the relative volume of blood ejected from the left ventricle with each contraction, is calculated by dividing stroke volume by LVEDV. In this patient, the ejection fraction is 0.4 (50 mL / 125 mL). The fraction is typically multiplied by 100 and expressed as a percentage; therefore, this patient's left ventricular ejection fraction is **40%** (0.4 × 100), which is lower than the expected normal value of ≥50%.

Cardiac output, the volume of blood ejected into the aorta per unit time, is estimated by multiplying stroke volume by heart rate. This patient's cardiac output is 5 L/min (50 mL × 100/min), which is normal. Although it is not pertinent to this patient, this equation overestimates cardiac output in the setting of mitral regurgitation because a portion of the stroke volume flows backward into the left atrium with each contraction.

This patient's presentation is consistent with left ventricular **systolic dysfunction** leading to **heart failure with reduced ejection fraction**. He has only mild symptoms at present because the low stroke volume is being compensated by an elevated heart rate to maintain cardiac output; however, further decompensation with reduced cardiac output is likely over time.

Educational objective:
Stroke volume is the absolute volume of blood ejected from the left ventricle with each contraction and is calculated by subtracting left ventricular end-systolic volume from left ventricular end-diastolic volume (LVEDV). Ejection fraction is the relative volume of blood ejected from the left ventricle with each contraction; it is calculated by dividing stroke volume by LVEDV. Cardiac output, the volume of blood ejected into the aorta per unit time, is estimated by multiplying stroke volume by heart rate.

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A 42-year-old man is brought to the emergency department 1 hour after the onset of severe headache, nausea, vomiting, and confusion. The patient has primary hypertension and chronic kidney disease; he has been prescribed 2 antihypertensive agents but has been noncompliant with therapy recently. Temperature is 36.8 C (98.2 F), blood pressure is 240/150 mm Hg, heart rate is 90/min, and respirations are 20/min. Ophthalmologic examination shows bilateral papilledema. The lungs are clear to auscultation. Cardiac examination reveals an S4 and no murmurs. Laboratory results are as follows:

	Today	2 months ago
Hematocrit	23%	30%
Platelets	78,000/mm^3	150,000/mm^3
Blood urea nitrogen	60 mg/dL	26 mg/dL
Serum creatinine	4.5 mg/dL	1.8 mg/dL
Peripheral smear	numerous schistocytes	

Which of the following pathologic findings in the kidney is most correlated with this patient's acute condition?

- A. Fibrinoid necrosis of small arteriolar walls
- B. Focal glomerulosclerosis
- C. Granulomatous inflammation of the media
- D. Homogenous acellular thickening of arteriolar walls
- E. Interstitial fibrosis and tubular atrophy

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Blood urea nitrogen	60 mg/dL	26 mg/dL
Serum creatinine	4.5 mg/dL	1.8 mg/dL
Peripheral smear	numerous schistocytes	

Which of the following pathologic findings in the kidney is most correlated with this patient's acute condition?

- ✓ A. Fibrinoid necrosis of small arteriolar walls (40%)
- B. Focal glomerulosclerosis (10%)
- C. Granulomatous inflammation of the media (3%)
- D. Homogenous acellular thickening of arteriolar walls (30%)
- E. Interstitial fibrosis and tubular atrophy (14%)

Correct

40% Answered correctly

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Hypertensive emergency is a condition defined as severely elevated blood pressure (typically >180/120 mm Hg) with evidence of ongoing end-organ damage (eg, renal injury). Hypertensive emergencies typically occur in patients with preexisting essential hypertension and may be induced by medication noncompliance, which can result in a rapid increase in systemic blood pressure. Severe hypertension can cause damage to the vascular endothelium, leading to increased vascular permeability to coagulation factors (eg, fibrinogen), platelet activation, and endothelial cell death. In the kidney, this manifests as **malignant nephrosclerosis**, which is characterized by 2 distinct histopathologic patterns:

- **Fibrinoid necrosis:** Cell death and excessive fibrin deposition within the arteriolar walls is visible as circumferential, amorphous, pink material with smudged, necrotic endothelial cells that lack cytologic detail.
- **Hyperplastic arteriolosclerosis:** Over time, activated platelets and injured endothelial cells release growth factors, which induce concentric hyperplasia and layering of smooth muscle cells and collagen, resulting in intimal thickening and an "onion-skin" appearance.

These changes result in narrowing and/or obliteration of the arteriolar lumen; the resultant erythrocyte fragmentation and platelet consumption can cause a **microangiopathic hemolytic anemia** that is histologically indistinguishable from hemolytic uremic syndrome. Laboratory abnormalities reflect renal injury (eg, elevated creatinine and blood urea nitrogen) and hemolytic anemia (eg, schistocytes [fragmented red blood cells] on peripheral smear, elevated lactate dehydrogenase and indirect bilirubin).

Other end-organ manifestations of hypertensive emergency include papilledema, retinal hemorrhages, pulmonary edema, acute coronary syndrome, encephalopathy, and stroke.

(Choices B, D, and E) Hypertensive nephrosclerosis (ie, long-term essential hypertension) is characterized by interstitial fibrosis, tubular atrophy, and varying degrees of glomerulosclerosis (eg, focal or global). Hyaline arteriolosclerosis is characterized by deposition of hyaline (glassy, homogenous) material in the intima and media of small arteries and arterioles. These changes may be seen in chronic hypertension; however, the severely elevated blood pressure and evidence of end-organ dysfunction in this patient are indicative of acute hypertensive emergency and malignant nephrosclerosis.

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(Choice C) Granulomatous inflammation of the media characterizes temporal (giant cell) arteritis, the most common form of systemic vasculitis in adults.

Educational objective:
Hypertensive emergency is severely elevated blood pressure (typically >180/120 mm Hg) with evidence of end-organ damage. In the kidneys, this can manifest as malignant nephrosclerosis, characterized by fibrinoid necrosis and hyperplastic arteriolosclerosis ("onion-skin" appearance). A microangiopathic hemolytic anemia can occur due to erythrocyte fragmentation and platelet consumption at the narrowed arteriolar lumen.

Pathology	Cardiovascular System	Primary hypertension
Subject	System	Topic

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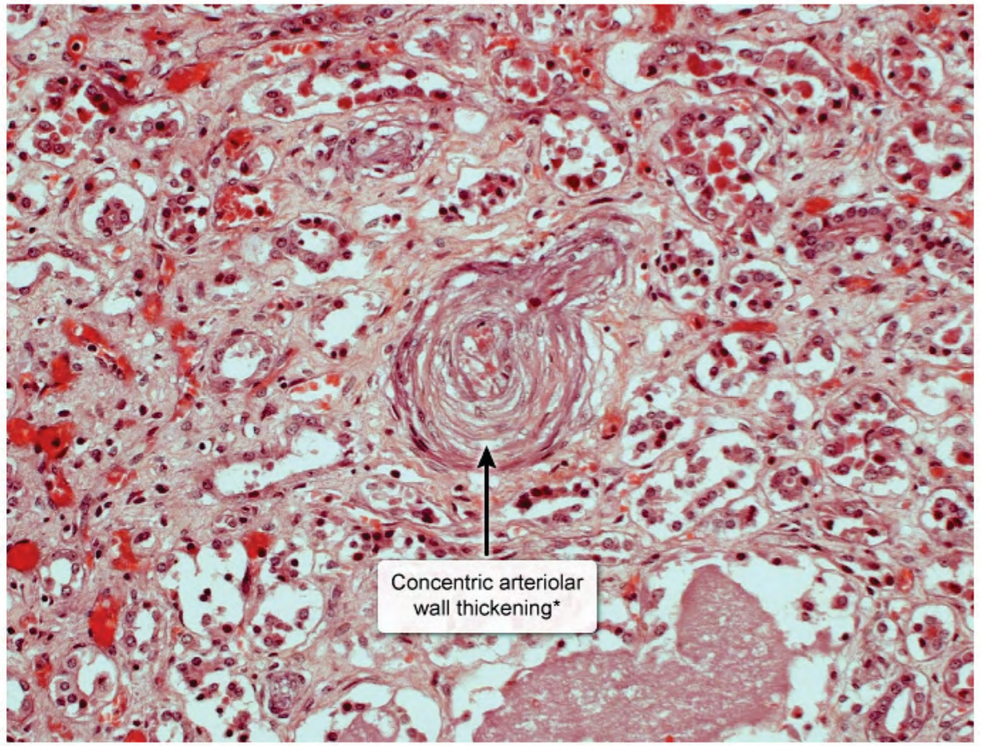
Hyperplastic arteriolosclerosis



*"Onion skin" appearance.

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- **Fibrinoid necrosis:** Cell death and excessive fibrin deposition within the arteriolar walls is visible as ci
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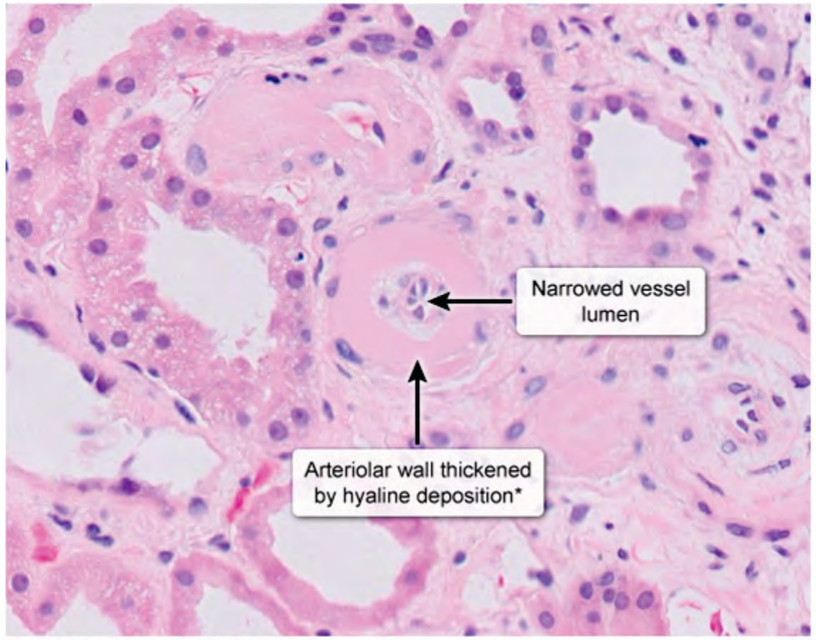
Hyaline arteriolosclerosis



*Homogenous, glassy, eosinophilic material.

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factors, which induce concentric hyperplasia and layering of smooth muscle cells and collagen, resulting in

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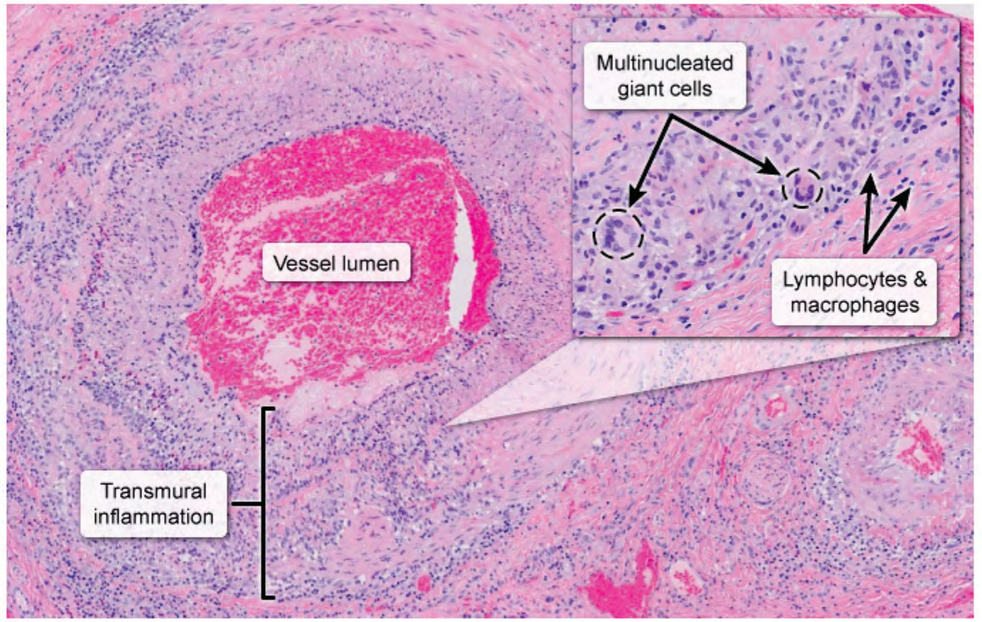
Giant cell arteritis



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Pathology (Subject) | Cardiovascular System (System) | Primary hypertension (Topic)

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At autopsy, the heart of an 80-year-old man has a small left ventricular cavity and thickening of the left ventricle walls, which measure 1.7 cm (normal: 0.7-1.1) at the posterior and septal wall locations. Microscopic examination shows diffusely enlarged cardiomyocytes, with prominent nuclei evident at low power. Increased expression of which of the following substances most likely contributed to these pathologic findings?

- A. Adenosine
- B. Angiotensin II
- C. Calcitonin gene-related peptide
- D. Interleukin-1
- E. Nitric oxide synthase

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- A. Adenosine (9%)
- ✓ B. Angiotensin II (71%)
- C. Calcitonin gene-related peptide (11%)
- D. Interleukin-1 (4%)
- E. Nitric oxide synthase (3%)

Correct | 71% Answered correctly | 03 secs Time Spent | 2023 Version

Explanation

The pathology evaluation of this patient's heart shows uniformly thickened left ventricle walls (resulting in decreased ventricular cavity size) and enlarged cardiomyocytes with highly visible nuclei, which are consistent with **concentric left ventricular hypertrophy** (LVH). This condition commonly results from **chronic hypertension**, which leads to **increased local expression** of the vasoconstrictors **angiotensin II** and **endothelin** in the heart; these 2 mediators likely have a prominent role in the development of LVH.

Although all medications that lower systemic blood pressure (eg, calcium channel blockers, thiazide diuretics) can reduce concentric LVH, **angiotensin II receptor blockers** provide the greatest **reduction in remodelling**, suggesting a role of angiotensin II in the development of LVH that is independent of blood pressure. Endothelin

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Although all medications that lower systemic blood pressure (eg, calcium channel blockers, thiazide diuretics) can reduce concentric LVH, **angiotensin II receptor blockers** provide the greatest **reduction in remodelling**, suggesting a role of angiotensin II in the development of LVH that is independent of blood pressure. Endothelin receptor blockers (eg, bosentan) are not typically used to treat systemic hypertension, but they may reduce right ventricular hypertrophy when used to treat pulmonary arterial hypertension.

Local angiotensin II production also likely has a prominent role in the development of eccentric LVH in response to left ventricular volume overload.

(Choice A) A compensatory increase in adenosine expression occurs in response to myocardial ischemia and reperfusion of the heart. Adenosine dilates blood vessels, especially coronary arteries, to improve circulation; it does not contribute to LVH.

(Choice C) Calcitonin gene-related peptide (CGRP) is a neuropeptide that is a potent vasodilator, and it may have a role in protecting the heart from heart failure and LVH. CGRP blockers are used to prevent migraines by reducing the size of abnormally dilated blood vessels in the brain.

(Choice D) Interleukin-1 is a proinflammatory cytokine implicated in atherosclerosis development; high levels are expected in coronary artery disease but not in LVH. Interleukin-1 blockers can be used to treat autoimmune inflammatory conditions (eg, rheumatoid arthritis).

(Choice E) Nitric oxide synthase is an enzyme that catalyzes the production of nitric oxide (a potent vasodilator). Nitric oxide is protective against LVH, and relatively low levels of nitric oxide synthase are expected with LVH.

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(Choice E) Nitric oxide synthase is an enzyme that catalyzes the production of nitric oxide (a potent vasodilator). Nitric oxide is protective against LVH, and relatively low levels of nitric oxide synthase are expected with LVH.

Educational objective:
Concentric left ventricular hypertrophy (LVH) is a common complication of chronic hypertension, characterized by uniformly thickened ventricular walls and enlarged cardiomyocytes and nuclei. The vasoconstrictors angiotensin II and endothelin likely have a prominent role in the development of concentric LVH independent of their effects on blood pressure. Angiotensin II receptor blockers are more effective than other antihypertensive agents in reducing LVH.

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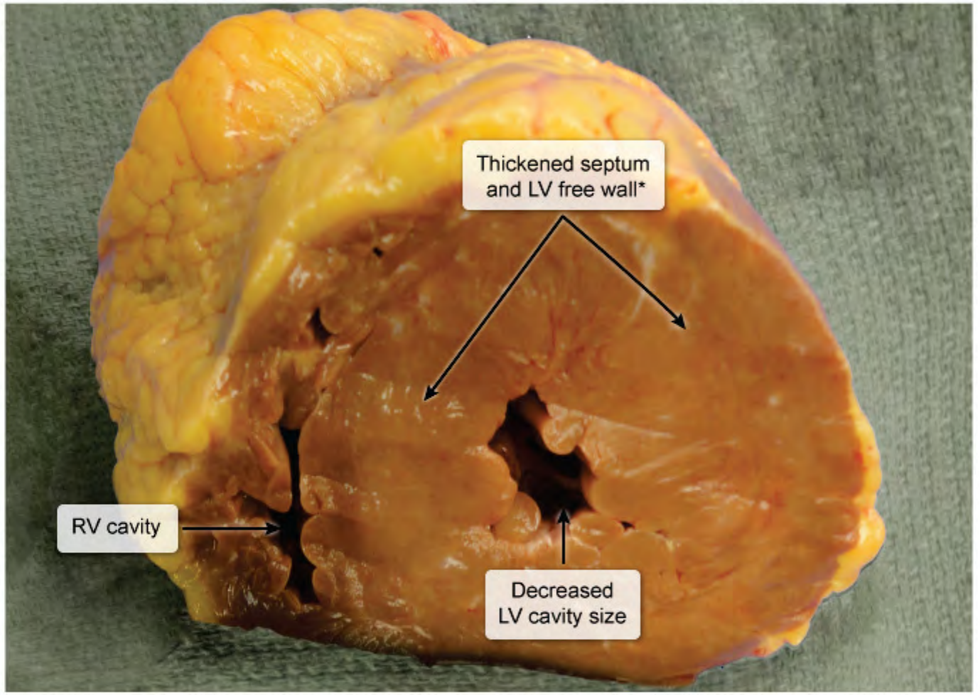
Hypertensive heart disease



*LV walls thickened via addition of sarcomeres in parallel (ie, concentric hypertrophy), resulting in decreased LV cavity size

LV = left ventricular; **RV** = right ventricular

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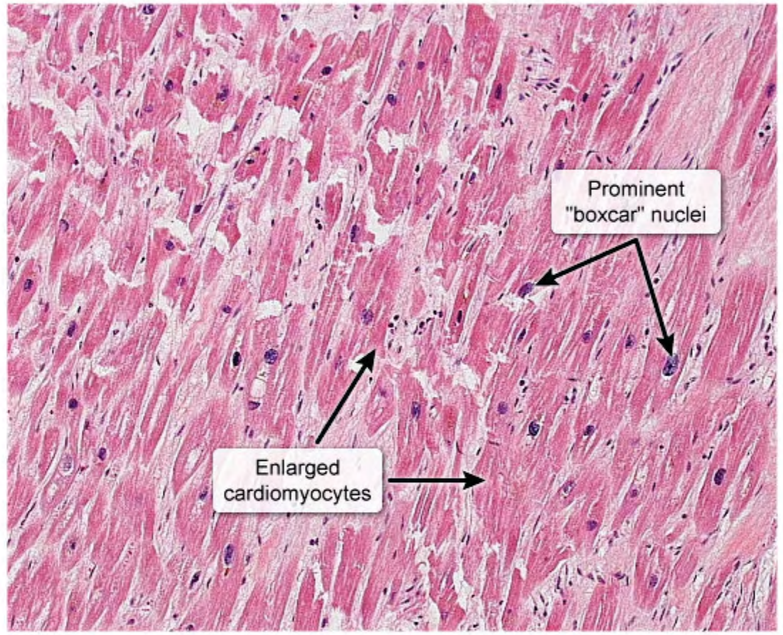
Cardiac hypertrophy



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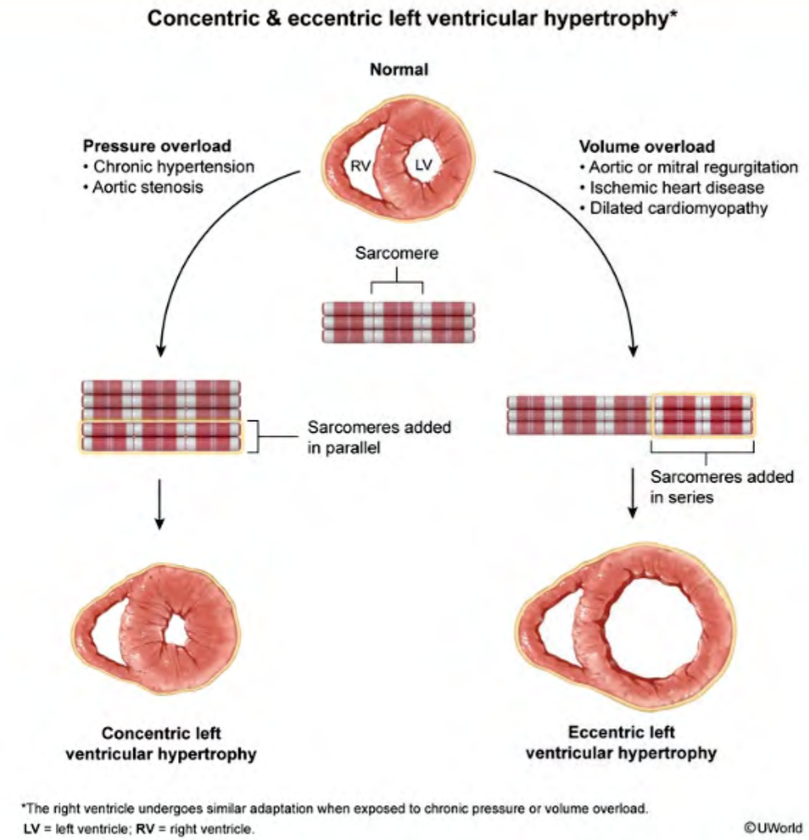

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Nitric oxide is protective against LVH, and relatively low levels of nitric oxide synthase are expected with LVH.

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A 70-year-old woman comes to the office due to progressive exertional dyspnea over the last year. She has no chest pain or lower-extremity swelling. The patient has long-standing hypertension but has not always been adherent to her medical regimen. She is a lifetime nonsmoker. Blood pressure is 164/73 mm Hg and pulse is 88/min and regular. An S4 is heard on cardiac auscultation. The lungs are clear. Echocardiogram reveals left atrial enlargement, moderate concentric left ventricular hypertrophy, and a left ventricular ejection fraction of 67%. Which of the following changes are most likely to be seen in this patient?

	Left ventricular compliance	Angiotensin II signaling	Cardiac myosin heavy chain-beta expression
○ A.	Decreased	Increased	Increased
○ B.	Decreased	Decreased	Increased
○ C.	Decreased	Increased	Decreased
○ D.	Increased	Increased	Increased
○ E.	Increased	Increased	Decreased
○ F.	Increased	Decreased	Decreased

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	Left ventricular compliance	Angiotensin II signaling	Cardiac myosin heavy chain-beta expression	
✓ A.	Decreased	Increased	Increased	(63%)
✗ B.	Decreased	Decreased	Increased	(25%)
C.	Decreased	Increased	Decreased	(5%)
D.	Increased	Increased	Increased	(3%)
E.	Increased	Increased	Decreased	(0%)
F.	Increased	Decreased	Decreased	(1%)

Incorrect
Correct answer
A

63%
Answered correctly

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Explanation

This patient's clinical presentation and echocardiography findings are consistent with **concentric left ventricular (LV) hypertrophy** leading to **impaired diastolic functioning** (heart failure with preserved ejection fraction)

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Explanation

This patient's clinical presentation and echocardiography findings are consistent with **concentric left ventricular (LV) hypertrophy** leading to **impaired diastolic functioning** (heart failure with preserved ejection fraction).

In response to LV pressure overload (eg, prolonged systemic hypertension), there is **upregulation** of genes encoding for contractile proteins of the cardiac sarcomere, including **beta-myosin heavy chain**. The additional contractile fibers are added in parallel to increase the contractile strength of the LV wall. Although LV contractility and ejection fraction are increased, the thickened LV walls are now stiff and less compliant, which is often evidenced by an S4 on cardiac auscultation as blood strikes the stiff LV wall during atrial systole.

Subsequent impaired diastolic filling of the **noncompliant left ventricle** leads to reduced stroke volume with reduced cardiac output and decreased renal perfusion. Falsely sensing a need to increase blood volume and organ perfusion, the kidneys activate the renin-angiotensin-aldosterone system (RAAS) to stimulate sodium retention (via increased aldosterone) and vasoconstriction (via **increased angiotensin II**). Although this response can temporarily maintain cardiac output, the increasing fluid retention will ultimately precipitate decompensated heart failure.

(Choices B, C, E, and F) These combinations of changes in LV compliance, angiotensin II signaling, and beta-myosin heavy chain expression are not consistent with readily identifiable, untreated disease processes.

(Choice D) Increases in LV compliance, angiotensin II signaling, and beta-myosin heavy chain expression are consistent with heart failure with reduced ejection fraction due to dilated cardiomyopathy. There is upregulation of contractile proteins; however, this occurs in response to volume overload rather than pressure overload, and therefore the contractile fibers are added in series (ie, eccentric hypertrophy) rather than in parallel. This leads to dilation of the LV cavity with increased wall compliance and decreased contractility. Reduced cardiac output with subsequent activation of the RAAS results in increased angiotensin II signaling.

Educational objective:
Prolonged systemic hypertension leads to concentric left ventricular (LV) hypertrophy via the addition of myocardial

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Educational objective:
Prolonged systemic hypertension leads to concentric left ventricular (LV) hypertrophy via the addition of myocardial contractile fibers in parallel. The thickening of the LV walls reduces LV compliance, leading to impaired diastolic filling and heart failure with preserved ejection fraction. In response to reduced cardiac output, the kidneys activate the renin-angiotensin-aldosterone system, stimulating sodium retention and vasoconstriction that worsens volume overload and can lead to decompensated heart failure.

Physiology	Cardiovascular System	Diastolic dysfunction
Subject	System	Topic

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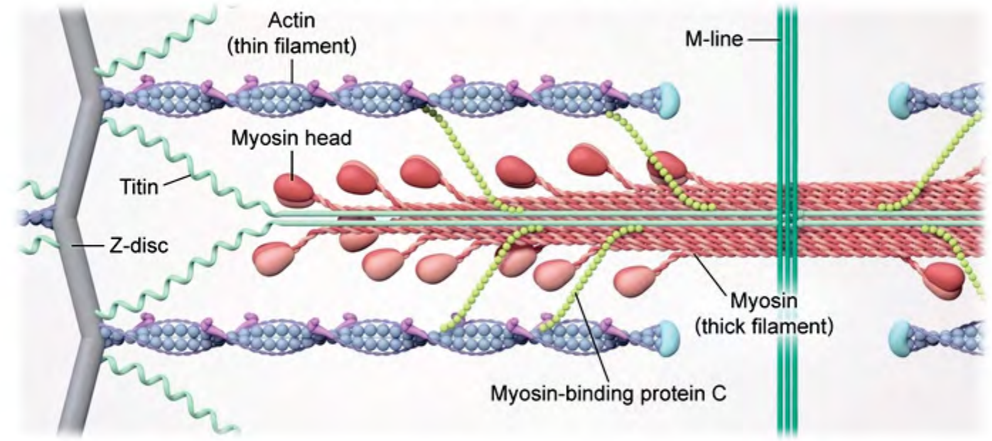
Cardiac sarcomere protein structure



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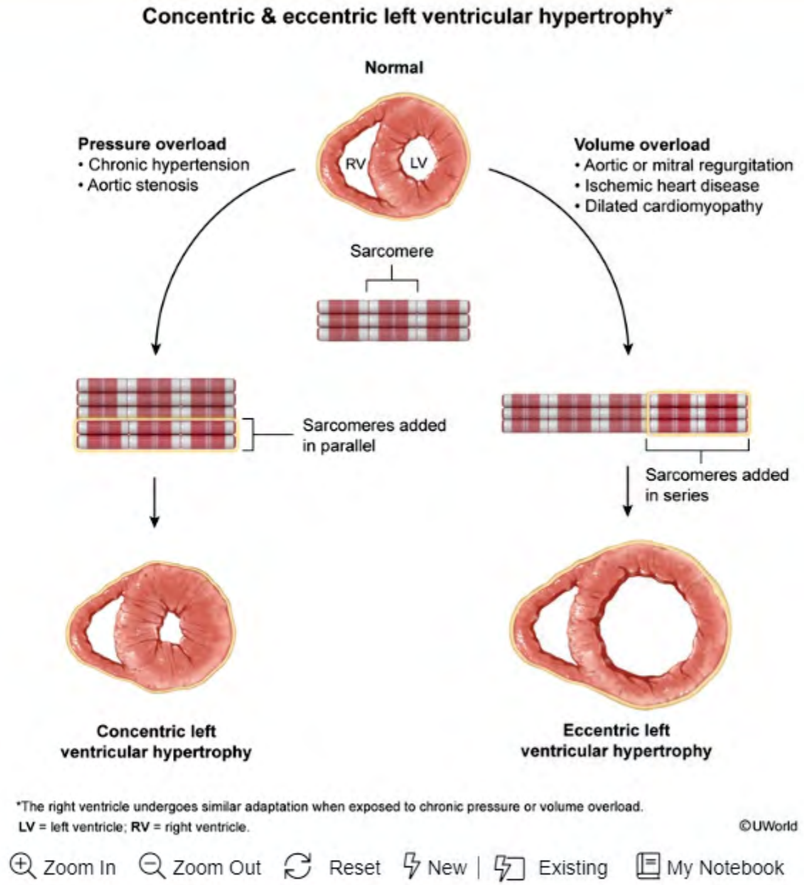

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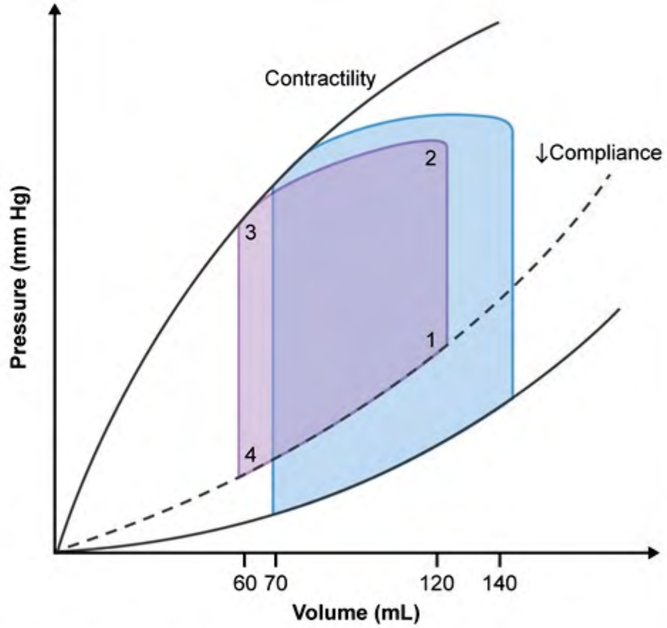
Heart failure with preserved ejection fraction due to LV diastolic dysfunction



1. End of diastole
2. Aortic valve opens
3. End of systole
4. Mitral valve opens

Normal
SV ≈ 70 mL
EF >50%

Diastolic dysfunction
SV ≈ 60 mL
EF >50%

EF = ejection fraction; **LV** = left ventricular; **SV** = stroke volume.

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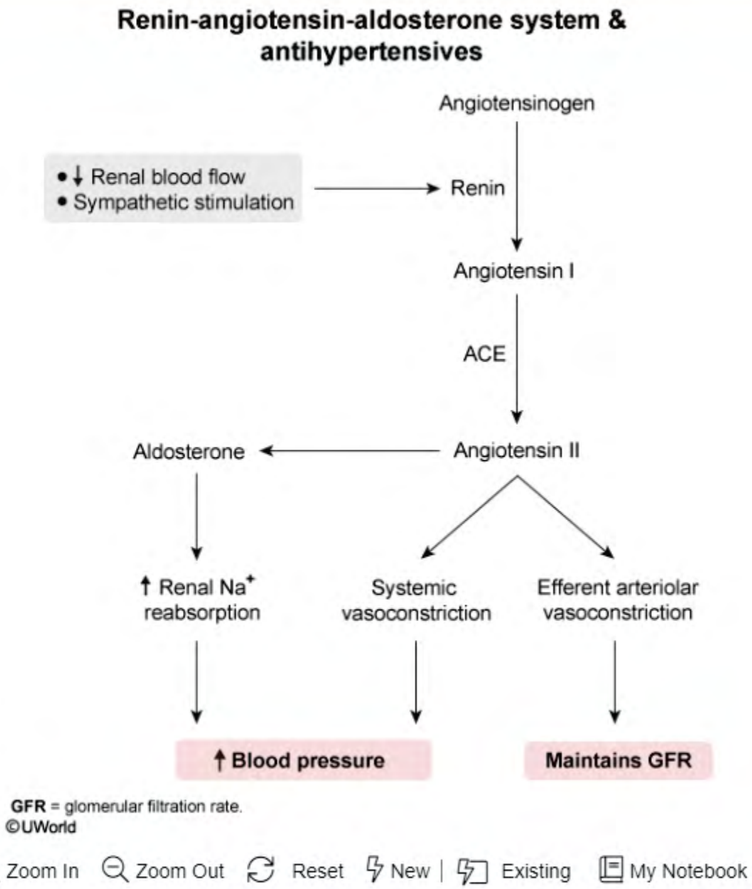

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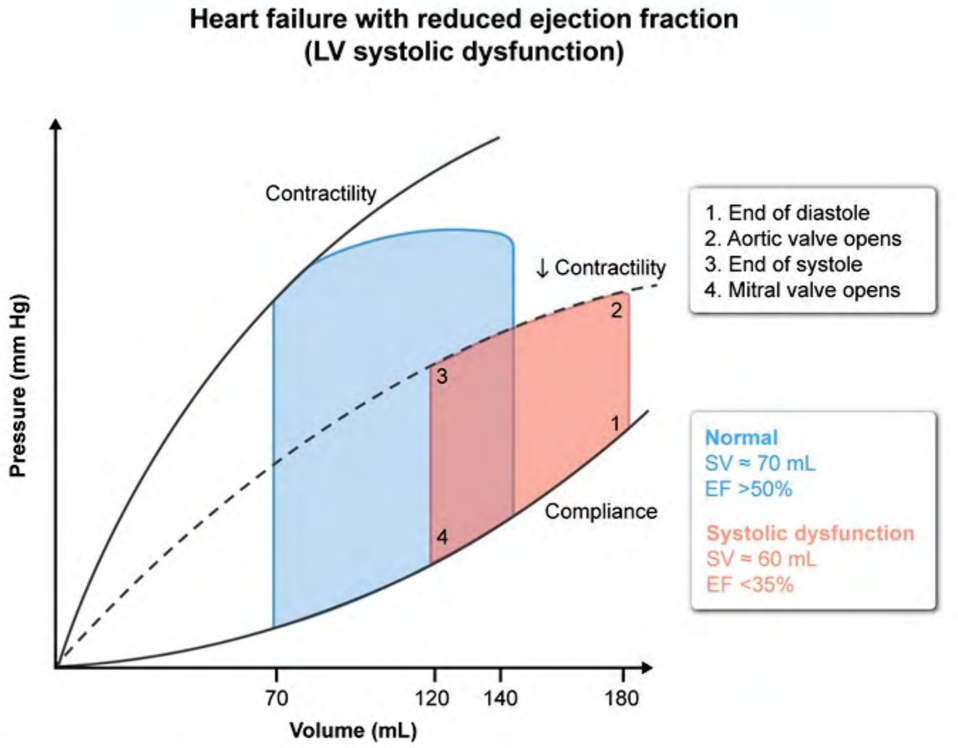

EF = ejection fraction; LV = left ventricular; SV = stroke volume.

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A 52-year-old man comes to the office due to a chronic cough for the last 3 weeks. The patient says, "I haven't been coughing up phlegm, but sometimes I notice some blood on the tissue when I cough particularly hard." He has smoked 2 packs of cigarettes daily for the past 30 years and drinks 3 or 4 cans of beer on weekends. He works as a welder on an assembly line and says his father died of lung cancer at age 70. Physical examination shows right-sided face and arm swelling and engorgement of subcutaneous veins on the same side of the neck. Which of the following veins is most likely obstructed in this patient?

- A. Axillary
- B. Brachiocephalic
- C. External jugular
- D. Internal jugular
- E. Subclavian
- F. Superior vena cava

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- A. Axillary (0%)
- ✓ B. Brachiocephalic (34%)
- C. External jugular (5%)
- D. Internal jugular (3%)
- E. Subclavian (21%)
- F. Superior vena cava (35%)

Correct

34% Answered correctly

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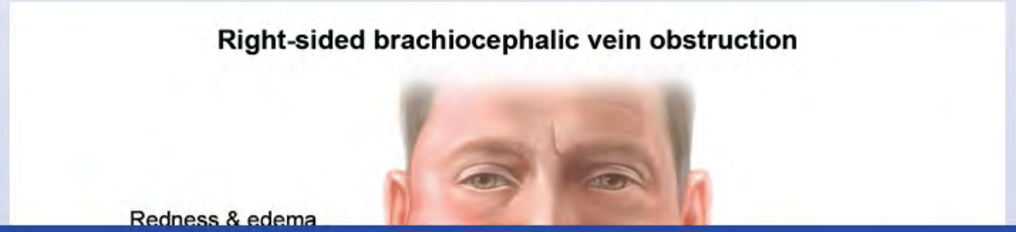
Explanation



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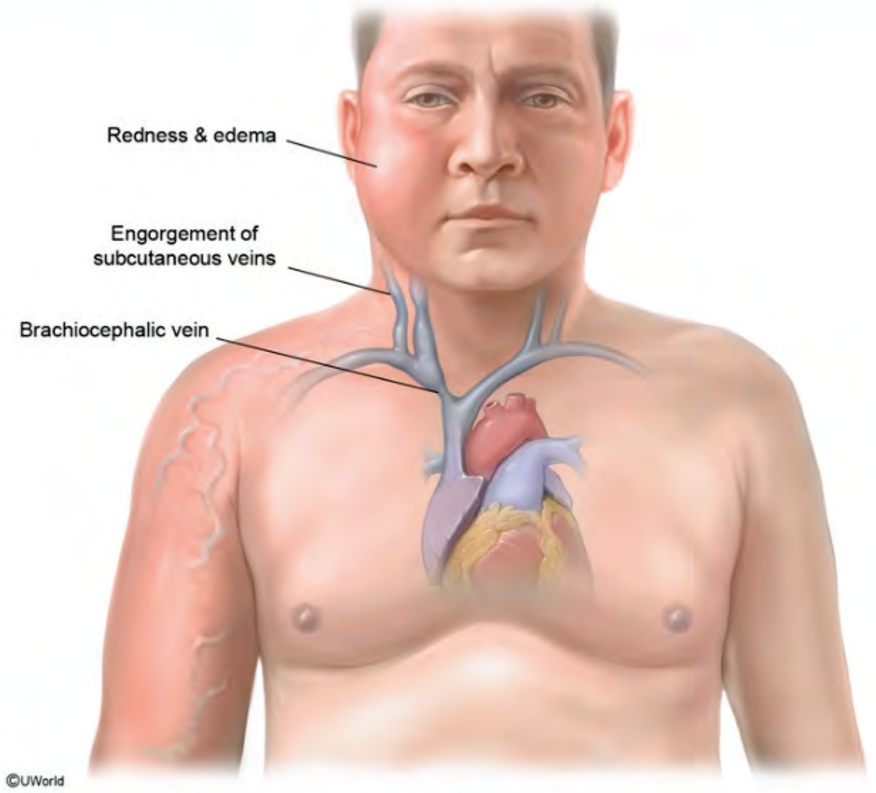
Right-sided brachiocephalic vein obstruction



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This patient has symptoms consistent with an **obstructed right brachiocephalic (innominate) vein**. This may be the result of external compression by an apical lung tumor or thrombotic occlusion as can occur when a central catheter has been in place for an extended period. The right brachiocephalic vein is formed by the union of the right subclavian vein and the right internal jugular vein. The right external jugular vein drains into the right subclavian vein, so obstruction of the right brachiocephalic vein will also cause venous congestion of structures drained by the external jugular vein. It is important to note that the right brachiocephalic vein also drains the right lymphatic duct, which drains lymph from the right upper extremity, the right face and neck, the right hemithorax, and the right upper quadrant of the abdomen.

(Choices A and E) The subclavian vein is the continuation of the axillary vein. Both drain blood from the upper extremity. Blockage at any of these 2 sites would cause unilateral arm swelling without associated facial swelling.

(Choices C and D) The external jugular vein drains the scalp and portions of the lateral face. The internal jugular vein drains the brain and superficial face and neck. Obstruction of the internal jugular veins would not cause arm swelling.

(Choice F) Superior vena cava (SVC) syndrome results when the superior vena cava is obstructed. The signs and symptoms of SVC syndrome are similar to those of obstructed right brachiocephalic (innominate) vein, except that both sides of the face, neck, and chest and both arms would be involved.

Educational objective:
The brachiocephalic vein drains the ipsilateral jugular and subclavian veins. The bilateral brachiocephalic veins combine to form the superior vena cava (SVC). Brachiocephalic vein obstruction causes symptoms similar to those seen in SVC syndrome, but only on one side of the body.

Anatomy	Cardiovascular System	Superior vena cava syndrome
Subject	System	Topic

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A 62-year-old man who underwent mitral valve replacement 1 month ago is being evaluated in the emergency department for low-grade fevers. He has some malaise and dyspnea. Multiple sets of blood cultures are drawn and, within hours, all bottles grow gram-positive cocci in clusters that are catalase-positive and coagulase-negative. The decision is made to begin empiric antibiotic therapy. Initial empiric treatment should include which of the following antibiotics?

- A. Ceftriaxone
- B. Ciprofloxacin
- C. Clindamycin
- D. Nafcillin
- E. Penicillin G
- F. Vancomycin

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- A. Ceftriaxone (10%)
- ✗ B. Ciprofloxacin (2%)
- C. Clindamycin (2%)
- D. Nafcillin (19%)
- E. Penicillin G (18%)
- ✓ F. Vancomycin (46%)

Incorrect
Correct answer
F

46%
Answered correctly

03 secs
Time Spent

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Version

Explanation

This patient who was recently hospitalized for mitral valve replacement now has fevers and bacteremia with catalase-positive, coagulase-negative, gram-positive cocci in clusters. This is consistent with infection due to **coagulase-negative staphylococci** (CoNS). These organisms produce a polysaccharide **slime** facilitating prosthetic device adherence. As a result, CoNS are a major cause of infection in patients with indwelling catheters or implanted foreign bodies (eg, peritoneal dialysis catheters, vascular grafts) and a common cause of prosthetic valve endocarditis (PVE). For example, *Staphylococcus epidermidis* (a type of CoNS) can cause an indolent

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This patient who was recently hospitalized for mitral valve replacement now has fevers and bacteremia with catalase-positive, coagulase-negative, gram-positive cocci in clusters. This is consistent with infection due to **coagulase-negative staphylococci** (CoNS). These organisms produce a polysaccharide **slime** facilitating prosthetic device adherence. As a result, CoNS are a major cause of infection in patients with indwelling catheters or implanted foreign bodies (eg, peritoneal dialysis catheters, vascular grafts) and a common cause of prosthetic valve endocarditis (PVE). For example, *Staphylococcus epidermidis* (a type of CoNS) can cause an indolent endocarditis following valve replacement; if left untreated, it can lead to intracardiac abscess, prosthetic valve dehiscence, and septic embolization. As CoNS are a component of normal skin flora, it may be difficult initially to distinguish blood culture contamination from true infection; however, recovery of the same organism from several blood culture bottles indicates infection.

Most (>80%) CoNS strains are methicillin-resistant. Therefore, particularly with nosocomial infection (as is likely in this patient), CoNS should be assumed to be **methicillin-resistant** until proven otherwise. **Vancomycin** should be a component of initial therapy. Additional antimicrobial agents (eg, gentamicin and/or rifampin, which kills bacteria on foreign material) are administered in some cases of deep-seated methicillin-resistant CoNS infection or staphylococcal PVE. If susceptibility results later demonstrate a methicillin-susceptible isolate, vancomycin can be switched to a semi-synthetic β-lactamase-resistant penicillin such as nafcillin or oxacillin. Native valve endocarditis due to methicillin-sensitive staphylococci is best treated with nafcillin or oxacillin **(Choice D)**. Very few strains of *S epidermidis* are susceptible to penicillin G **(Choice E)**.

(Choice A) A methicillin-resistant strain of *S epidermidis* will likely be cephalosporin-resistant, as the mechanism of methicillin resistance (*mecA* chromosomal gene) involves alterations in the penicillin-binding protein PBP 2a, which is used by both penicillins and cephalosporins.

(Choice B) Ciprofloxacin is a fluoroquinolone antibiotic. Ciprofloxacin resistance appears to accompany methicillin resistance in staphylococci due to the simultaneous presence of multiple antibiotic resistance genes.

(Choice C) Although clindamycin can be used for the treatment of certain infections caused by oxacillin-resistant *Staphylococcus*, it is not appropriate initial therapy for CoNS bacteremia. Inducible clindamycin resistance exists

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(Choice B) Ciprofloxacin is a fluoroquinolone antibiotic. Ciprofloxacin resistance appears to accompany methicillin resistance in staphylococci due to the simultaneous presence of multiple antibiotic resistance genes.

(Choice C) Although clindamycin can be used for the treatment of certain infections caused by oxacillin-resistant *Staphylococcus*, it is not appropriate initial therapy for CoNS bacteremia. Inducible clindamycin resistance exists in some *S aureus* strains, resulting in treatment failure.

Educational objective:
Initial empiric treatment of coagulase-negative staphylococcal infection should include vancomycin due to widespread methicillin resistance, especially in nosocomial infections. If susceptibility results indicate a methicillin-susceptible isolate, vancomycin can be switched to nafcillin or oxacillin.

References

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